PROCEEDINGS

EIGHTY-SIXTH

ANNUAL MEETING

UNITED STATES ANIMAL HEALTH ASSOCIATION

RADISSON PLAZA & HYATT REGENCY HOTELS
Nashville, Tennessee
November 7-12, 1982
CONTENTS

Officers and Committees ......................................................... ix
Record of Previous Meetings .................................................... xxv
Invocation and Memorial Service ................................................ xxviii
Welcome to Tennessee — Roscoe Pickering ................................... xxx
Response to Welcome — Jack N. Armstrong .................................... xxxii
Report of the Secretary-Treasurer — J. C. Shook .............................. xlii
Address of the President-Elect — J. R. Ragan .............................. xlviii
Presentation of Plaque to President — J. R. Ragan ........................... li
Remarks of the President — G. B. Rea ....................................... lii
Animal Health Award—presented by H. C. Mussman, Administrator, USDA/APHIS ........................................ liii
Report of the Committee on Nominations and Resolutions —
L. W. Hunchman, et al ....................................................... liv
Amendment to the Bylaws of the United States Animal
Health Association ............................................................. lxiv
Proposed Amendment to the Constitution and Bylaws
of the United States Animal Health Association ........................ lxv
Forging A New Partnership — H. C. Mussman .............................. lxvii
Report of the Committee on Animal Welfare —
E. Mickey Stewart, et al ..................................................... 1
Report of the Committee on Food Animal Hygiene —
D. M. Bedell, et al ........................................................... 5
Report of the Committee on Infectious Diseases of Horses —
C. L. Campbell, et al ......................................................... 6
Presentation at the Animal Indentification Committee Meeting .......... 9
Report of the Committee on Livestock Identification —
H. Mindermann, et al ......................................................... 20
Report of the Committee on Professional Oversight —
P. L. Smith, et al ............................................................ 30
Report of the Committee on Parasitic Diseases and Parasiticides —
R. L. Pyles, et al ............................................................ 32
Report of the Committee on Public Health and
Environmental Quality — A. J. Roth, et al .................................. 36
Report of the Committee on State Federal Relations —
J. R. Ragan, et al ............................................................ 50
Report of the Committee on Wildlife Diseases —
E. Tom Thorne, et al ........................................................ 58

ANAPLASMOSIS

Economic and Epidemiological Implications of Anaplasmosis
in Texas Cattle Herds — F. J. Alderink and R. A. Dietrich ................ 66
Ultrastructure and Development of Colonies of Anaplasma
marginale in the Gut of Incubated Dermacentor andersoni —
K. M. Kocan, D. Holbert, S. A. Ewing, J. A. Hair and S. J. Barron ... 76

BIOLOGICS

The Role of FDA in the Regulation of Veterinary Biologics —
L. Crawford ............................................................... 98
Progress in Multivalent Modified Live Virus Bluetongue Vaccines: Comments on Safety and Efficacy—S. McConnell and C. W. Livingston, Jr. ........................................... 103
Development of Chemically Synthesized Antigens for Vaccines— J. L. Bittle ........................................................................ 114

Bluetongue-Bovine Leukosis
A Review of Methods to control Bovine Leukosis—J. M. Miller and M. J. Van Der Maaten ........................................ 119

Brucellosis
An Epidemiological Study of an Adult Vaccinated Jersey Herd Infected with Brucella abortus—S. L. Reynolds and J. L. Alexander .................................................. 136
Brucellosis Information System (BIS) in Field and Office and Impact on Tennessee Program—C. E. Barton and J. R. Ragan ............ 142
Status of the Cooperative State-Federal Brucellosis Eradication Program—B. G. Johnson ..................................................... 168
A Project to Determine Feasibility of Utilizing Cull Breeding Boars as Sentinel Animals for Swine Brucellosis Surveillance— G. H. Frye, P. Pickerill and I. T. Rhodes .................................................. 178
Report of the Committee on Brucellosis—J. B. Armstrong, et al ............ 185

Infectious Diseases of Cattle
Theileriosis and Prospects for Immunization—C. M. Groocock .................. 202
Report of the Committee on Infectious Diseases of Cattle— J. B. Young, et al ................................................................. 207

Leptospirosis
Hebdomadis Serogroup Leptospires in Florida Cattle— F. H. White and K. R. Sulzer ......................................................... 209
Leptospira Grippotyphosa Infection in the Dog—D. N. Tripathy, L. E. Hanson, J. Davis and K. Sulzer ........................................ 216
Report of the Committee on Leptospirosis—S. L. Diesch, et al ............... 219

Mastitis
Today’s Challenge for Preventing Mastitis in Dairy Herds— A. Bringe ................................................................................... 221
Mastitis Control—Present Technology and Future Prospect— J. S. McDonald .......................................................................... 226
ENVIRONMENTAL RESIDUES
Effect of Selenium on Toxicity of Aflatoxin B, and Pyrrolizidine Alkaloids in Animals—G. T. Edds and R. Bortell 236

FOREIGN ANIMAL DISEASES
Kinetics of Inactivation of ASF Antigen Using Binary Ethylenimine—G. M. Schloer 253
Rift Valley Fever: Global Spread or Global Control?—H. W. Lupton, C. J. Peters and G. A. Eddy 261
Vesicular Stomatitis Outbreak—Colorado—P. R. Henry 276
Report of the Committee on Foreign Animal Diseases—C. J. Mare, et al. 284

IMPORT-EXPORT
Export—The Bottom Line on Animal Health—H. A. Waters 295
Report of the Committee on Import-Export—Clint Booth, et al. 298

MORBIDITY AND MORTALITY
Historical Aspects of Animal Disease Reporting and the Developing of a National Animal Disease Surveillance System—W. R. McCallon and V. C. Beal, Jr. 341
The Fallacy of Drawing Inferences from Biased Data: Some Case Examples—W. R. McCallon and V. C. Beal, Jr. 350
Statistical Consideration In Sampling For Disease Surveillance—F. B. Martin 359

PHARMACEUTICALS, PESTICIDES AND RELATED TOXICOLOGY
Generic Competition in Animal Health Products—T. K. Shotwell 368
Monensin—Rumensin/Coban, Indications and Adverse Effects—G. T. Edds, R. Bortell 375
Inadvertent Residues in Food Animals Resulting from Contamination or Adulteration of Feeds—W. B. Buck 377
PSEUDORABIES
Update on DNA Fingerprinting of Pseudorabies Virus DNA— W. C. Lawrence .................................................. 402

RABIES
Rabies Diagnosis, A New Approach: Skin Biopsy Method for Antemortem Diagnosis of Rabies—D. R. Howard .................. 411
Intradermal Use of Human Diploid Cell Vaccine for Pre-Exposure Rabies Vaccinations of Humans—D. W. Dreesen, J. Brown, J. W. Sumner, D. T. Kemp .............................................. 413

SALMONELLA
The National Poultry Improvement Plan—An Update— R. D. Schar and I. L. Peterson .............................................. 445
Salmonella and Arizona Serotypes From Animals and Related Sources Reported During Fiscal Year 1981—B. O. Blackburn, K. Sutch and R. Harrington ......................................................... 454

SHEEP AND GOATS
Coronavirus and Adenovirus Infections in Lambs—J. A. Schmitz ........ 477
Report of the Committee on Sheep and Goats—M. C. Howard, et al .................. 478

TRANSMISSIBLE DISEASES OF POULTRY
ELISA and Flock Profiling in Management and Disease Control—W. W. Marquardt, D. B. Snyder and E. T. Mallinson .............. 480
Report of the Committee on Transmissible Diseases of Poultry and Other Avian Species—R. A. Bankowski, et al ...................... 482

SWINE
Streptococcal Lymphadenitis of Swine: A Review of Incidence and Research Activities—R. L. Wood .............................................. 503
Report of the Committee on Transmissible Diseases of Swine— J. P. Kluge, et al ......................................................... 511

TUBERCULOSIS
Effectiveness of Disinfectants on Mycobacterium paratuberculosis — R. S. Merkal and D. L. Whipple ...................... 514
Decontamination, Media, and Culture Methods for *Mycobacterium paratuberculosis*—R. S. Merkal, P. A. S. Lyle and D. L. Whipple.......... 519
Epidemiological Study of An Outbreak of Bovine TB in Confined Elk Herds—C. D. Stumpff.................................................. 524
Experimental Infection of Swine with *Mycobacterium avium* Serotype 4: Lesions and Transmission—H. M. Acland and R. H. Whitlock ................................................................. 528
Status of the State-Federal Tuberculosis Eradication Program—Fiscal Year 1982—R. L. Hosker .............................................. 534
Tuberculosis in Feedlot Animals—M. A. Essey and W. L. Searles........ 542
Report of the Committee on Tuberculosis and Johne's Disease—J. M. Dick, *et al* ................................................................. 546

ZOLOGICAL ANIMALS
Malignant Catarrhal Fever in Wild Ruminants—A Review and Current Status Report—W. P. Heuschele ......................... 552
Investigations of Malignant Catarrhal Fever in Ruminants at the Oklahoma City Zoo—E. R. Ramsay, A. E. Castro, B. M. Baumeister................................................................. 571
Report of the Committee on Zoological Animals—M. S. Silberman, *et al* ................................................................................. 583
OFFICERS AND COMMITTEES FOR 1983

PRESIDENT
J. R. Ragan .................................. Nashville, Tennessee

PRESIDENT-ELECT
J. O. Pearce, Jr. ............................ Okeechobee, Florida

FIRST VICE-PRESIDENT
D. U. Walker ................................ Montpelier, Vermont

SECOND VICE-PRESIDENT
N. W. Kruse ................................ Lincoln, Nebraska

THIRD VICE-PRESIDENT
J. F. Hudelson ................................ Denver, Colorado

SECRETARY-TREASURER
J. C. Shook ................................... Annapolis, Maryland

COMMITTEES

Committee on Anaplasmosis—1983

Chairman: K. L. Kuttler, Moscow, ID
Vice Chairman: A. A. Cuthbertson, Elko, NV

J. Lee Alley, Montgomery, AL          M. J. Jochim, Arvada, CO
J. F. Badger, Jefferson City, MO      E. Wynn Jones, Mississippi State, MS
D. M. Bedell, Tifton, GA              Stuart Lincoln, Caldwell, ID
G. M. Brown, Ames, IA                M. L. Main, Keldron, SD
G. M. Buening, Columbia, MO          Duane Miksch, Princeton, KY
D. L. Brinkmeyer, New London, IA     Dave Nash, Nampa, ID
W. B. Fairchild, Baton Rouge, LA     F. C. Neal, Gainesville, FL
R. I. Hail, Frankfort, KY            W. G. Nelson, Boise, ID
R. F. Hall, Tifton, GA               J. O. Pearce, Jr., Okeechobee, FL
R. L. Hartin, Oklahoma City, OK      E. J. Richey, Pawhuska, OK
T. J. Holt, Bowie, MD                M. Ristic, Urbana, IL
J. A. Howarth, Davis, CA            R. C. Searl, Ft. Dodge, IA
J. D. Huber, Reno, NV               N. R. Swanson, Cheyenne, WY

Committee on Animal Welfare—1983

Chairman: E. Mickey Stewart, Lincoln, NE
Vice Chairman: Neal Black, St. Paul, MN

G. C. Cilley, Concord, NH              Ann Gonernean, Kansas City, MO
Oscar Clabaugh, McLouth, KS            Carl Graham, Kansas City, MO
A. E. Decoteau, Dunstable, MA          T. M. Gustafson, Wakefield, NE
B. H. Ewald, Blacksburg, VA           Barbara Heffernan, Washington, DC
M. W. Fox, Washington, DC             Michele C. Howard, Sacramento, CA
H. M. Frederick, Arlington, VA         Donald Jónes, Netawaka, KS
Committee on Biologics—1983

*Chairman:* R. W. Loan, College Station, TX  
*Vice Chairman:* Major Huff, Denver, CO

<table>
<thead>
<tr>
<th>Name</th>
<th>Affiliation</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. C. Alexander</td>
<td>Ottawa, Ont., Canada</td>
</tr>
<tr>
<td>M. H. Bairey</td>
<td>Ames, IA</td>
</tr>
<tr>
<td>D. E. Baldwin</td>
<td>Omaha, NE</td>
</tr>
<tr>
<td>W. H. Beckenhauer</td>
<td>Lincoln, NE</td>
</tr>
<tr>
<td>D. E. Bordt</td>
<td>White Hall, IL</td>
</tr>
<tr>
<td>A. C. Braemer</td>
<td>Cupertino, CA</td>
</tr>
<tr>
<td>E. L. Drake</td>
<td>Reno, NV</td>
</tr>
<tr>
<td>D. A. Espeseth</td>
<td>Bowie, MD</td>
</tr>
<tr>
<td>John Finnell</td>
<td>White Hall, IL</td>
</tr>
<tr>
<td>J. S. Gloyd</td>
<td>Schaumburg, IL</td>
</tr>
<tr>
<td>R. F. Hall</td>
<td>Tifton, GA</td>
</tr>
<tr>
<td>B. B. Hancock</td>
<td>Ft. Dodge, IA</td>
</tr>
<tr>
<td>L. E. Hanson</td>
<td>Urbana, IL</td>
</tr>
<tr>
<td>R. E. Horton</td>
<td>Trenton, NJ</td>
</tr>
<tr>
<td>D. W. Johnson</td>
<td>St. Paul, MN</td>
</tr>
<tr>
<td>G. L. Johnson</td>
<td>Shawnee, KS</td>
</tr>
<tr>
<td>D. E. Kahn</td>
<td>Washington Crossing, NJ</td>
</tr>
<tr>
<td>L. H. Lauerman, Jr.</td>
<td>Auburn, AL</td>
</tr>
<tr>
<td>Vincent Marshall</td>
<td>Omaha, NE</td>
</tr>
<tr>
<td>Duane Pankratz</td>
<td>Freeman, SD</td>
</tr>
<tr>
<td>R. J. Price</td>
<td>Gaithersburg, MD</td>
</tr>
<tr>
<td>D. C. Randall</td>
<td>Evergreen, CO</td>
</tr>
<tr>
<td>R. C. Stewart</td>
<td>Shawnee Mission, KS</td>
</tr>
<tr>
<td>W. C. Stewart</td>
<td>Ames, IA</td>
</tr>
<tr>
<td>K. E. Thayer</td>
<td>Ontario, OR</td>
</tr>
<tr>
<td>J. D. Todd</td>
<td>Kansas City, KS</td>
</tr>
<tr>
<td>Hsi-tang Tung</td>
<td>White Hall, IL</td>
</tr>
<tr>
<td>P. R. Turner</td>
<td>Water Valley, TX</td>
</tr>
<tr>
<td>C. D. Van Houweling</td>
<td>Centreville, VA</td>
</tr>
<tr>
<td>G. B. E. West</td>
<td>Davis, CA</td>
</tr>
<tr>
<td>Robert Williams</td>
<td>Terre Haute, IN</td>
</tr>
</tbody>
</table>

Committee on Bluetongue and Bovine Leukosis—1983

*Chairman:* B. I. Osburn, Davis, CA  
*Vice Chairman:* Janice Miller, Ames, IA

<table>
<thead>
<tr>
<th>Name</th>
<th>Affiliation</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. A. Abt</td>
<td>Aldan, PA</td>
</tr>
<tr>
<td>H. D. Anthony</td>
<td>Manhattan, KS</td>
</tr>
<tr>
<td>Joan Arnoldi</td>
<td>Madison, WI</td>
</tr>
<tr>
<td>T. L. Barber</td>
<td>Lakewood, CO</td>
</tr>
<tr>
<td>D. E. Bartlett</td>
<td>Madison, WI</td>
</tr>
<tr>
<td>W. D. Bolton</td>
<td>Burlington, VT</td>
</tr>
<tr>
<td>G. M. Brown</td>
<td>Ames, IA</td>
</tr>
<tr>
<td>C. S. Card</td>
<td>University Park, PA</td>
</tr>
<tr>
<td>J. F. Cavanaugh</td>
<td>Columbus, OH</td>
</tr>
<tr>
<td>R. W. Fulton</td>
<td>Stillwater, OK</td>
</tr>
<tr>
<td>C. M. Groocock</td>
<td>New York, NY</td>
</tr>
<tr>
<td>Myron Holscher</td>
<td>Nashville, TN</td>
</tr>
<tr>
<td>T. J. Holt</td>
<td>Bowie, MD</td>
</tr>
<tr>
<td>J. A. House</td>
<td>Cutchogue, NY</td>
</tr>
<tr>
<td>M. C. Howard</td>
<td>Sacramento, CA</td>
</tr>
<tr>
<td>M. M. Jochim</td>
<td>Arvada, CO</td>
</tr>
<tr>
<td>L. L. Larson</td>
<td>Madison, WI</td>
</tr>
<tr>
<td>S. M. McConnell</td>
<td>College Station, TX</td>
</tr>
<tr>
<td>Blaine McGowan</td>
<td>Davis, CA</td>
</tr>
<tr>
<td>Mort Mertz</td>
<td>Eldorado, TX</td>
</tr>
<tr>
<td>H. E. Metcalf</td>
<td>Lakewood, CO</td>
</tr>
<tr>
<td>L. D. Miller</td>
<td>Ames, IA</td>
</tr>
<tr>
<td>M. E. Mix</td>
<td>Brattleboro, VT</td>
</tr>
<tr>
<td>D. R. Monke</td>
<td>Plain City, OH</td>
</tr>
<tr>
<td>R. S. Morris</td>
<td>St. Paul, MN</td>
</tr>
<tr>
<td>Dave Nash</td>
<td>Nampa, ID</td>
</tr>
<tr>
<td>V. F. Nettles</td>
<td>Athens, GA</td>
</tr>
<tr>
<td>J. E. Pearson</td>
<td>Ames, IA</td>
</tr>
<tr>
<td>Brent Perry</td>
<td>San Antonio, TX</td>
</tr>
<tr>
<td>J. R. Pitcher</td>
<td>Hyattsville, MD</td>
</tr>
</tbody>
</table>
Committee on Brucellosis—1983

Chairman: J. B. Armstrong, Kingsville, TX
Vice Chairman: L. D. Vanderwagen, Sacramento, CA

J. A. Acree, Jacksonville, FL
J. L. Alley, Montgomery, AL
J. F. Badger, Jefferson City, MO
C. E. Barton, Nashville, TN
J. W. Bitter, Edna, TX
Ramsay Burdett, Salem, OR
J. S. Cargile, San Angelo, TX
R. W. Cellon, Alachua, FL
John Cobb, Atlanta, GA
C. H. Cole, Lansing, MI
Tom Cook, Washington, DC
John R. Dahl, Gackle, ND
J. M. Dick, Harrisburg, PA
M. L. Dierks, Ewing, NE
Francis Drazek, Ithaca, NY
H. F. Embry, Peoria, IL
B. H. Espe, Oklahoma City, OK
W. B. Fairchild, Baton Rouge, LA
D. E. Flagg, Bismarck, ND
Bill Gallagher, Highmore, SD
Denny Gentry, Albuquerque, NM
J. H. Hagler, Arlington, TX
G. A. Hall, Oklahoma City, OK
R. L. Hartin, Oklahoma City, OK
J. W. Holcombe, Austin, TX
J. F. Hudelson, Denver, CO
C. N. Jewett, Cabot, AR
B. G. Johnson, Glenn Dale, MD
A. W. Keating, Park Ridge, IL
W. D. Knox, Ft. Atkinson, WI
W. E. Lyle, Madison, WI
F. E. Mann, Jr., Wharton, TX
W. F. Martin, Springer, NM
H. F. McCrory, Jackson, MS
Joyce Mitteness, St. Paul, MN
Sid Moore, Washington, DC
W. G. Nelson, Boise, ID
R. E. Nelson, Brattleboro, VT
B. F. Newcomb, Helena, MT
J. H. Niemi, Buffalo, SD
J. O. Pearce, Jr., Okeechobee, FL
W. D. Prichard, Salem, OR
W. C. Ray, Beltsville, MD
A. J. Roth, Richmond, VA
Larry Schaffer, O'Neil, NE
Raymond Schnell, Dickinson, ND
W. E. Stemler, Waterloo, IL
N. R. Swanson, Cheyenne, WY
D. U. Walker, Montpelier, VT
Taylor Woods, Humansville, MO

Brucellosis Scientific Advisory Committee—1983

L. G. Adams, College Station, TX
R. K. Anderson, Minneapolis, MN
D. T. Berman, Madison, WI
B. L. Deyoe, Ames, IA
C. S. Gue, Russellville, MO
M. E. Meyer, Davis, CA
D. E. Pietz, Ames, IA

Subcommittee on Swine Brucellosis—1983

Chairman: P. B. Doby, Springfield, IL

H. N. Becker, Gainesville, FL
Neal Black, St. Paul, MN
John Cobb, Atlanta, GA
Robert Combs, Las Vegas, NV
G. H. Frye, Hyattsville, MD
R. E. Hall, Madison, WI
Ralph Howe, Clemons, IA
M. E. Lang, Des Moines, IA
W. E. Lyle, Madison, WI
David Meisinger, Des Moines, IA
Phillip Pickerill, Des Moines, IA
Roy Poage, DeKalb, IL
Committee on Environmental Residues—1983

Chairman: G. D. Osweiler, Columbia, MO
Vice Chairman: W. B. Bixler, Rockville, MD

John Adams, Washington, DC
H. D. Anthony, Manhattan, KS
F. M. Applehans, Knoxville, TN
D. M. Bedell, Tifton, GA
W. B. Buck, Urbana, IL
S. J. Cougar, Austin, TX
C. R. Dorn, Columbus, OH
G. T. Edds, Gainesville, FL
H. M. Frederick, Arlington, VA
R. A. Gessert, Arlington, VA
H. S. Gosser, Tifton, GA
Carl Graham, Kansas City, MO
J. W. Howder, Tumwater, WA
C. S. Johnson, Denton, TX
F. R. Robinson, W. Lafayette, IN
D. G. Rollins, Springfield, MO
R. M. Scott, East Lansing, MI
J. L. Shupe, Hyde Park, UT
H. M. Trabosh, LaPlata, MD
T. M. Wilson, University Park, PA

Committee on Epizootic Attack—1983

Chairman: B. W. Hawkins, Ontario, OR
Vice Chairman: H. A. McDaniel, Silver Spring, MD

John Adams, Washington, DC
J. B. Anderson, Nashville, TN
R. A. Bankowski, Davis, CA
Neal Black, St. Paul, MN
J. L. Blair, Annandale, VA
W. O. Boaz, Laredo, TX
W. W. Buisch, Lanham, MD
Ramsay Burdett, Salem, OR
M. J. Burridge, Gainesville, FL
S. J. Cougar, Austin, TX
R. O. Drummond, Kerrville, TX
Joe Finley, Jr., Encinal, TX
W. C. H. Glaze, Helotes, TX
J. H. Graves, Southold, LI, NY
F. A. Hayes, Athens, GA
P. R. Henry, Denver, CO
J. L. Hyde, College Park, MD
E. T. Mallinson, Columbia, MD
N. L. Meyer, Alexandria, VA
M. A. Mixson, Montgomery, AL
T. G. Murnane, Mexico, DF
J. E. Novy, Laredo, TX
J. S. Orsborn, Sacramento, CA
B. I. Osburn, Davis, CA
H. G. Purchase, Beltsville, MD
T. B. Ryan, Cary, NC
E. C. Sharman, Hyattsville, MD
W. L. Sippel, Oviedo, FL
G. F. Slonka, Ft. Worth, TX
Kenneth Thomsen, Olympia, WA
M. J. Tillery, Laurel, MD
M. A. Van Buskirk, Harrisburg, PA

Committee on Food Animal Hygiene—1983

Chairman: D. M. Bedell, Tifton, GA
Vice Chairman: P. J. Friedman, Richmond, VA

A. F. Bailey, Oklahoma City, OK
J. A. Bell, Raleigh, NC
L. G. Billingsley, Sacramento, CA
J. L. Blair, Annandale, VA
A. D. Bond, Washington, DC
D. C. Breeden, Alameda, CA
W. H. Dubbert, Falls Church, VA
G. B. Estes, Richmond, VA
T. M. Gustafson, Wakefield, NE
R. E. Hall, Madison, WI
C. S. Johnson, Denton, TX
J. C. Leighty, Columbia, MD
T. E. Liner, Lubbock, TX
C. S. McCain, Stillwater, OK
H. O. Miller, Springfield, IL
J. K. Payne, Washington, DC
Wayne Wiekhorst, Phoenix, AZ
Committee on Foreign Animal Diseases—1983

Chairman: G. S. Trevino, College Station, TX

Vice Chairman: C. J. Maré, Tucson, AZ

Committee Members:

- P. N. Acha, Washington, DC
- W. W. Buisch, Lanham, MD
- A. R. Burgess, Cheyenne, WY
- J. J. Callis, Southold, NY
- Robert Combs, N. Las Vegas, NV
- A. H. Dardiri, South, LI, NY
- J. B. Finley, Jr., Encinal, TX
- W. C. H. Glaze, Helotes, TX
- J. H. Graves, Southold, LI, NY
- W. B. Grene, Gainesville, FL
- F. M. Hamdy, Southold, LI, NY
- Frank Harding, Geneva, IL
- C. P. Hughes, Atlanta, GA
- J. L. Hyde, College Park, MD
- E. W. Jenney, Ames, IA
- F. M. Jones, APO, Miami
- D. D. King, Mt. Airy, MD
- K. L. Kuttler, Moscow, ID
- C. A. Lamb, Sacramento, CA
- Vincent Marshall, Omaha, NE
- E. H. McCauley, Big Timber, MT
- S. McConnell, College Station, TX
- H. A. McDaniel, Silver Spring, MD
- D. G. McKercher, Davis, CA
- P. D. McKercher, Southold, NY
- N. L. Meyer, Alexandria, VA
- T. G. Murnane, Mexico, DF
- I. C. Pan, Southold, NY
- E. I. Pilchard, Hyattsville, MD
- G. Poppensiek, Ithaca, NY
- H. G. Purchase, Beltsville, MD
- S. L. Reynolds, College Station, TX
- Willard Rhynes, Stonewall, OK
- J. D. Roswurm, Sacramento, CA
- E. C. Sharman, Hyattsville, MD
- R. O. Spertzel, Frederick, MD
- P. Suttmoller, Rio de Janeiro, Brazil
- Dennis L. Thompson, Fair Oaks, CA
- O. H. Timm, Dixon, CA
- S. T. Wilson, Washington, DC
- T. M. Wilson, University Park, PA
- R. J. Yedloutschnig, Southold, NY

Committee on Import-Export—1983

Chairman: Clint Booth, Dallas, TX

Committee Members:

- J. N. Armstrong, Reno, NV
- Ken Baumgartner, Windsor, IL
- R. B. Caffey, Bowie, MD
- D. B. Childs, Lake Placid, FL
- S. J. Couger, Austin, TX
- Jack Dahl, Gackle, ND
- J. R. Day, Uvalde, TX
- R. L. Evinger, Boise, ID
- W. H. Fales, Columbia, MO
- A. E. George, Mt. Airy, MD
- D. J. Gilhooley, Kaneohe, HI
- R. C. Goulding, Sacramento, CA
- J. H. Gray, Austin, TX
- W. B. Grene, Miami, FL
- A. E. Hall, Springfield, IL
- Frank Harding, Geneva, IL
- Rube Harrington, Jr., Ames, IA
- B. W. Hawkins, Ontario, OR
- D. E. Herrick, Bowie, MD
- J. A. House, Cutchogue, NY
- R. C. Knowles, Silver Spring, MD
- M. L. Main, Keldron, SD
- Bob Mathis, Tempe, AZ
- M. E. Mix, Brattleboro, VT
- Robert Nicholas, Sonoma, CA
- E. G. Ongert, Reno, NV
- Brent Perry, San Antonio, TX
- D. A. Price, Littleton, CO
- William Prichard, Salem, OR
- G. B. Rea, Salem, OR
- Charles Reid, Clewiston, FL
- J. D. Roswurm, Sacramento, CA
- R. M. Scott, East Lansing, MI
- J. S. Walker, Greenport, LI, NY
- H. A. Waters, Arlington, VA
- C. R. Weston, Walpole, NH
- Walker Wilson, Overton, TX
Committee on Infectious Diseases of Cattle—1983

Chairman: Vaughn A. Seaton, Ames, IA
Vice Chairman: Willis E. Lyle, Madison, WI

W. F. Alexander, Oklahoma City, OK  M. L. Main, Keldon, SD
A. A. Anderson, Beltsville, MD  C. S. McCain, Stillwater, OK
H. L. Arnold, Charleston, W.VA  A. W. McClurkin, Ames, IA
R. P. Azelton, St. Joseph, MO  C. A. Mebus, Greenport, LI, NY
H. T. Barron, Knoxville, TN  Joyce Mitteness, St. Paul, MN
D. E. Bartlett, Madison, WI  M. A. Mixson, Montgomery, AL
Joe Bearden, Mississippi State, MS  B. F. Newcomb, Helena, MT
L. N. Brown, Centralia, WA  B. I. Osburn, Davis, CA
E. A. Carbrey, Ames, IA  J. O. Pearce, Jr., Okeechobee, FL
C. S. Card, University Park, PA  M. W. Peterson, Mt. View, CA
Pablo Correa Giron, Mexico, DF  S. L. Reynolds, San Antonio, TX
R. P. Crawford, College Station, TX  L. F. Roth, Wisner, NE
G. L. Crenshaw, Woodland, CA  J. A. Schmitz, Corvallis, OR
J. F. Evermann, Pullman, WA  R. D. Schultz, Madison, WI
R. W. Fulton, Stillwater, OK  W. L. Sippel, Oviedo, FL
G. D. Gurses, Topeka, KS  Richard Smith, Shawnee Mission, KS
R. F. Hall, Tifton, GA  P. L. Spencer, Springfield, IL
R. E. Horton, Trenton, NJ  Dan Suther, Redding, CA
N. W. Kruse, Lincoln, NE  N. R. Swanson, Cheyenne, WY
G. Lambert, Boone, IA  D. H. Tice, Bartow, FL
A. J. Luedke, Arvada, CO  M. Van der Maaten, Ames, IA
L. M. Siegfried, Madison, WI

Committee on Infectious Diseases of Horses—1983

Chairman: C. L. Campbell, Tallahassee, FL
Vice Chairman: R. C. Knowles, Silver Spring, MD

J. B. Anderson, Nashville, TN  W. O. Kester, Golden, CO
C. E. Boyd, Elgin, SC  M. J. Nolan, Washington, DC
G. C. Cilley, Concord, NH  S. R. Nusbaum, Trenton, NJ
Jesus Castaneda G., Maracay, Venezuela  M. A. Owen, Boston, MA
W. W. Clark, McAllen, TX  W. E. Pace, Tallahassee, FL
LeRoy Coggins, Cary, NC  Linda Schlater, Ames, IA
G. B. Estes, Richmond, VA  Victor Schroeder, Mexico, DF
P. M. Epple, University Park, MD  John Smiley, Augusta, ME
R. C. Goulding, Sacramento, CA  M. B. Tiegland, Hialeah, FL
J. B. Healy, Sacramento, CA  C. D. Vail, Littleton, CO
F. M. Jones, APO Miami  T. E. Walton, Lakewood, CO
M. J. Kemen, Ithaca, NY

Committee on Leptospirosis—1983

Chairman: S. L. Diesch, St. Paul, MN
Vice Chairman: H. C. Ellinghausen, Jr., Ames, IA

J. J. Cecil, Charles City, IA  John Finnell, White Hall, IL
J. R. Cole, Jr., Tifton, GA  R. F. Hall, Tifton, GA
### Committee on Livestock Identification—1983

**Chairman:** Harold Mindermann, West Des Moines, IA  
**Vice Chairman:** R. E. Nelson, Brattleboro, VT

<table>
<thead>
<tr>
<th>Name</th>
<th>City, State</th>
</tr>
</thead>
<tbody>
<tr>
<td>J. B. Ashcraft</td>
<td>Littleton, CO</td>
</tr>
<tr>
<td>J. H. Baldwin</td>
<td>Omaha, NE</td>
</tr>
<tr>
<td>P. E. Bradshaw</td>
<td>Griggsville, IL</td>
</tr>
<tr>
<td>D. R. Bridgewater</td>
<td>Northglenn, CO</td>
</tr>
<tr>
<td>H. F. Embry</td>
<td>Peoria, IL</td>
</tr>
<tr>
<td>G. B. Estes</td>
<td>Richmond, VA</td>
</tr>
<tr>
<td>Robert Gadd</td>
<td>Highmore, SD</td>
</tr>
<tr>
<td>Bill Gallagher</td>
<td>Highmore, SD</td>
</tr>
<tr>
<td>H. E. Goldstein</td>
<td>Columbus, OH</td>
</tr>
<tr>
<td>Tom Haas</td>
<td>Newport, KY</td>
</tr>
<tr>
<td>J. N. Huff</td>
<td>Denver, CO</td>
</tr>
<tr>
<td>G. M. Jones</td>
<td>Albuquerque, NM</td>
</tr>
<tr>
<td>Dee Likes</td>
<td>Topeka, KS</td>
</tr>
</tbody>
</table>

### Committee on Mastitis—1983

**Chairman:** C. A. Jordan, Morgan Center, VT  
**Vice Chairman:** J. S. McDonald, Ames, IA

<table>
<thead>
<tr>
<th>Name</th>
<th>City, State</th>
</tr>
</thead>
<tbody>
<tr>
<td>J. B. Adams</td>
<td>Washington, DC</td>
</tr>
<tr>
<td>R. W. Bennett</td>
<td>Conyers, GA</td>
</tr>
<tr>
<td>A. N. Bringe</td>
<td>Madison, WI</td>
</tr>
<tr>
<td>Robert Bushnell</td>
<td>Davis, CA</td>
</tr>
<tr>
<td>Charles Emerick</td>
<td>Cathlamet, WA</td>
</tr>
<tr>
<td>Carl Graham</td>
<td>Kansas City, MO</td>
</tr>
<tr>
<td>Thomas Fuhrmann</td>
<td>Chandler, AZ</td>
</tr>
<tr>
<td>Francis Gregerson</td>
<td>Longmont, CO</td>
</tr>
<tr>
<td>D. E. Jasper</td>
<td>Davis, CA</td>
</tr>
<tr>
<td>C. N. Jewett</td>
<td>Cabot, AR</td>
</tr>
</tbody>
</table>

### Committee on Morbidity and Mortality—1983

**Chairman:** G. Poppensiek, Ithaca, NY  
**Vice Chairman:** G. P. Combs, San Juan Puerto Rico

<table>
<thead>
<tr>
<th>Name</th>
<th>City, State</th>
</tr>
</thead>
<tbody>
<tr>
<td>R. K. Anderson</td>
<td>Roseville, MN</td>
</tr>
<tr>
<td>V. C. Beal, Jr.</td>
<td>Hyattsville, MD</td>
</tr>
<tr>
<td>S. L. Diesch</td>
<td>St. Paul, MN</td>
</tr>
<tr>
<td>J. G. Flint</td>
<td>St. Paul, MN</td>
</tr>
</tbody>
</table>
Committee on Nominations and Resolutions—1983

Chairman: G. B. Rea, Salem, OR

L. W. Hinchman, Indianapolis, IN
B. W. Hawkins, Ontario, OR

Committee on Parasitic Diseases and Parasiticides—1983

Chairman: R. L. Pyles, Albuquerque, NM
Vice Chairman: J. H. Gray, Austin, TX

Committee on Pharmaceuticals, Pesticides and Related Toxicology—1983

Chairman: W. A. Knapp, Jr., Raleigh, NC
Vice Chairman: G. D. Lindsey, Indianapolis, IN

---

M. E. Hugh-Jones, Baton Rouge, LA
N. E. Hutton, Corvallis, OR
J. L. Hyde, College Park, MD
R. F. Kahrs, Columbia, MO
Larry Mark, Springfield, VA
W. R. McCallon, Hyattsville, MD
E. H. McCauley, Big Timber, MT
H. A. McDaniel, Silver Spring, MD
W. R. Miller, Auburn, AL
L. G. Morehouse, Columbia, MO
R. S. Morris, St. Paul, MN
T. G. Murnane, Mexico, DF
J. C. New, Knoxville, TN
S. R. Nusbaum, Trenton, NJ
E. I. Pilchard, Hyattsville, MD
J. C. Prucha, Highland, MD
Philip Ross, Washington, DC
Leon Russell, College Station, TX
Vaughn A. Seaton, Ames, IA
G. H. Snoeyenbos, Amherst, MA
C. D. Van Houweling, Centreville, VA

---

Committee on Nominations and Resolutions—1983

Chairman: G. B. Rea, Salem, OR
L. W. Hinchman, Indianapolis, IN
B. W. Hawkins, Ontario, OR

Committee on Parasitic Diseases and Parasiticides—1983

Chairman: R. L. Pyles, Albuquerque, NM
Vice Chairman: J. H. Gray, Austin, TX

Committee on Pharmaceuticals, Pesticides and Related Toxicology—1983

Chairman: W. A. Knapp, Jr., Raleigh, NC
Vice Chairman: G. D. Lindsey, Indianapolis, IN

---

D. A. Armstrong, Lincoln, NE
D. T. Bechtol, Canyon, TX
W. B. Bixler, Fairfax, VA
Jerry Brunton, Alexandria, VA
W. B. Buck, Urbana, IL
H. F. Burton, Lutherville, MD
F. Carter, Springfield, MO
Tom Cook, Washington, DC
L. M. Crawford, Potomac, MD
G. T. Edds, Gainesville, FL
D. O. Farrington, Terre Haute, IN
J. E. Fox, Norcross, GA
D. A. Gable, Fairfax, VA
R. A. Gessert, Arlington, VA
Committee on Professional Oversight—1983

Chairman: P. L. Smith, Sacramento, CA  
Vice Chairman: H. E. Goldstein, Columbus, OH

C. L. Campbell, Tallahassee, FL  J. R. Ragan, Nashville, TN  
B. W. Hawkins, Ontario, OR Saul Wilson, Washington, DC  
J. D. Jefferies, Tampa, FL T. F. Zweigart, Raleigh, NC

Committee on Pseudorabies—1983

Chairman: L. W. Hinchman, Indianapolis, IN  
Vice Chairman: P. E. Bradshaw, Griggsville, IL

D. D. Gingerich, Parnell, IA  J. R. Ragan, Nashville, TN  
Don Hoogestraat, Chancellor, SD Carson Rogers, Ord, NE  
C. L. Kanitz, W. Lafayette, IN E. J. Slauter, Jefferson City, MO  
Myron Laffoon, Lafayette, IN Paul Spencer, Springfield, IL  
M. H. Lang, Des Moines, IA Willard Waldo, DeWitt, NE  
K. E. Myers, Grundy Center, IA T. F. Zweigart, Raleigh, NC  
J. P. Quigley, Jr., Riverdale, GA

Committee on Public Health and Environmental Quality—1983

Chairman: A. J. Roth, Richmond, VA  
Vice Chairman: R. H. Singer, Winchester, KY

R. K. Anderson, Roseville, MN W. R. Miller, Auburn, AL  
F. M. Applehans, Knoxville, TN E. V. Morse, W. Lafayette, IN  
A. W. Bailey, Oklahoma City, OK R. L. Parker, Columbia, SC  
R. P. Crawford, College Station, TX W. C. Patterson, Athens, GA  
S. L. Diesch, St. Paul, MN J. E. Pearson, Ames, IA  
C. R. Dorn, Columbus, OH J. C. Prucha, Highland, MD  
S. L. Hendricks, Watertown, MN L. W. Schnurrenberger, Silver Spring, MD  
W. T. Hubbert, Hyattsville, MD D. F. Schwindaman, Rockville, MD  
W. E. Jennings, San Antonio, TX T. P. Siburt, Blacksburg, VA  
J. C. Leighty, Columbia, MD T. B. Snodgrass, Dallas, TX  
M. R. Levy, Cherry Hill, NJ J. H. Steele, Houston, TX  
E. L. Menning, Annandale, VA C. D. Stumpff, DeSoto, KS
Committee on Public Relations—1983

Chairman: C. L. Campbell, Tallahassee, FL

Neal Black, S. St. Paul, MN                  Larry Mark, Springfield, VA
H. E. Goldstein, Columbus, OH              R. L. West, Schaumburg, IL
B. W. Hawkins, Ontario, OR

Committee on Rabies—1983

Chairman: Leon Russell, College Station, TX
Vice Chairman: W. R. Miller, Auburn, AL

W. H. Beckenhauer, Lincoln, NE            F. V. McCasland, Austin, TX
John Brown, Athens, GA                    J. C. New, Knoxville, TN
R. R. Brown, Auburn, AL                   J. C. Prucha, Highland, MD
D. W. Dreesen, Athens, GA                 F. T. Satalowich, Jefferson City, MO
J. W. Glosser, Helena, MT                 E. L. Shroyer, APO, NY
B. Hancock, Fort Dodge, IA                J. M. Shuler, Bargersville, IN
D. R. Howard, Manhattan, KS               A. Stratting, Ames, IA
Bruce Kaplan, Louisville, KY              Oscar Sussman, Tallahassee, FL
O. L. Kelsey, Little Rock, AR             W. G. Winkler, Atlanta, GA

Committee on Salmonella—1983

Chairman: B. S. Pomeroy, St. Paul, MN
Vice Chairman: G. H. Snoeyenbos, Amhurst, MA

C. W. Beard, Athens, GA                    C. S. McCain, Stillwater, OK
B. O. Blackburn, Ames, IA                  E. L. Menning, Annandale, VA
M. S. Cover, St. Louis, MO                 E. V. Morse, W. Lafayette, IN
M. L. Crandall, Gaithersburg, MD           J. P. Newman, East Lansing, MI
W. H. Dubbert, Falls Church, VA            Robert Nicholas, Sonoma, CA
R. D. Glock, Casa Grande, AZ               I. L. Peterson, Beltsville, MD
F. A. Hayes, Athens, GA                    R. A. Robinson, St. Paul, MN
R. L. Hogue, Lafayette, IN                 Raymond Schar, Beltsville, MD
W. L. Kadel, Hopkinsville, KY              J. D. Shroder, Des Plaines, IL
D. D. King, Mt. Airy, MD                   Keith Van Steenbergh, Springfield, MO
E. T. Mallinson, Columbia, MD              C. R. Weston, Walpole, NH

Committee on Sheep and Goats—1983

Chairman: Michele C. Howard, Sacramento, CA
Vice Chairman: J. R. Pitcher, Hyattsville, MD

J. A. Acree, Hyattsville, MD               William Hadlow, MT
Stan Allen, Logan, UT                       R. F. Hall, Tifton, GA
A. A. Anderson, Beltsville, MD             H. A. Hancock, Laramie, WY
M. H. Bairey, Ames, IA                      J. N. Huff, Denver, CO
R. B. Bushnell, Davis, CA                  M. M. Jochim, Arvada, CO
W. W. Clark, McAllen, TX                   Hyram Kitchen, Loudon, TN
J. E. Fox, Norcross, GA                     W. A. Knapp, Jr., Raleigh, NC
Committee on State-Federal Relations—1983

Chairman: J. O. Pearce, Jr., Okeechobee, FL

W. B. Fairchild, Baton Rouge, LA
H. E. Goldstein, Columbus, OH
B. W. Hawkins, Ontario, OR
J. F. Hudelson, Denver, CO
N. W. Kruse, Lincoln, NE

J. R. Ragan, Nashville, TN
G. B. Rea, Salem, OR
J. C. Shook, Annapolis, MD
M. A. Van Buskirk, Harrisburg, PA
D. U. Walker, Montpelier, VT

Committee on Transmissible Diseases of Poultry and Other Avian Species—1983

Chairman: R. A. Bankowski, Davis, CA
Vice Chairman: E. T. Mallinson, College Park, MD

W. W. Adams, Oakwood, GA
T. B. Angel, Jr., Florence, KY
R. E. Baer, Groveport, OH
C. W. Beard, Athens, GA
S. C. Clubb, Miami, FL
M. S. Cover, St. Louis, MO
H. M. Ghori, Little Rock, AR
H. E. Goldstein, Columbus, OH
E. E. Grass, Fair Oaks, CA
L. C. Grumbles, College Station, TX
R. L. Hogue, Lafayette, IN
I. H. Kahan, Doylestown, PA
D. D. King, Mt. Airy, MD
T. L. Landers, Hot Springs, AR
D. J. Ligda, Portage, IN

R. H. McCapes, Davis, CA
T. R. Mickle, Gainesville, GA
C. D. Murphy, Petersburg, IL
Mark S. Newman, Oklahoma City, OK
T. D. Njaka, Charleston, W. VA
W. C. Patterson, Athens, GA
J. E. Pearson, Ames, IA
L. L. Peterson, Beltsville, MD
B. S. Pomeroy, St. Paul, MN
S. S. Richeson, Crofton, MD
J. A. Smiley, Augusta, ME
J. W. Thomas, Newberry, SC
D. N. Tripathy, Urbana, IL
Carl Weston, Walpole, NH

Committee on Transmissible Diseases of Swine—1983

Chairman: J. P. Kluge, Ames, IA
Vice Chairman: D. G. Thawley, Columbia, MD

J. M. Alumbaugh, Macomb, IL
P. D. Beard, Ames, IA
L. G. Biehl, Urbana, IL
Neal Black, S. St. Paul, MN
C. E. Boyd, Elgin, SC
John Brown, Athens, GA

Jesus Castaneda, G., Marcy, Venezuela
R. A. Crandell, College Station, TX
A. M. Creswell, Nashville, TN
R. L. Daniel, Austin, TX
P. B. Doby, Springfield, IL
J. A. Downard, New Carrollton, MD
Gene Erickson, Ames, IA
D. P. Gustafson, Lafayette, IN
E. O. Haelterman, Lafayette, IN
R. E. Hall, Madison, WI
D. L. Harris, Ames, IA
G. W. Hausman, Des Moines, IA
H. T. Hill, Ames, IA
R. E. Horton, Trenton, NJ
C. L. Kanitz, W. Lafayette, IN
M. H. Lang, Des Moines, IA
Norman Lichtman, Sewell, NJ
Vincent Marshall, Omaha, NE
J. W. McVicar, Southold, NY
K. E. Myers, Grundy Center, IA
Carson Rogers, Ord, NE
Linda Schlater, Ames, IA
G. M. Schloer, Greenport, L.I. NY
L. W. Schnurrenberger, Silver Spring, MD
J. E. Slauder, Jefferson City, MO
W. C. Stewart, Ames, IA
Dennis L. Thompson, Fair Oaks, CA
R. E. Thompson, Jacksonville, AR
H. W. Towers, Dover, DE
Hsi-tang Tung, White Hall, IL
C. D. Van Houweling, Centreville, VA
J. D. Villari, Wenonah, NJ
B. D. Ward, Ballston Lake, NY
Fred Wertman, Des Moines, IA

P. A. O'Berry, Ames, IA

Committee on Tuberculosis and Johne's Disease—1983

Chairman: Sarah Hurley, Madison, WI
Vice Chairman: V. P. LaBranche, Boston, MA

J. A. Acree, Jacksonville, FL
Don Agresti, Ceres, CA
L. R. Barnes, Pendleton, IN
C. E. Boyd, Elgin, SC
J. M. Dick, Harrisburg, PA
M. A. Essey, Sacramento, CA
J. G. Flint, St. Paul, MN
G. H. Frye, Mitchellsville, MD
H. C. Hairson, Pomfret, MD
Stanley Harris, Ames, IA
Elmer Himes, Ames, IA
G. F. Hoffsis, Columbus, OH
R. L. Hosker, Calverton, MD
D. E. Hughes, Pierre, SD
C. A. Lamb, Sacramento, CA
L. L. Larson, Madison, WI
A. R. McLaughlin, Madison, WI
R. S. Merkal, Ames, IA
D. J. Myers, Nepean, Ont., CAN
W. J. Owen, Maxwell, IA
William Searles, Austin, TX
M. S. Silberman, Atlanta, GA
D. H. Smith, Olympia, WA
P. L. Smith, Sacramento, CA
G. R. Snyder, Reston, VA
P. L. Spencer, Springfield, IL
C. D. Stumpf, DeSoto, KS
C. O. Thoen, Ames, IA
Bruce Widger, Albany, NY
E. J. Wilson, Annapolis, MD

Committee on Wildlife Diseases—1983

Chairman: E. Tom Thorne, Laramie, WY
Vice Chairman: Victor F. Nettles, Athens, GA

W. D. Bolton, Burlington, VT
William W. Buisch, Lanham, MD
D. R. Cassidy, Ames, IA
A. H. Dardiri, Southold, L.I. NY
Gene Erickson, Ames, IA
Mitchell A. Essey, Sacramento, CA
Joe Finley, Jr., Encinal, TX
David J. Gilhooley, Kaneohe, HI
John H. Gray, Austin, TX
Frank A. Hayes, Athens, GA
D. A. Jessup, Rancho Cordova, CA
D. C. Johnson, Athens, GA
W. E. Ketter, Olney, MD
R. J. Lee, McLean, VA
H. A. McDaniel, Silver Spring, MD
E. V. Morse, Lafayette, IN
Lynne M. Siegfried, Madison, WI
J. S. Smith, Reno, NV
Committee on Zoological Animals—1983

Chairman: M. S. Silberman, Atlanta, GA
Vice Chairman: R. L. Crawford, Hyattsville, MD

R. A. Bowen, Wellington, CO          Robert Morgan, Bedford, TX
A. E. Decoteau, Dunstable, MA        G. P. Pierson, Glenn Dale, ND
P. M. Eppele, Weaver, AL             Jeanne Roush, Washington, DC
Gene A. Erickson, Ames, IA           D. F. Schwindaman, Rockville, MD
Milton Friend, Madison, WI           R. M. Scott, East Lansing, MI
E. E. Grass, Fair Oaks, CA          K. C. Sherman, Topeka, KS
D. E. Herrick, Bowie, MD             D. J. Williams, Athens, GA
C. J. Mikel, Oklahoma, OK            R. J. Yedloutschnig, Southold, NY
<table>
<thead>
<tr>
<th>Date</th>
<th>Place of Meeting</th>
<th>President</th>
<th>Secretary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sept. 27-28, 1897†</td>
<td>Fort Worth, Tex.</td>
<td>*Mr. C. P. Johnson, Springfield, Ill.</td>
<td>*Mr. D. O. Lively, Fort Worth, Tex.</td>
</tr>
<tr>
<td>Oct. 11-12, 1898</td>
<td>Omaha, Neb.</td>
<td>*Mr. C. P. Johnson, Springfield, Ill.</td>
<td>*Mr. Taylor Riddle, Kan.</td>
</tr>
<tr>
<td>Oct. 8-9, 1901</td>
<td>Buffalo, N.Y.</td>
<td>Dr. E. P. Niles, Va.</td>
<td>*Dr. F. T. Eisenman, Louisville, Ky.</td>
</tr>
<tr>
<td>Aug. 23-24, 1904</td>
<td>St. Louis, Mo.</td>
<td>Dr. J. C. Norton, Ariz.</td>
<td>*Mr. Wm. P. Smith, Monticello, Ill.</td>
</tr>
<tr>
<td>Dec. 5-6, 1911</td>
<td>Chicago, Ill.</td>
<td>Dr. John F. Devine, Goshen, N.Y.</td>
<td>*Mr. J. Ferguson, Chicago, Ill.</td>
</tr>
<tr>
<td>Dec. 3-5, 1912</td>
<td>Chicago, Ill.</td>
<td>Dr. Macayck P. Ravenel, Madison, Wis.</td>
<td>*Mr. J. Ferguson, Chicago, Ill.</td>
</tr>
<tr>
<td>Dec. 2-4, 1913</td>
<td>Chicago, Ill.</td>
<td>Dr. Peter F. Bahnson, Atlanta, Ga.</td>
<td>*Mr. J. Ferguson, Chicago, Ill.</td>
</tr>
<tr>
<td>Dec. 2-3, 1915</td>
<td>Chicago, Ill.</td>
<td>Dr. J. I. Gibson, Des Moines, Iowa</td>
<td>*Mr. J. Ferguson, Chicago, Ill.</td>
</tr>
<tr>
<td>Dec. 3-5, 1917</td>
<td>Chicago, Ill.</td>
<td>Dr. J. G. Wills, Albany, N.Y.</td>
<td>*Mr. J. Ferguson, Chicago, Ill.</td>
</tr>
<tr>
<td>Dec. 2-4, 1918</td>
<td>Chicago, Ill.</td>
<td>Dr. M. Jacob, Knoxville, Tenn.</td>
<td>*Mr. J. Ferguson, Chicago, Ill.</td>
</tr>
<tr>
<td>Nov. 29-30-Dec. 1, 1920</td>
<td>Chicago, Ill.</td>
<td>Dr. W. F. Crewe, Bismarck, N.D.</td>
<td>*Mr. Theo. A. Burnett, Columbus, Ohio</td>
</tr>
<tr>
<td>Nov. 28-30, 1921</td>
<td>Chicago, Ill.</td>
<td>Dr. T. E. Munse, Harrisburg, Pa.</td>
<td>*Mr. Theo. A. Burnett, Columbus, Ohio</td>
</tr>
<tr>
<td>Dec. 6-8, 1922</td>
<td>Chicago, Ill.</td>
<td>Dr. W. J. Butler, Helena, Mont.</td>
<td>*Dr. O. E. Dyson, Kansas City, Mo.</td>
</tr>
<tr>
<td>Dec. 5-7, 1923</td>
<td>Chicago, Ill.</td>
<td>Dr. J. G. Ferneyhough, Richmond, Va.</td>
<td>*Dr. O. E. Dyson, Kansas City, Mo.</td>
</tr>
<tr>
<td>Dec. 4-6, 1929</td>
<td>Chicago, Ill.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Date</td>
<td>Place of Meeting</td>
<td>President</td>
<td>Secretary</td>
</tr>
<tr>
<td>------------------</td>
<td>------------------</td>
<td>------------------------------------------------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>44. Dec. 4-6, 1940</td>
<td>Chicago, Ill.</td>
<td>*Dr. I. S. McDady, Auburn, Ala.</td>
<td>*Dr. L. Enos Day, Chicago, Ill.</td>
</tr>
<tr>
<td>62. Nov. 4-6, 1958</td>
<td>Miami Beach, Fla</td>
<td>*Dr. J. R. Hay, Chicago, Ill.</td>
<td>*Dr. R. A. Hendershott, Trenton, N.J.</td>
</tr>
<tr>
<td>63. Dec. 15-18, 1959</td>
<td>San Francisco, Calif</td>
<td>Dr. A. P. Schneider, Boise, Idaho</td>
<td>*Dr. R. A. Hendershott, Trenton, N.J.</td>
</tr>
<tr>
<td>64. Oct. 17-21, 1960</td>
<td>Charleston, W.Va.</td>
<td>Mr. T. J. Greenan, Jr., Providence, R.I.</td>
<td>*Dr. R. A. Hendershott, Trenton, N.J.</td>
</tr>
<tr>
<td>Date</td>
<td>Place of Meeting</td>
<td>President</td>
<td>Secretary</td>
</tr>
<tr>
<td>------------</td>
<td>-----------------------</td>
<td>------------------------------------------------</td>
<td>------------------------------------------------</td>
</tr>
<tr>
<td>70. Oct. 10-14, 1966</td>
<td>Buffalo, N.Y.</td>
<td>Dr. C. L. Campbell, Tallahassee, Fla.</td>
<td></td>
</tr>
<tr>
<td>76. Nov. 5-10, 1972</td>
<td>Miami Beach, Fla.</td>
<td>W. C. Tobin, Denver, Colo.</td>
<td></td>
</tr>
<tr>
<td>79. Nov. 2-7, 1975</td>
<td>Portland, Ore.</td>
<td>H. E. Goldstein, Columbus, Oh.</td>
<td></td>
</tr>
<tr>
<td>86. Nov. 7-12, 1982</td>
<td>Nashville, Tn.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Resigned Dec. 12, 1977**

*Deceased  †Reprinted in 54th Annual Report  ††Reprinted in the 68th Annual Report
+ This was the last meeting of the Interstate Association of Livestock Sanitary Boards
INVOCATION AND MEMORIAL SERVICE

F. James Schoenfeld, DVM
Salt Lake City, Utah

Our Father, who art in Heaven, hallowed be thy name. Thy will be done on earth as it is in heaven.

Father, we have assembled this night in the great State of Tennessee at the 86th Annual Meeting of the United States Animal Health Association and the 25th Annual Conference of the American Association of Veterinary Laboratory Diagnosticians.

Father, we are most grateful for the privilege of meeting together to learn, to teach, and to exchange knowledge pertaining to our responsibilities to the animal kingdom and our fellow man. We are grateful for this stewardship, and may we seek thee in guidance to fulfill these responsibilities.

We pray for the leaders of our two organizations, our presidents, our staff members who devote so much time and effort for the progress of these organizations. For the committee chairman and executive members may they continue to formulate the policies and examples that will provide for us the leadership for our various animal responsibilities in maintaining our stewardship.

We pray for our families and loved ones at home. May they be watched over and protected and that we may return to them and enjoy the good family relationship.

We are thankful for this land of America, a choice land and for these United States. We pray for the President, the Congress and all the newly elected persons. May thy blessings be with them that they may all work together in harmony that this nation may remain great and the people be entitled to thy blessings.

We ask thee that we might be prepared for the many troubles that lie ahead. That peace and harmony may prevail in our various countries and states and our communities.

We are thankful for the privilege of meeting in this beautiful city of Nashville, and for the fine hotels and facilities provided for us to make this a successful meeting and conference.

We pray for those who are ill amongst us and who are not able to be with us at this time. May thy healing influence be with them. We ask thee to especially be with those families who have lost loved ones as we remember them in our memorial service this night.

We are grateful for being able to meet this night in preparation for this coming week, and we ask thy blessings upon President Rea who is conducting this session and those who will be participating in the program
and the coming sessions of this meeting. And we offer this prayer unto thee in the name of Jesus Christ. Amen.

MEMORIAL SERVICE

Mr. President, Members of the United States Animal Health Association and the American Association of Veterinary Laboratory Diagnostics, Ladies and Gentlemen; it is our custom that we pause for a moment of silent prayer and meditation in reverence, that we may pay tribute and honor to those friends and colleagues who have completed their lives work here on earth and have passed on to Paradise.

Dr. J. B. Roberts — Roland, Oklahoma — October 14, 1981
Dr. Ernest S. Tierkel — Bethesda, Maryland — November 2, 1981
Dr. T. J. Grennan, Jr. — North Kingstown, R.I. — November 10, 1981
Dr. Don H. Spangler — Stanton, Nebraska — June 4, 1982
Mr. J. W. Ralph Bishop — Tipton, Indiana — July 20, 1982
Dr. Michael J. Polino — Enola, Pennsylvania — January 18, 1981
Mr. George Prosock — Phoenixville, Pennsylvania
WELCOME TO TENNESSEE
By Representative Roscoe Pickering
Chairman, House Agriculture Committee
General Assembly of Tennessee

It is a real honor for me to have the pleasure of welcoming such a distinguished group as this is to The Great State of Tennessee. It is even more meaningful to me because, not only am I doing this on behalf of our State' Government, but also on behalf of Governor Lamar Alexander, The Tennessee Department of Agriculture, and the Legislative Committee on Agriculture. I am proud to assist Commissioner William H. Walker, III in this. He is one of the best.

Agriculture is Number One in Tennessee. That's the new logo for The Tennessee Department of Agriculture. This logo emphasizes some of the things that many citizens seemed to have forgotten. Agriculture is this state's largest industry. Farmers' realized gross income was $2.2 billion in 1981, and farmers and farm-related industry produce over $12 billion in annual personal income. Agriculture is this state's largest exporter. Total agricultural exports were $435.8 million in 1981.

Three percent of Tennessee's 4.6 million population engages in crop and livestock production on 94,000 farms with an average 143 acres per farm. The livestock industry in Tennessee generated almost 50% of the total gross receipts in Agriculture in 1981.

Tennessee's top agricultural products are:

<table>
<thead>
<tr>
<th>Product</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soy Beans</td>
<td>$347 million</td>
</tr>
<tr>
<td>Dairy Products</td>
<td>308 &quot;</td>
</tr>
<tr>
<td>Cattle &amp; Calves</td>
<td>253 &quot;</td>
</tr>
<tr>
<td>Tobacco</td>
<td>252 &quot;</td>
</tr>
<tr>
<td>Hogs &amp; Pigs</td>
<td>153 &quot;</td>
</tr>
<tr>
<td>Poultry &amp; Eggs</td>
<td>115 &quot;</td>
</tr>
</tbody>
</table>

4 of the top 6 are livestock products for a total of $829 million.

Beef and dairy cattle provide 14% for the state's third leading agricultural commodity at $253 million. Tennessee ranks 13 among the 50 states in the inventory of beef cows. Dairy products, the state's second major income producer, provide 17% or $308 million to farmers. Tennessee also ranks 13th among all states in milk cows.

Swine provide 8% or $153 million of which $23 million is derived through feeder pig sales. The state leads the U.S. in the number of feeder pigs sold through organized, graded sales. Tennessee farmers sold 665,304 feeder pigs at 534 sales for an average of $33.96 per head in 1981. Since 1972, Tennessee has been nationally recognized as Hog Cholera Free. Tennessee ranks 14th among the 50 states with an inventory of 900,000 swine.
Poultry and eggs provide 6.3% of our agricultural income at $115 million of which $51 million or 2.8% is egg sales and $63 million or 3.4% is broiler sales.

There are 200,000 horses of all breeds in Tennessee. The Tennessee Walking Horse, which originated in this state, provides entertainment for all during the National Walking Horse Celebration in Shelbyville, the Tennessee Walking Horse Capital of the World. Besides Quarterhorses, Arabians, and Hunters and Jumpers, the state is a recognized collection point for pack mules for trail rides.

We are proud to have a top-notch Veterinary School in the State of Tennessee with some of the best instructors available, and we look forward to continued growth in this area. The U.T. School of Veterinary Medicine has been and continues to be one of the greatest assets to livestock farmers.

Here at the Ellington Agriculture Center, we have just completed a new Animal Diagnostic Lab Building which we are also mighty proud of, and we expect it to be highly beneficial to our livestock farmers. I have long been interested in and supported the development of an in-house veterinary toxicology capability for our diagnostic lab. This process is now under way.

You, as delegates to the 1982 Joint Meeting of the United States Animal Health Association and American Association of Veterinary Laboratory Diagnosticians, have an opportunity to make some of the most important decisions that have faced the livestock industry. I commend you for your diligence . . . I respect you for your ability and dedication . . . and I encourage you to assure that actions which you take will be in the long-term interest of the livestock industry of this nation.

It is indeed a pleasure to have you in our state.
RESPONSE TO WELCOME

Jack N. Armstrong, D.V.M.
Reno, Nevada

Thank you Representative Pickering. Good evening Ladies and Gentlemen. Speaking on behalf of The United States Animal Health Association and The American Association of Veterinary Laboratory Diagnosticians, I wish to express our sincere appreciation for the courtesy and hospitality already demonstrated by the Volunteer State of Tennessee.

That hospitality was also exemplified Saturday night for the 304 of us who had an opportunity to attend the splendid entertainment provided by your “Grand Ole Opry” of Music City, USA., a long to be remembered pleasurable experience.

We look forward to our stay in Nashville and to the achievements that will be forthcoming this week. Those achievements shall be accomplished through combined efforts of dedicated representatives from the many segments of our national livestock community. Those accomplishments will serve to promote and protect the agricultural productivity of Tennessee and of our great nation.

Upon reviewing this evening’s program, I noticed that 10 minutes have been reserved for this response. However, being of a merciful nature, I am reducing that period to 3 minutes.

In closing, I wish to say that the “Silver State” of Nevada extends a hand of friendship for our 1983 meeting in Las Vegas. Bob Combs, Nevada pork producer with National Facts, and I shall expend every effort to assist you in making your 1983 visit to the “Entertainment Capitol of the World” both productive and enjoyable!
ANIMAL HEALTH — WHOSE RESPONSIBILITY?

Thank you for giving me the opportunity to participate this evening in the annual meeting of the U.S. Animal Health Association here in Nashville.

During the next few minutes, I want to talk with you about my agency, the Agricultural Research Service, and describe how it fits into the Federal government's research establishment. Then, I would like to share with you our mission and goals, and some of our research discoveries.

I also will touch on some criticisms that have surfaced in recent reports on government research, and share with you our response to those criticisms. And finally, I will give you my personal opinion on our research outlook—what I see for us in the months and years ahead. I hope that together we can answer the question asked as the topic of my remarks today: "Whose responsibility is animal health?"

The reorganization effected by the Agriculture and Food Act of 1981, often referred to as the Farm Bill, reestablished the Agricultural Research Service, the Cooperative State Research Service, Extension Service, and the National Agricultural Library as separate agencies under an Assistant Secretary for Science and Education. The visibility of the missions of each of these agencies is understood by this reorganization.

I have brought a few slides with me, to help explain where ARS fits into the Departmental structure and how we are organized.

SLIDE SEQUENCE —

1. USDASecretary—Four Assistant Secretaries
2. Science and Education—4 agencies and their Administrators
3. ARS—Regional Administrators & NPS
4. ARS Regional Organization (Map)
5. Regional Administrators
6. NPS (Organization Chart)
7. National Research Program Leaders
8. National Technical Advisors
10. Public Agricultural Research Conducted (art)
11. Cash Receipts from Animal Commodities
12. ARS Livestock Program Increases, FY '82
13. ARS Beef Cattle Programs, FY '81
14. Beef Research—National SY's Allocated

The ARS facilities for agricultural research are extremely diverse and unequaled throughout the world. Research is conducted at many centers, laboratories and locations that range in size from small stations to the large 450-scientist facilities at the Beltsville, Maryland, Agricultural Research Center.

Most facilities are owned and operated by ARS, but several locations are provided by the U.S. General Services Administration or are leased. Some locations are designed to address research problems with a very specific focus. Others, such as the Beltsville Center, have a wide array of programs that are best addressed by a multidisciplinary approach.

FACILITIES

Let me give you a brief glimpse of some of the centers where animal health research is conducted.

15. Athens, Georgia—Newcastle disease
16. Beltsville, Maryland—Mastitis in cattle
17. East Lansing, Michigan—Marek's disease
18. Clay Center, Nebraska—Aerial view
19. Plum Island, New York—Aerial view
20. Miles City, Montana—Cattle nutrition study
21. Madison, Wisconsin—Aerial view of Dairy Forage Research Center
22. Denver, Colorado—Bluetongue disease (laboratory)

And I could continue for some time showing you research centers which are of importance in other ARS research areas—such as the new National Soil Erosion Laboratory located on the Purdue University Campus—the only laboratory in the U.S. devoted to soil erosion by water.

ARS facilities are located throughout the United States and in eight foreign countries so that regional, national, and international problems can be addressed in the most favorable research environment. Many are located on university campuses and State Agricultural Experiment Stations to assure cooperation and interaction with State scientists.

This type of arrangement encourages joint use of expensive research equipment, as well as of other essential facilities such as libraries, computers, and experimental plots.

ARS MISSION

Let me take a moment to list the various aspects of our Federal research role, which is—

* National in scope,
* Responsive to Executive and Congressional mandates,
* Meeting America's long-term food and fiber needs, and
* Responding to critical and emergency agricultural requirements.
More specifically, ARS conducts research that is—

* National in perspective in that it focuses on significant problems affecting the entire nation or its several broad geographic areas.
* Sufficiently long range, high risk and of such broad scope to require the unified planning, continuity of effort, and stable scientific environment maintained by the Federal research organization.
* Not undertaken by other agricultural research institutions because of their narrower geographic focus or shorter-term perspective.
* Uniquely a Federal responsibility in that it—
  * Supports APHIS and other Federal action and regulatory agencies and programs.
  * Is requested by Congress or the Executive Branch and requires special skills, facilities, or capabilities of ARS.
  * Requires a structure ready to respond to emergency situations of regional or national significance.
  * Is international in nature, supporting foreign policy initiatives of the U.S. government.
  * Supports the development and maintenance of important national collections that are essential to scientific activities.

To serve these goals, we must know our capabilities, recognize real and potential problems, listen to and communicate with our colleagues and clientele, and—perhaps most importantly—understand that we must do things differently because we continue to be told to do more with less.

These objectives will shape the manner in which ARS carries out its work. They deal with the responsibilities and effectiveness of Area, Regional, and Headquarters managers in terms of programmatic, administrative, and organizational roles and functions. Ultimately, these objectives translate into what I call the bottom-line questions:

* Are we sure that the mission pursued by ARS is currently true and on course?
* Are we assured and, in turn, can we assure others that our research is the highest priority and focuses on national concerns?
* Can we assure the American taxpayer, who is underwriting our performance, that our research is significantly productive, efficient, and responsive to America's needs.

ARS DISCOVERIES

Let me just touch on some of the work being conducted by ARS scientists—often in partnership with State and private industry researchers—and some of our recent discoveries.

I'm sure you are all aware of the new, safe, genetically engineered vaccine for foot and mouth disease. This disease is the world's greatest barrier to providing protein for human nutrition in developing countries. This breakthrough in the application of recombinant DNA technology can mean an increase in the world's supply of meat.
Improved control of the cattle fever tick may be possible now that ARS researchers, working with university scientists, have discovered hybrid sterility. Two almost identical tick species that cause the disease in many parts of the world have been identified and crossmated, producing sterile progeny. The ARS technique of releasing hybrid male ticks has proven accurate in mathematical models and gives promise of being useful in developing control methods.

A successful method of control could have world-wide implications, since cattle fever is found not only in the United States, but also in Australia, Asia, parts of Africa, Central and South America, Southern Europe and the Philippines.

An experimental vaccine administered to gilts before they are bred prevents porcine parvovirus (PPV) infection of their litters, should the gilts be exposed to virulent virus during gestation. PPV infection and disease accounts for losses in excess of 25 million dollars to the American hog industry annually.

A simple injection could become an effective replacement for the costly, potentially hazardous and waste-producing dipping of cattle to rid them of infecting mites that cause scabies. Presence of scabies in the United States has increased from one outbreak in 1970 to 262 outbreaks in 1980. An ARS scientist has shown that calves injected with low doses of avermectin (50 to 200 micrograms/kg) were free of infestations of scabies mites within seven days.

Coccidiosis is a disease produced by several species of protozoa that multiply inside the cells lining an animal’s intestine. This disease costs poultry producers over two hundred and fifty million dollars a year because birds fail to grow as they should and require continuous medication.

Recent progress by ARS scientists seeking to find a means of protection against coccidiosis involves the hybridoma technique. In this technique, a particular type of cancer cell from mice is fused with spleen cells from mice immunized with the coccidial organism. Monoclonal antibodies from some hybridomas restricted growth and multiplication of the parasites in preliminary trials, indicating that more effective protection against the disease may be possible.

There is good news for poultry producers in knowing that researchers have successfully vaccinated chicken embryos against Marek’s disease three days before they hatch. This new technique demonstrates for the first time that resistance can be established in chicken embryos through vaccination.

The protozoan Sarcocystis, a common parasite of dairy cattle, until recently was considered relatively harmless. However, ARS scientists have found that experimental infection with this parasite causes abor-
tions in cattle and sheep and reduces the quantity and quality of milk production of dairy cows. These findings have enabled the scientific community to initiate research on developing methods of diagnosis, prevention, and control.

Sheep are the most susceptible domestic animals to bluetongue virus; goats are considerably more resistant than sheep, and cattle are somewhere between the two. With no proven or economical method for control, we know that the disease is a problem in domestic livestock and has a severe impact on exporting sheep and cattle.

ARS scientists working to control bluetongue have developed an inactivated vaccine that protects sheep. The vaccine is prepared from two of the four American strains of the disease, and may give enough cross-protection so that all four strains do not have to be included. We are continuing investigations to find a multi-strain vaccine that will protect cattle as well.

The fact that African swine fever has penetrated our hemisphere and is on our doorstep in Haiti points up the importance of maintaining our defenses against disease and pest penetration from outside our borders. We are expanding our efforts in African swine fever research.

ARS is cooperating with the University of Minnesota for Salmonella studies in the turkey, and with the University of Wisconsin for Newcastle disease and influenza studies. Initial trials by our State cooperators have shown that turkey breeder flocks can be reared through three generations free from Salmonella infection.

In mentioning the progress our researchers are making in better animal health and protection, I haven't even touched on the benefits to be derived from the genetic breeding successes coming out of ARS laboratories.

These include a twinning rate in calves of about 20 percent in contrast with one to eight percent multiple births, depending on breed. Or crossbreeding under a three-breed rotational system under which four cows can produce as much weight of calf at weaning as five straightbred cows of the same breed.

Or increased efficiency in converting feed energy to lean meat by about one-half for beef and lamb, one-third for pork, one-fourth for rabbit and turkey, and one-sixth for broiler meat.

Or the concept of Integrated Reproduction Management (IRM), which is a good example of a program involving more than one agency as well as the users of the research. The program will provide a coordinated systems approach to improving the reproductive performance of food producing animals.

Because there are so many possible causes of poor reproductive perfor-
mance, integrated teams of research, teaching and extension scientists will be involved in assessment of research findings and needs, application of research results, and concurrent research to refine technology and fill the voids. The program will include studies on the role of malnutrition in infertility; toxic causes of early embryonic death; the role of microbial agents, hormones, and toxins—alone or in combination—in causing poor reproductive performance of the male or female.

Improving reproduction in food animals could realistically yield an annual economic benefit of one and a half billion dollars.

REPORTS, RECOMMENDATIONS, RESPONSES

Having spoken with confidence about our mission and with pride about our recent accomplishments, I must acknowledge that ARS has some problems. Over the recent past, our agency has been the subject of a number of studies, and there have been criticisms. Let me identify some of these studies, comment on their recommendations, and share with you our responses.

In July 1981, the General Accounting Office (GAO) submitted a study which concluded that the U.S. Agricultural R&D system does not perform national long-range planning, and that the USDA/State programs are independently planned.

The Office of Technology Assessment (OTA) submitted a study in December 1981. The OTA study noted that the USDA research expenditures are proportionately the smallest of any major Federal research agency. In 1978, USDA's share of Federal expenditures for R & D was 1.5 percent of total expenditures, compared with Defense (45 percent), Department of Energy (16 percent), and the Department of Health and Human Services (12 percent).

The Winrock Conference, held earlier this year, provided a summary of key issues confronting the agricultural research community. The Conference noted that many reasons have "greatly exacerbated national institutional resistance to change, resulting in excessive parochialism and preoccupation with institutional protection and maintenance." The Conference went on to point out that inadequate attention has been given to identifying critical research needs or to developing institutional relationships to bring about needed changes.

I submit to you that the Agricultural Research Service is basic to the future success of American agriculture. The work of ARS has been basic to the success of American agriculture since the agency was created in 1953. We have every reason to believe that the importance of ARS work will increase, if anything, because of the importance of American agricultural exports in the world market.

Of the ARS scientific staff, 77 percent are 40 years of age or older. That
figure implies that turnover and loss of experience will be heavy during the next few years.

We also face a shortage of young, recently trained research engineers, veterinarians, molecular biologists, and other critical specialists in the rapidly evolving disciplines needed to solve problems of the future.

The current ARS budget of about 450 million dollars annually has approximately the same purchasing power as the 1966 budget. Clearly, there are limits to the kinds and numbers of problems that can be undertaken within the budget levels. Hard choices must be made.

We are fortunate that Secretary Block has continued to assert that research is vital to achieving the Department's objectives for increased agricultural productivity and for expanded exports, while protecting and maintaining our natural resource base.

In fact, the budget of the Agricultural Research Service has fared relatively well, in comparison with other non-defense agencies. The Congress realizes that investments in the area of agricultural research will contribute to future agricultural productivity, economic returns, and national security.

ARS must maintain a research climate that attracts and retains the best scientists and thinkers in the nation in order to fulfill the Federal commitment of delivering the science and technology that is essential to meet the continuing long-term needs of agriculture.

In response to the report I mentioned, the Agricultural Research Service has initiated the most significant strategic planning activity in the agency's history.

The six objectives of the strategic plan develop the means to:

* Manage and conserve the nation's soil and water resources for a stable and productive agriculture;
* Maintain and increase the productivity and quality of crop plants;
* Increase the productivity of animals and the quality of animal products;
* Improve the system for delivery and conversion of raw agricultural commodities into food and useful products for domestic consumption and export;
* Promote optimum human health and performance through improved nutrition; and
* Integrate scientific knowledge on agricultural production and processing into systems that optimize resource management and facilitate the transfer of technology to end users.

I note that these six objectives describe the aims of ARS science. The words "develop the means to" are important. ARS is a research agency. Farmers and ranchers, action agencies, and the private sector will actually achieve these objectives.
I have within the past few weeks accepted one of the recommendations coming from the report of a study carried out by the Professional Oversight Committee of your association, which met in St. Louis in June 1981. The report addressed the responsibilities of the Agricultural Research Service (ARS) and of the Animal and Plant Health Inspection Service (APHIS).

APHIS is described in the report as having the primary responsibility for diagnostic services and training. I agree with that, and it is acknowledged in our mission statement.

In ARS we will work to strengthen support for APHIS programs, making sure that our research planning will incorporate the priority needs of APHIS.

We will also begin working toward implementing the recommendation that the diagnostic services and training of the Plum Island Animal Disease Center be assigned to APHIS. We will need to work with both the legislative and executive branches of the government to implement this recommendation, which we plan to complete no later than 1985. Only by working together can we provide the very best diagnostic service, training and research available anywhere. With that in mind, I am appointing a group to work with APHIS to ensure the orderly implementation of these recommendations.

I stand ready to accept recommendations for the improvement of ARS—recommendations about how we can best meet the challenges of the future—and there are a lot of them. But we have had quite enough studies, controversy and criticism to last a long time. Please give us a chance to respond to what has been said about America's agricultural research system.

We must address our critics. If they are wrong, we will tell them why they are wrong. If they are right, we'll respond, and we'll respond promptly.

We have already begun to respond. The whole Federal establishment is changing around us. And we are changing to meet the needs of the times.

We are developing the strategic plan I mentioned earlier to achieve the ARS mission and goals. This strategic plan identifies and explains the main problems that confront the agricultural industry and charts the minimum courses of action that will provide the research needed for solutions.

This plan is being developed under the leadership of the National Program Staff with input from more than 500 ARS scientists, and in consultation with colleagues from other agencies, the universities, and industry. I assure you, we are going to make this strategic plan work. We
will begin with our Senior Staff Conference on November 15—just one week from now.

And now, let's look at the question posed in the title of these remarks: Whose responsibility is animal health?

I hope my thoughts today have provided some answers to this important question. It is a responsibility all the actors must share—APHIS, Extension Service, the State Agricultural Experiment Stations, the Colleges of Veterinary Medicine, all the ranchers and farmers, and ARS. We all must be a part of it, working together, if we are to achieve the success we seek within the economic parameters of the times. Each actor must cooperate and trust the other, assuming and upholding a share of the responsibility.

Communication—open and free communication—is fundamental—vital. That is the reason I am glad to be here today to talk with you about my agency, ARS. Please accept my very best wishes for a successful meeting.
REPORT OF THE SECRETARY—TREASURER

J. C. Shook, V.M.D.

Mr. President, members and friends of USAHA and AAVLD, distinguished guests, Representative Pickering.

On behalf of the secretary's office, we welcome you to Nashville. After a rather cool weekend, we have arranged to have beautiful weather the remainder of the week. Our office has been busy, but with two great gals like Ella and Linda, we have managed to conduct our business and duties with a minimum of problems.

The organization continues to grow with approximately 1300 paid up members presently. As in all organizations with growth goes more work and responsibilities for our office and—more expenses. Unfortunately, we have fallen behind due to inflationary increases in cost of printing, postage, travel, etc., and are now facing a financial crisis. The Board of Directors are aware of our situation and I'm sure will take an objective look at our status and take the necessary action to alleviate our problems.

We have participated on a number of committees regarding cooperative programs during the past year and followed with a presentation of our organization policies to the appropriate Congressional Committees.

We are aware that the use of two hotels is not an ideal situation for the members of our two Associations. It also creates a great many problems for our staff, but in Nashville there was not a hotel that could give us ample sleeping and meeting rooms. It is hoped that with the beautiful weather the accommodations will be unduly inconvenient.

One of the lingering problems in our office is the failure of members to keep us informed of address changes and the failure to pay their dues. The cost of postage and office time make the necessity of billing members as many as three times very unprofitable. We remind all of you that we cannot continue this practice and in order to serve on a committee and receive communications from our office, you have to be a paid up member.

We appreciate the increasing number of members who preregister and hope that more of you will take advantage of this system in the future. Norm and Jay Powers are managing the registration as in the past and we can't thank them enough for the great job they do for us each year. Their hours of dedication are of immeasurable benefit to the meeting.

All committee chairmen are reminded that resolutions from their committee should not be included in their committee report. The resolution should be turned in to Dr. Lowell Hinchman, Chairman of the Resolutions Committee or left at the registration desk when completed on a form which is available at the registration desk. Resolutions not presented in the proper format cannot be acted on.
We hope your meetings are a success and that all of you enjoy your visit to Nashville.

All speakers should be reminded that papers must be turned in to the Chairman of each session when presentation is completed. This give us an opportunity to organize and expedite the printing of the Proceedings.

You have been sent a form to update our records on individual members and their membership affiliations. We would appreciate everyone taking a few minutes to complete this form and sending it back to us so that our records are current.

If our staff can do anything for you while you are here, please let us know. Thank you.
STATEMENT OF CASH RECEIPTS AND DISBURSEMENTS FOR PERIOD
OCTOBER 1, 1981 through SEPTEMBER 30, 1982

CASH BALANCE—OCTOBER 1, 1981:

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash on Hand — September 1, 1981</td>
<td>$ -</td>
</tr>
<tr>
<td>Bank of Virginia</td>
<td></td>
</tr>
<tr>
<td>Checking Account</td>
<td>211.18</td>
</tr>
<tr>
<td>Savings Certificate</td>
<td>10,000.00</td>
</tr>
<tr>
<td>Savings Account</td>
<td>1,346.98</td>
</tr>
<tr>
<td>Savings Account</td>
<td></td>
</tr>
<tr>
<td>(prepaid dues, registrations &amp; tours)</td>
<td>25,244.00</td>
</tr>
<tr>
<td>Savings Account</td>
<td>10,000.00</td>
</tr>
<tr>
<td>(Avian Influenza Symposium)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$ 46,802.16</td>
</tr>
</tbody>
</table>

INCREASED BY CASH RECEIPTS:

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individual Dues</td>
<td>$15,536.36</td>
</tr>
<tr>
<td>Official Dues</td>
<td>10,010.00</td>
</tr>
<tr>
<td>Proceedings</td>
<td>7,109.74</td>
</tr>
<tr>
<td>Reprints</td>
<td>4,537.82</td>
</tr>
<tr>
<td>Foreign Animal Books</td>
<td>192.00</td>
</tr>
<tr>
<td>Registration Fees</td>
<td>27,920.00</td>
</tr>
<tr>
<td>Tours</td>
<td>940.00</td>
</tr>
<tr>
<td>Interest Income</td>
<td>2,925.42</td>
</tr>
<tr>
<td>Change Fund</td>
<td>500.00</td>
</tr>
<tr>
<td>Repayment of I.R.A.—Ella R. Blanton</td>
<td>1,500.00</td>
</tr>
<tr>
<td>Lapel Pins</td>
<td>205.00</td>
</tr>
<tr>
<td>Avian Books</td>
<td>236.50</td>
</tr>
<tr>
<td>Audio Visuals, AAVLD and USDA</td>
<td>1,028.25</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>$72,641.09</td>
</tr>
</tbody>
</table>

TOTAL BEGINNING BALANCE AND RECEIPTS $119,443.25

STATEMENT OF CASH RECEIPTS AND DISBURSEMENTS FOR PERIOD
OCTOBER 1, 1981 through SEPTEMBER 30, 1982

ADD—RECEIPTS RECEIVED FOR YEAR

1982-83 prior to September 30, 1982:

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Registration Fees</td>
<td>$14,025.00</td>
</tr>
<tr>
<td>Individual Dues</td>
<td>5,180.00</td>
</tr>
</tbody>
</table>
Tours
Grand Old Opry
Transportation

3,325.00
3,243.00
788.75

$ 26,561.75
$ 146,005.00

DECREASED BY EXPENDITURES:

Annual Meeting
Printing
Office Supplies
Salaries
Office Secretary
Wages
Social Security Tax
Communication
Public Relations Coordinator
Public Relations Expense
I. R. A.—Ella R. Blanton
Public Relations Equipment
Travel:
   Dr. Glenn B. Rea
   Dr. J. C. Shook
   Dr. L. W. Hinchman
   Ella R. Blanton
Rent—Office Space
Other Meetings
American Association of Veterinary
   Livestock Diagnosticians
   Virginia Unemployment Insurance
Surety Bond—Treasurer
Miscellaneous Expense

3,143.22
45.45
242.00
800.52
3,475.00
2,846.82
8,125.00
205.54
32.00
1,004.39

122,132.60

CASH BALANCE—September 30, 1982:

Cash on Hand—September 30, 1982
Bank of Virginia
Richmond, Virginia
Checking Account
Savings Certificate
Savings Account
repaid Dues, Registration Fees, Tours, Grand Old Opry and Transportation

4,171.75
754.66
10,000.00
8,945.99

$ 23,872.40
SUMMARY OF OPERATIONS  
PERIOD OCTOBER 1, 1981 through SEPTEMBER 30, 1982

REVENUE:
Total Cash Receipts (1981-82) $97,885.09
Less — Expenditures 118,308.60
Excess of Expenditures over Receipts $20,423.51

NET WORTH — SEPTEMBER 30, 1982:
Cash on Hand — September 30, 1982 $ 4,171.75
Balance:
Bank of Virginia
Richmond, Virginia 754.66
Checking Account 8,945.99
Savings Account #0011563889 10,000.00
Savings Certificate
Accounts Receivable 3,463.88
Petty Cash Fund 25.00
Deposit — C. & P. Telephone Company Richmond, Virginia 100.00
Inventory — Supplies and Proceedings 28,500.00
Furniture and Fixtures 7,675.05

NET WORTH — SEPTEMBER 30, 1982 $ 63,636.33

SUMMARY OF OPERATIONS
FOR PERIOD OCTOBER 1, 1981 through SEPTEMBER 30, 1982

ANALYSIS OF CHANGE IN NET WORTH:
Net Worth — September 30, 1981 $80,073.79
Increased by:
Cash on Hand 4,171.75
Checking Account 543.48
Accounts Receivable 1,657.00
Inventory — Supplies and Proceedings 1,200.00
Furniture and Fixtures 3,635.30
Sub-Total $ 91,281.32
Decreased by:
Avian Influenza Symposium 10,000.00
Savings Account 17,644.99

NET WORTH — SEPTEMBER 30, 1982 $ 63,636.33

Henry H. Budd, Accountant

FOR PERIOD OCTOBER 1, 1981 through SEPTEMBER 30, 1982

ANALYSIS OF MISCELLANEOUS EXPENSE:
Auditing of Books $ 500.00
United Parcel Service 171.96
Safe Deposit Box Rent 12.00
Dr. Becton's Retirement Luncheon 17.00
Printing Checks 25.67
Flowers:
Dr. J. B. Roberts 17.16
Dr. T. J. Grennan 27.56
Mrs. Ella R. Blanton's Mother 13.00
Ralph Bishop 26.78
Contributions:
American Cancer Society 25.00
Dr. D. H. Spangler Merit
Scholarship Fund 25.00
Taxes:
Registration Fee-State of Delaware 10.00
Henrico County Tax 133.26

$ 1,004.39
ADDRESS OF THE PRESIDENT-ELECT

John R. Ragan, D.V.M.
Nashville, Tennessee

Distinguished guests, ladies and gentlemen, It is a particular pleasure for me to be able to add a word of welcome to each of you to the State of Tennessee and to Nashville; and also, to say a few words to you about how I perceive our organization as we embark on its eighty-seventh year.

I have had two local requests regarding this portion of the program. One was not to talk too long, and the other was to relate to you an interesting incident that recently took place near here. I'll try to honor both requests.

A man was visiting the home farm of an old school chum after a period of many years. As he was proudly shown around the barns, fields, and pastures, he noticed several times a large pig in a lot near the barn. He was startled to note that the pig was getting around rather well on a wooden rear leg. Hesitating until late in the day after commenting on the pleasant, well-kept premises, he asked his friend about the pig with the peg leg. "Well," the friend began, "there's a long story behind that pig." "One night last winter, my wood stove overheated and set the house on fire." "Being a sound sleeper, I was unaware that anything was wrong until I was awakened by a loud crash!" "That pig broke down the front door, rushed up the stairs, and dragged me downstairs and out into the yard to safety." "He revived me with mouth-to-mouth resuscitation—saved my life." "You know, when an animal does all that for you, you don't eat him all at once."

The relationship which we humans have with our animals has grown and developed almost since the history of man can be documented. We have used animals as beasts of burden, sources of food, and, increasingly, as friends. By and large, we have done an excellent job in use of animals as a food source. I recently saw a statistic which bears witness to this. It stated that with 8% of the world's cattle and sheep, our nation now produces about 20% of the world's food from those two species. Many other examples could be cited attesting to our success with dairy cattle, swine, poultry, and even fish. In most societies, the quality of life is seen as being closely related to the availability and consumption of animal-source foods.

Development of our relationship with animals as friends and companions is in a much earlier stage. In both areas, however, control of animal diseases is a critical part of any successful process. Also, the United States Animal Health Association (USAHA) should make a
continuing major contribution in administration of programs and articulation of needs in animal health.

USAHA has been for several years in a sustained period of growth in both depth and breadth. The thirty-odd standing committees discuss programs, policies, and scientific developments in far-ranging subjects from Anaplasmosis to Zoo animal care. The deliberations and opinions developed here this week will have major interest and impact for industries, governments, and people throughout our nation and beyond.

I have always stood in awe of the sheer volume of expertise in animal health assembled each time these organizations gather for an annual meeting. This evening is no exception. My fervent hope is that our organizational activities will serve to focus and utilize those abilities on the practical problems that exist, rather than to diffuse and frustrate them.

Often the most controversial and apparent activities of our Association deal with the regulation of commerce and the content and administration of organized state-federal programs. A casual observer might tend to overlook the solid bedrock of scientific ability represented in USAHA by our large contingent of research, teaching, and extension members. These include AAVLD Joint Members and others, such as those in the Agricultural Research Service, whose work has been noted here tonight. Without detracting from any of the variety of government and industry representatives here, I would like to pay particular note tonight to the contribution which this group of people has made to our success over the years, and continues to make. Truly, the productive mix that is USAHA is continually enriched by their labors.

The very diversity of individuals in our organization make it imperative that we have an active membership and a strong, functional committee system. Our record in this area is both good and bad. On the one hand, a group of able, dedicated committee chairmen continue to provide high quality discussion of current information and issues, and to develop well-considered proposals for actions and policy positions for the Association. On the other hand, we still have too many members who do not take an active part in the business of the organization. It is my intent to vigorously search for ways to encourage broader member participation this year, and to expand on the progress made in this area in the past year.

As you know, there are hazards in any system of operating an organization with membership of widely varying backgrounds and interests. One of the very real potential problems which we must guard against is the projection of minority opinions or positions as Association policy. This can occur through a failure of the process for developing
those positions. The considerable respect and influence of USAHA, earned over many years by the dedication and wisdom of its membership must be protected from abuse by special interests of any viewpoint. Each of us has a responsibility to be certain that recommendations which are produced by our deliberations are soundly based upon consensus judgment of scientific and economic fact and not upon wishful thinking or political expedience. A major responsibility in this function lies with the Association’s Executive Committee. I believe that that body, with representation from every official and affiliate member of USAHA, and a number of outstanding individual members, must take a more active role in oversight of committee reports and resolutions.

I will ask that careful consideration be given to every report, resolution, and policy statement which becomes part of a USAHA approved position.

You know, if we make a mistake in judgment or process on any matter, and adopt an unwise position, we almost certainly must live with it for a year, until the next annual meeting.

It is my view that no organization should have committees which are unnecessary or non-functional. Further, I believe that committees should not retain members who are unwilling to participate in the group's work. Committee reports should reflect the considered judgment of the entire membership, and not the views of the chairman or one or two members who get hooked into writing a report. I will ask each committee chairman this year to critically examine the role of his committee and to recommend desirable changes in structure or membership.

In the years just ahead, the availability of public funds will continue to be at least as uncertain as this year. This means that concerned parties in industry and government will have to do a better job of establishing animal health priorities and of making efficient use of the funds that are available for meeting those priorities. USAHA has great potential to aid in achieving those ends. That potential will be considerably enhanced if we can strengthen the participation of private sector veterinarians and livestock producers. One way we will seek to get this done this year is through public information about USAHA. We will look to our Public Relations Committee for direction in this effort to get the most public awareness of USAHA possible within our means.

The challenges in animal health are greater than ever. The role which you can play in meeting those challenges is virtually unlimited. It is a singular honor for me to be a part of your efforts.
Dr. John R. Ragan, President-Elect, presents plaque to outgoing President, Dr. G. B. Rea, for his outstanding leadership for 1981-1982.

REMARKS BY THE PRESIDENT

G. B. Rea, D.V.M.
Salem, Oregon

Honorable Roscoe Pickering, Dr. Kinney, Ladies and Gentlemen!

This 86th session of the United States Animal Health Association will bring to an end my term as your president. This organization has meant a lot to me over the past years and I hope I have been able to pay a little interest on a long standing debt.

Last year we gave every member an opportunity to participate in at least one committee in order that they might not only contribute to the deliberations of their organization, but also that they might actually be part of it rather than just a dues paying joiner. Don't get me wrong—under present economic conditions we'll take all the joiners we can get; however, joiners do not give strength and substance to an organization. Most of the committee chairmen have continued to brief me concerning their activities during the year. I want you to know that many of these men—yes and ladies too—as we do have one committee chair-
man and one co-chairman who can cook, sew and keep house as well as administer a business. These people not only chair the committee while in session, but continue aggressively month after month around the year. I want to thank all of you who have labored in this fashion.

During this past year we have augmented our public relations activities by developing and displaying a descriptive booth which has been exhibited at the National Cattlemens Association, the American Veterinary Medical Association and the Southern Animal Health Association. Experts in this field have complimented us on the quality of this display. We have been promised space at the Intermountain Veterinary Conference in Las Vegas this year, and we anticipate exhibiting again at the National Cattlemens Association, American Veterinary Medical Association and other prominent conventions of allied organizations.

Although all of these organizations have given us complimentary booth space, we still have a financial problem in that it costs money to ship this equipment around the country. We also have to have qualified people to man the exhibit. Hopefully these problems can be solved.

With all the good things that have happened this past year, a very sticky problem has developed, and you might as well know about it right now. In order to keep this organization going, we have had to exceed our budget for 1982. This means that we either have to develop more income or decrease expenditures and probably do both. This is a task that will be addressed by the Executive Board and the Board of Directors. If any of you have any sage advice, please bring it forth.

You have honored me greatly by electing me to this office. I hope I have responded as you desired. Many times over the years I have been admonished to use the K.I.S.S. principle—Keep It Simple, Stupid. That is what I have tried to do tonight.

Although you will see me at certain times and places during this convention, this will be my last official presentation before this body. As such I would like to leave you with this hope for the future. —A Little Irish Prayer

May the road rise gently to meet you
May the wind be always at your back
May the sun shine warm upon your face,
May the rains fall soft upon your field, and
May the good Lord always hold you in the Hollow of His Hand!
Dr. H. C. Mussman, Administrator, presents Animal and Plant Health Inspection Service's Animal Health Award to Dr. J. N. Armstrong, State Veterinarian of Nevada, accepting for Dr. John L. O'Harra, former State Veterinarian of Nevada.

Dr. G. B. Rea, President, USAHA, presents to Dr. C. L. Campbell the Presidential Award for his outstanding contributions and dedication as Chairman of the Public Relations Committee.
REPORT OF THE COMMITTEE ON NOMINATIONS AND RESOLUTIONS

The Committee on Nominations and Resolutions presents the following slate of officers for election at this meeting for your consideration. Their names will be posted on the bulletin board at the registration desk for 24 hours according to our Bylaws:

President ........................................... John R. Ragan
Nashville, Tennessee

President-Elect ................................. J. O. Pearce, Jr.
Okeechobee, Florida

First Vice President ......................... David U. Walker
Montpelier, Vermont

Second Vice President ....................... N. W. Kruse
Lincoln, Nebraska

Third Vice President ......................... John Hudelson
Denver, Colorado

Treasurer ........................................ J. C. Shook
Annapolis, Maryland

Regional Representatives
Northeast ................................. Victor LaBranche
Boston, Massachusetts

                     Everett Bryant
Storrs, Connecticut

North Central ......................... Bill Gallagher
Stephan, South Dakota

                     Phil Bradshaw
Griggsville, Illinois

South .......................... Joe Finley, Jr.
Encinal, Texas

                     William Baisley
Daiton, Georgia

West .......................... Olin Timm
Dixon, California

                     Richard McCapes
Davis, California
RESOLUTIONS
United State Animal Health Meeting
Dates: November 7-12, 1982
Nashville, TN

RESOLUTION No. 1
Source: Animal Welfare Committee
Subject Matter: Farm Animal Welfare

Resolution

BE IT RESOLVED that advances in farm animal welfare based on scientific studies and practical application in the United States and abroad, be further encouraged by the USAHA.

RESOLUTION No. 2
Source: Committee on Sheep and Goat, Zoological, Wildlife Disease & Import-Export
Subject Matter: Import Policy

Resolution

THEREFORE BE IT RESOLVED, that the USAHA officers and membership go on record as supporting APHIS in their stand to protect the domestic livestock industry and this nation's wildlife resources from disease both domestic and foreign as their No. 1 priority.

RESOLUTION No. 3
Source: Zoological Animal Committee
Subject Matter: African type of malignant catarrhal fever (MCF)

Resolution

THEREFORE, BE IT RESOLVED, that the U.S. Animal Health Association (USAHA) urges each state to promulgate regulations to require landowners to obtain a permit from the office of the respective State Veterinarian to breed and/or raise any type of inapparent carrier animals recognized in the world, such as wildebeest and other wild ruminants, so current and future landowners and users can be duly informed of the risk involved.

Furthermore, the USAHA urges the U.S. Department of Agriculture (USDA) to establish policies for a reference center to study MCF within the Continental United States with appropriate biological security (at least P III), so that the threat to zoological animals and livestock can be more accurately assessed and reduced to the maximum extent feasible.
RESOLUTION No. 4
Source: Bluetongue and Bovine Leukosis, Sheep and Goats
Subject Matter: Bluetongue Virus Diagnostics

Resolution

NOW LET IT BE RESOLVED THAT:
The U.S. Department of Agriculture continue to support research directed at the development and application of new and improved technologies to detect and diagnose bluetongue virus infection.

RESOLUTION No. 5
Source: Committee on Sheep and Goats and Import-Export
Subject Matter: Safe Importation and Exportation of Embryos

Resolution

THEREFORE BE IT RESOLVED, that the U.S.A.H.A. recognize the emerging importance of embryo transplant techniques, and requests that U.S.D.A. initiate a major effort to support and encourage research necessary to develop a policy for safe importation and exportation of embryos through increased federal funding for ARS research project in the specific area of embryo disease transmission.

RESOLUTION No. 6
Source: Bluetongue and Bovine Leukosis Committee
Subject Matter: Bovine Leukosis Virus Free Herd Certification

Resolution

BE IT RESOLVED, the USAHA recommends that Veterinary Services, APHIS proceed with plans to implement the proposed Uniform Methods and Rules for Establishment and Maintenance of Designated Bovine Leukosis Virus Free Herds.
RESOLUTION No. 7
Source: Committee on Sheep and Goats
Subject Matter: Scrapie Eradication Program

Resolution

THEREFORE BE IT RESOLVED, that U.S.A.H.A support a scrapie program in which bloodline animals only are destroyed and exposed animals are permanently identified and maintained under surveillance.

RESOLUTION No. 8
Source: Transmissible Diseases of Poultry and Other Avian Species
Subject Matter: Proposed Resolution

Resolution

THEREFORE BE IT RESOLVED THAT:
USDA should endorse the proposed National Cage and Aviary Bird Improvement Plan (NCABIP) through assignment of personnel and necessary resources to speed and further advance the development and implementation of the NCABIP Plan.

RESOLUTION No. 9
Source: Transmissible Diseases of Poultry and Other Avian Species
Subject Matter: Proposed Resolution

Resolution

THEREFORE, BE IT RESOLVED, that
I. USDA encourage intramural and extramural research with the goal of development of technique for the diagnosis of Newcastle disease and chlamydiosis that will be rapid and accurate, and thus widely applicable in the field.

II. USDA make necessary resources available to determine the parameters of effective antibiotic treatment of the various cage bird types marketed in the United States.
RESOLUTION No. 10
Source: Transmissible Diseases of Poultry and Other Species Committee
Subject Matter: Proposed Resolution

Resolution

NOW, THEREFORE, BE IT RESOLVED that the USAHA endorse the model program developed by the Committee on Transmissible Diseases of Poultry and other Avian Species for the "Certification of VVND Negative Primary Breeding Flocks" for the egg and broiler industries and requests that USDA adopt and implement a program which carries out this intent.

RESOLUTION No. 11
Source: Zoological Animal Committee
Subject Matter: Interstate Health Certificate

Resolution

LET IT THEREFORE BE RESOLVED, that the U.S. Animal Health Association (USAHA) urge each state and the U.S. Department of Agriculture to consider changing the certification statement to read "The above described animal(s) have been examined by me and are free of clinical signs of disease, have reacted as indicated to the several tests as noted, and have received the vaccinations as indicated."

RESOLUTION No. 12
Source: Food Animal Hygiene Committee
Subject Matter: Disposition of Mycobacteriosis Swine Carcasses

Resolution

NOW BE IT RESOLVED that if current research shows no evidence of hazard to human health, the disposition of swine carcasses containing lesions attributed to mycobacteriosis should be reassessed.
RESOLUTION No. 13
Source: Foreign Animal Disease Committee
Subject Matter: Foreign Animal Disease Manual

Resolution
BE IT THEREFORE RESOLVED, that the USAHA urges the FAD committee to take responsibility for reviewing the expanding "The Foreign Animal Disease Manual."

RESOLUTION No. 14
Source: Foreign Animal Disease Committee-Co-sponsors with Wildlife Committee
Subject Matter: Foreign Animal Disease Training of Wildlife Veterinarians

Resolution
THEREFORE BE IT RESOLVED, that the United States Animal Health Association urges USDA to open the Foreign Animal Disease Training Course to wildlife veterinarians as class space and budgetary considerations allow.

RESOLUTION No. 15
Source: Swine Brucellosis Subcommittee
Subject Matter: Enforcement of Swine Brucellosis Identification Requirements

Resolution
NOW THEREFORE BE IT RESOLVED that the United States Animal Health Association call on Secretary of Agriculture John Block to stimulate increased prosecution of violators of the identification regulations of the swine brucellosis eradication program.
RESOLUTION No. 16
Source: Committee on Salmonella
Subject Matter: Voluntary Good Manufacturing Practices for Renderers, Blenders and Feed Manufacturers

Resolution
WHEREAS, animal feeds are frequently contaminated by Salmonella and may serve as a source of infection for animals and
WHEREAS, improvement in manufacture and process of feed stuffs may significantly reduce such contamination, therefore, be it resolved that the USDA recommends Guidelines for Voluntary Good Manufacturing Practices be prepared by USDA in cooperation with other Governmental Agencies and industries involved for use by (a) Renderers, (b) Blenders and (c) Feed Manufacturers.

RESOLUTION No. 17
Source: Committee on Salmonella
Subject Matter: Veterinary Epidemiologists

Resolution
WHEREAS, there is insufficient information to allow the development of a control program, THEREFORE, BE IT RESOLVED that the USAHA recommends that one or more epidemiologists be made available by VS-APHIS-USDA to conduct field investigations, to analyze all available data and to determine economic impact of animal salmonellosis such as S. dublin in cattle and swine.

RESOLUTION No. 18
Source: Committee on Sheep and Goats
Subject Matter: Study of Border Disease of Sheep

Resolution
THEREFORE, BE IT RESOLVED, that the U.S.A.H.A. recommend that U.S.D.A. support a study to determine the prevalence of Border Disease, and the economic impact of this disease on the sheep industry of the United States.
RESOLUTION No. 19
Source: Mastitis Committee
Subject Matter: Increased Funding for Basic Mastitis Research

Resolution

THEREFORE, the USAHA Mastitis Committee recommends to the USAHA that a letter be transmitted through Dr. Earl Splitter to the USDA Animal Health Advisory Committee, that there is an immediate need for increased funding of basic mastitis research.

RESOLUTION No. 20
Source: Tuberculosis and Johne's Disease Committee
Subject Matter: To restore funds

Resolution

THEREFORE, BE IT RESOLVED, that APHIS-Veterinary Services initiate efforts for an increased appropriation for tuberculosis indemnity to restore the funds deleted from the 1983 budget.

RESOLUTION No. 21
Source: Committee on Livestock Identification
Subject Matter: Animal Identification

Resolution

BE IT RESOLVED, that USAHA support research and evaluation of types of identification that are most acceptable to the livestockmen, dealers, markets, regulatory officials, practicing veterinarians, and slaughter recovery personnel.
RESOLUTION No. 22  
Source: Committee on Livestock Identification  
Subject Matter: Animal Identification  

Resolution  
THEREFORE BE IT RESOLVED, at the annual meeting of the Committee on Livestock Identification of the United States Animal Health Association in Nashville, Tennessee, that the U.S.D.A. APHIS make every attempt to avoid the same problem that resulted in 1977 and void the awarded 1983 contract predicated upon the performance evaluations.

It was also recommended that representatives of the U.S.D.A. collaborate with members of the U.S.A.H.A. Committee on Livestock Identification to provide revised specifications for such means of identification which will provide additional assurance to the livestock industry and to the state and federal regulatory agencies.

RESOLUTION No. 23  
Source: Epizootic Attack Plans Committee  
Subject Matter: Eradication or Controlling heartwater from the Western Hemisphere  

Resolution  
BE IT THEREFORE RESOLVED, that increased funds and efforts by APHIS be directed toward controlling and eradication heartwater from the Western Hemisphere.

RESOLUTION No. 24  
Source: Foreign Animal Disease Committee  
Subject Matter: Swine Repopulation in The Dominican Republic and Haiti  

Resolution  
THEREFORE BE IT RESOLVED, that the United States Department of Agriculture, Agency for International Development, Canadian Department of Agriculture, Canadian International Development Agency and other such agencies be urged to facilitate the repopulation of swine in the current African Swine Fever programs in The Dominican Republic and Haiti.
RESOLUTION No. 25
Source: Foreign Animal Disease Committee
Subject Matter: Veterinary College Curriculum—Foreign Animal Diseases

Resolution

BE IT THEREFORE RESOLVED, that the USAHA requests that, The American Veterinary Medical Association Council on Education develop criteria to be added to the requirements for accreditation of veterinary colleges to assure that those receiving the degree of Doctor of Veterinary Medicine from these accredited educational institutions are adequately trained in foreign disease.

RESOLUTION No. 26
Source: Professional Oversight
Subject Matter: Personal Liability of Governmentally Employed Personnel

Resolution

BE IT RESOLVED, that the USAHA urges each state to take all measures possible to protect state/federal employees working in each respective state against personal liability litigations involved in the proper enforcement of their defined responsibilities.

BE IT FURTHER RESOLVED, that USAHA urge USDA and the Department of Justice to take whatever measures are required to protect federal employees carrying out their official duties and responsibilities required for animal health programs against personal liability litigations in federal and state courts; and

BE IT FURTHER RESOLVED, that the President of USAHA appoint a sub-committee composed of state and federal employees to investigate the matter and develop suggested solutions.

RESOLUTION No. 27
Source: Morbidity and Mortality Committee
Subject Matter: Statistically Valid Frequency rates, Food Animal Diseases

Resolution

THEREFORE BE IT RESOLVED, that the APHIS, USDA plan on animal disease surveillance, as outlined, be instituted as a pilot phase in 1983 in 2 states and continue to maintain the schedule until finally fully implemented on a nationwide basis in 1990.
AMENDMENT TO THE BYLAWS OF THE
UNITED STATES ANIMAL HEALTH ASSOCIATION

Tuesday, November 9, 1982
Nashville, Tennessee

The following amendment, properly presented and acted upon by the Executive Committee in session in St. Louis, Missouri, on October 14 and 15, 1981, is now brought before this organization for affirmative two-thirds vote for incorporation into the ByLaws of the United States Animal Health Association:

#1. Strike the last two words, “Said Organization,” on line 32 and insert: “Such organizations applying for membership;” further, after the word “have” on line 33, insert the following: “and shall continue to maintain.”

#2. After the word “Constitution” on line 239, insert the following: “and ByLaws.”

#3. Change the words “twenty dollars ($20.00)” on lines 309 and 310, to “thirty dollars ($30.00);” and the words “one hundred fifty dollars ($150)” on lines 315 and 316, to “two hundred dollars ($200).”
PROPOSED AMENDMENT TO THE CONSTITUTION AND BYLAWS OF THE UNITED STATES ANIMAL HEALTH ASSOCIATION

Thursday, November 11, 1982
Nashville, Tennessee

The Constitution and ByLaws are amended as follows:

Wherever it shall appear in the Constitution and ByLaws, strike the term "Executive Committee" and substitute the term "Board of Directors;" and wherever it shall appear in the Constitution and ByLaws, strike the term "Board of Directors" and substitute the term "Executive Committee."
FORGING A NEW PARTNERSHIP

I wish to thank President Rea and the U.S. Animal Health Association for this opportunity to discuss with you some matters of special, mutual importance in these days of serious budget constraints. This Association performs a vital service by providing a forum for State, Federal and private industry people to get together and share their concerns about animal health and the need to protect this country’s agriculture.

My topic is “Forging a New Partnership.” We’ve long known at APHIS that things get done when all of us—you and me—are working together with a purpose. But knowing is not always doing. The spirit of cooperation can sometimes wear thin. Before you know it, we’re not sharing our concerns... not mutually striving for common goals. But you can be sure, no effort to control an animal disease—or especially to eradicate it nationally—will ever work without a partnership approach.

We—APHIS—pledge ourselves to work more closely with you—the representatives of industry, the States and the scientific community—at every stage of our future actions from planning... to execution... to assessment. But to carry this off, we’ll need a reciprocal commitment from you. I’d like to relate to you some events that helped us to appreciate the need for a new partnership.

IMPACT OF PROPOSED BUDGET CUTS

When first announced back in January, the Administration’s proposed budget for Fiscal Year 1983 and beyond had dramatic repercussions—not only on Veterinary Services but the entire APHIS organization. Proposed reductions for Plant Protection and Quarantine programs were extensive. In Veterinary Services, brucellosis, along with animal welfare, cattle ticks, tuberculosis, emergency programs, and horse protection, stood to take some deep cuts. Several PPQ programs, as well as scabies, were to be eliminated.

We were immediately concerned that the cuts could endanger our ability to monitor interstate movement of animals, keep out exotic disease, provide adequate laboratory support, and do other essential things expected of us.

I’m sure everyone in this room agrees that APHIS must be prepared to quickly find and deal with an outbreak of a foreign animal disease such as foot-and-mouth disease, rinderpest, African swine fever or hog cholera.
And we need to assure importing countries throughout the world that our animals and products are healthy and safe—thus protecting our sizable export markets and improving the U.S. balance of trade. Our agricultural export market, in animal and animal products alone, was valued at slightly more than $4 billion in 1981.

To come to grips with these concerns, however, we needed a reappraisal. We needed to better define the requirements of an effective national animal and plant protection program—"a basic organization." This was undertaken, in part, by an internal study. But we also sought some outside help.

LUHRS TEAM CONSULTS INDUSTRY AND GOVERNMENT LEADERS

We contracted for an independent study by Caro Luhrs Associates. Dr. Luhrs is a Georgetown Medical School faculty member and former director of Booz-Allen and Hamilton's Health and Medical Division.

The Luhrs team came up with some additional recommendations ... beyond those that came out of our own study. But we felt very comfortable with their recommendations because they coincided nicely with how we perceive our role.

Begun in April and concluded in July, the Luhrs study was based on comprehensive review of pertinent documents and in-depth interviews with well over 125 knowledgeable individuals. They included officials of the Department, industry, States, international organizations, foreign governments, other Federal agencies, and OMB, many of whom have been involved with successful programs of disease control.

Let me briefly discuss the Luhrs' findings generally, and then we'll look at some findings in a little more detail.

Of the people interviewed by Luhrs, there was unanimous agreement that APHIS is doing a good job. Obviously, some were critical of certain programs that they'd like to see handled a little differently. But when you brush away the small critiques of specific programs, everyone reacted favorably to what APHIS was doing, why we're doing it, and—more or less—how we're going about it.

That's reassuring and important to know. But the Luhr's study also pointed out that, even if we got the same amount of money for fiscal '83 as we did in '82, we'd still have an inflationary loss for '83. Our needs aren't static, and even the same funding level would buy discernibly less for this year.

PARTNERSHIP EVEN MORE IMPORTANT NOW

This means the Administration is indeed expecting industry and the States to continue picking up a larger share of the program costs in which
they have a strong interest and commitment. This applies whether a program is carried on in control form or is being pressed to a successful conclusion through eradication. If we all can agree that a program should be carried forward, reduced Federal spending can be mitigated to a great extent by increased State of industry support and should permit achieving our common objective.

As I see it, the partnership approach we've always encouraged is more important now than ever before. And I can assure you that where APHIS might have been perceived occasionally in the past as not working closely enough with the States and industry, we will most certainly not let this happen in the future. We value the assistance of outside counsel, and we especially welcome your advice and consultation.

APHIS BASIC FUNCTIONS AND ORGANIZATION

Our internal study had previously identified five functions considered essential to the protection of American agriculture and requiring a strong Federal role. They are: port-of-entry interception; domestic surveillance and detection; laboratory and technical support; emergency response capability; and international cooperation and coordination.

The Luhrs team found these functions to be consistent with our primary mission which is "to protect the animal and plant resources of the Nation from diseases and pests." And, after careful study, they found few opportunities to reduce the APHIS role if the Nation is to continue having effective agricultural health protection.

They further said that APHIS should neither reorganize nor budget along functional lines. What they're saying is, Congress is not going to be receptive to a proposal that only talks about five basic functions. So what we'll be doing in the future—and what we've already done in our presentation to the Secretary for 1984—is to use a program/functions matrix to develop two budgets.

In the presentation to the Secretary, we described what we'll need to fulfill our basic obligations to agriculture for 1984. That gives us the number of people and the kind of resources we need to fulfill our role. It also sets a kind of threshold, below which we begin to compromise the ability of APHIS to do its job.

Individual programs were then reviewed to establish priorities and obtain a combination of activities which would meet the basic requirements of an effective system of agricultural protection. For example, the brucellosis program contributes to several of APHIS's essential components, such as maintaining adequate disease surveillance and detection—not just for brucellosis, but for other diseases as well.

Another program may have a bigger component, say, for laboratory and technical support. While we maintain the laboratory for one par-
ticular program, it's also available for a variety of other activities. So in all of our programs, we can identify parts of each that also really are contributing to our basic organization.

That's the position we've taken in our 1984 budget presentation to the Secretary. And, we have made the same presentation to the Office of Management and Budget. We want OMB people to understand that they simply cannot cut this program or that without considering what the impact might be on a whole spectrum of APHIS activities. We intend to establish with them the concept of a basic, minimum funding level for APHIS.

But when we go to Congress, we'll be talking about the brucellosis program ... the gypsy moth program ... and so on. That's because Congress wants to exercise control over these individual programs; and we have no problem with that, as long as what the total arrived at permits us to fulfill the needs of our basic organization.

CONGRESSIONAL PARTNERSHIP

The Luhrs study, by-the-way, drew favorable comment by Congress. The report by the Senate Appropriation Committee said, in part, and I quote: "This Committee concurs with the position of the House that the results of this study are in line with the views and intent of Congress."

This reaction by Congress is encouraging. After all, we are also in partnership with Congress—working with committees and individual Congressmen to help assure the success of animal and plant health programs. Their ideas reflect the views and attitudes of constituents—the farmers and ranchers of their home districts and States.

AD HOC GROUP REVIEWS BRUCELLOSIS PRIORITIES

In May, soon after the Luhrs study began and without reference to previous "in-house" APHIS budget planning, we convened a special ad hoc review group to look at the elements of the brucellosis program and to assist us in establishing program priorities. The reduced level of funding for 1983 has made this consultation imperative. The team consisted of APHIS people outside the brucellosis staff, plus four people from outside the agency.

We were very pleased to have these distinguished participants from industry: Burt Eller of the National Cattlemen's Association, John Adams of the National Milk Producers Federation, Al Keating of the American Farm Bureau Federation, and Dr. John Shook, Maryland State Veterinarian, but also representing USAHA. In the course of fulfilling his role on the review group, Dr. Shook consulted with a number of his colleagues in USAHA.

Although some of the industry members of the review group said their organizations did not support the proposed funding reduction for
brucellosis, all were willing to work with APHIS to establish priorities for spending the money that would become available as of October 1 of this year.

The group took a long, hard look at the problem. In their deliberations, they worked with the overall elements of the brucellosis program and did not attempt to make any State-by-State allocation of resources. Rather, they attempted to determine: what basic functions in the eradication effort must be fully funded; what functions are less critical; and what functions or elements could be reduced or responsibility for their completion turned over to the States and affected industries.

MEETINGS WITH STATES AND NASDA

In August, a meeting was held with officials of Florida, Mississippi, and Texas to discuss the rationale for the allocations to individual States. Subsequently, our regional Veterinary Services directors conducted meetings with officials in all other States to provide the same information and opportunity for discussion.

We recognize that more effort or emphasis to control and eradicate brucellosis in the higher-incidence States will help reduce spread to the low-incidence and free States. I might point out that 13 States and the U.S. Virgin Islands are now free of brucellosis . . . meaning no field strain infection for at least one year. Another 24 States and Puerto Rico are Class A States with herd infection rates of 0.25 percent or less.

In September, Dr. John Atwell, Deputy Administrator, Veterinary Services, attended the National Association of State Departments of Agriculture meeting in Des Moines to get that organization's views on an alternative method of using and distributing Federal funds for brucellosis eradication in FY '83.

Meetings and discussions such as those cited above have been invaluable in helping APHIS work its way through some difficult program decisions necessitated by fiscal '83 budget constraints. But these events have also shown us how important these partnership reviews are, and we are determined to use them more in the future.

MORE FLEXIBILITY FOR FIELD STATIONS

In the past, I’ve heard the word “inflexibility” associated with APHIS or APHIS programs, and I intend to do something about it. State officials and others have identified our Work-Based Budgeting System, or WBBS as it’s called, as a culprit. All too frequently, WBBS has been invoked as a reason for not doing something saying words to the effect, “Sorry, but we don’t have any dollars in our WBBS budget for that.”

That kind of inflexibility simply doesn’t buy us anything. We must give our regional and area leaders more discretion, within their professional judgement, to decide how their resources can best be spent.
We must identify key components of our programs, determine funding levels for each, and then set up meetings between the regional, area and State people to carve out some discretionary monies. In that way, we can get away from some of the constraints of WBBS.

CONCLUSION

One of the severest tests of an organization's effectiveness is the verdict of its clients on its performance. We have reviewed the criticisms of the past and have had to rethink our budget planning and work approach. Several things emerge:

We—APHIS—need to recreate or reinforce the cooperation we've enjoyed over the years with industry and the States. We need to consult more—a la the brucellosis policy review committee and scrapie committee—with everyone having a voice in the matter before a decision is arrived at.

We may have to guess, to some extent, about funding levels; but we'll determine not only direction, but also how best to implement the Federal role... the State role... the industry role for various APHIS programs. We'll even plan at what point there should be a follow-up evaluation of how well it's working and whether to rethink the direction.

And we'll be working harder on the APHIS attitude. Specifically, we will be encouraging an attitude that takes into account fully the wishes and capabilities of the industry when it comes to adopting technology for animal disease and control or eradication activities.

Finally, though we will undoubtedly continue to need a WBBS-type system to help with our accounting, we will make every attempt to avoid using it as an excuse for not cooperating on animal health activities that State, Federal and industry people agree need to be done.

That's what I mean by forging a new partnership. We intend to do more than ever to give meaning to the word "cooperation." We are reviewing our commitment to work side by side with State officials and industry representatives to achieve our common objectives in each of our animal and plant health programs, whether it be for effective control or eradication.
REPORT OF THE ANIMAL WELFARE COMMITTEE

Chairman: E. Mickey Stewart, Lincoln, NB
Vice Chairman: Neal Black, St. Paul MN

R. K. Anderson, MN; R. R. Bowen, NB; G. C. Cilley, NH; Oscar Clabaugh, KS; A. E. Decoteau, MA; B. H. Ewald, VA; M. W. Fox, D.C.; H. M. Frederick, VA; Ann Gonnerman, MO; Carl Graham, MO; T. M. Gustafson, NB; G. A. Hall, OK; R. J. Lee, VA; M. R. Levy, NJ; Arnett Matchett, MD; D. J. Meisinger, IA; W. T. Nagao, Hawaii; C. J. Nelson, CO; D. H. Person, SD; Ronnie Polen, NJ; D. C. Randall, CO; R. A. Rice, NC; J. D. Roswurm, CA; D. F. Schwindaman, MD; K. W. Scritchlow, IL; L. M. Siegfried, WI; M. S. Silberman, GA; Christine Stevens, D.C.; R. M. S. Temple, OH; Max Van Buskirk, PA; F. D. Wertman, IA; E. J. Wilson, MD.

The Animal Welfare Committee was called to order by the Chairman, m. E. Mickey Stewart at 1:30 PM on Tuesday, November 9, 1982. There were 19 committee members and 22 guests present.

The Chairman introduced the committee members by a roll call. Comments were requested on the published minutes of the previous meeting. There being none, the tentative agenda previously mailed to committee members was read and followed.

Dr. Dyarl King, USDA, Agricultural Research Service, presented a brief update on the USDA research projects funded by USDA with $380,000 in late 1981. The projects are farm animal stress related research involving modern livestock husbandry systems. Although the research is being continued, one significant factor is known which is that all stress cannot be removed entirely or the animals system will not remain in an adaptive state.

Mr. James A. Mallman from Provini, Inc., presented a short summary of the findings from recent research they have conducted. The purpose of the study was to determine the adaptability of the loose housing system used in Great Britain for commercial raising of veal calves to the United States. A video tape of the field trial facility in Watertown, Wisconsin was shown. In summary, Mr. Mallman stated that, at this time, the results of the research are very inconclusive, but are encouraging enough to continue the project.

Ms. Diane Halverson representing the Animal Welfare Institute, Washington, DC, gave a report and presented slides describing her recent visit to 7 European countries to look at the farm animal welfare situation. She had visited about 45 scientists and farmers who are studying or using substitutes for intensive methods of keeping swine, laying hens, and veal calves. Ms. Halverson highlighted examples of research and livestock husbandry systems in use which she described as representing behaviorally appropriate and economically feasible methods of husbandry.
Dr. Stan Curtis, a swine researcher from the University of Illinois, reported on a recent visit he had made to the Northern European countries of England, Scotland, Denmark, Sweden, West Germany, Switzerland, Netherlands, and Belgium. During the 4 week trip, he visited with about 85 people representing humane activists, farm groups, government officials, and scientists. Dr. Curtis observed that the less intensive livestock management systems being promoted by several more radical animal welfare and animal rights groups in this country are not in fact, being used to any extent in Europe. It is his feeling that other than the two countries, Sweden and Denmark, outside the European Economic Community, any uniform laws or regulations requiring changes related to farm animal welfare are 10, 15, or 20 years in the future.

After the above four reports, short discussions were held concerning animal inspections in research facilities, pending animal welfare related legislation and amendments and USDA enforcement of the Animal Welfare Act.

Six resolutions were introduced for consideration. After discussion and voting, four were passed for consideration by the Resolutions Committee.

Mr. Neal Black discussed the educational efforts taking place by a number of agricultural organizations to educate school age children about food animal production and farm animal welfare.

The committee meeting was adjourned at 5:00 PM.

Committee Members in Attendance were:

E. Mickey Stewart, Chairman—Lincoln, NB

Neal Black, Vice Chairman—St. Paul, MN

R. R. Bowen, NB; G. C. Gilley, NH; Oscar Clabaugh, KS; H. M. Frederick, VA; Ann Gonnerman, MO; Carl Graham, MO; R. J. Lee, VA; D. J. Meisinger, IA; W. T. Nagao, Hawaii; D. C. Randall, CO; R. A. Rice, NC; J. D. Roswurm, CA; D. F. Schwindaman, MD; Christine Stevens, DC; Max Van Buskirk, PA.
REPORT OF THE COMMITTEE ON EPIZOOTIC ATTACK

Chairman: B. W. Hawkins, Ontario, OR
Vice Chairman: H. A. McDaniel, Silver Spring, MD

John Adams, D.C.; J. B. Anderson, TN; R. A. Bankowski, CA; Neal Black, MN; J. L. Blair, VA; W. O. Boaz, TX; W. W. Buisch, MD; Ramsay Burdett, OR; M. J. Burridge, FL; S. J. Cougar TX; R. O. Drummond, TX; Joe Finley, Jr., TX; W. C. H. Glaze, TX; J. H. Graves, NY; F. A. Hayes, GA; P. R. Henry, CO; J. L. Hyde, MD; E. T. Mallinson, MD; N. L. Meyer, VA; M. A. Mixson, AL; T. G. Murnane, DF; J. E. Novy, TX; J. S. Orsborn, CA; B. I. Osburn, CA; T. B. Ryan, NC; E. C. Sharman, MD; W. L. Sippel, FL; G. F. Slonka, TX; Kenneth Thomsen, WA; M. J. Tillery, MD; M. A. Van Buskirk, PA.

Chairman Bert Hawkins called the Epizootic Attack Committee to order at 1:30 p.m., November 11, 1982. The continually increasing interest in epizoological assessment reports and discussions were manifest by the more than 65 members and guests present.

Dr. P. R. Henry presented a vividly illustrated discussion of the vesicular stomatitis outbreak in 1982. Concern was expressed for lack of information provided during this outbreak.

Chairman Hawkins summarized the highlights from the recent meeting of the USDA Secretary’s Advisory Committee on foreign livestock and poultry diseases. He endorsed the initiative to examine more closely the international issue relating to exotic animal disease, but expressed reservation for any actions that might increase vulnerability to foreign diseases and pests.

Dr. Dan Goodwin's presentation described and illustrated a most serious outbreak of malignant catarrhal fever (MCF) in The Oklahoma City Zoo. The source of the outbreak appeared to be animals from the San Diego Zoo or Game Park.

Dr. Scott Reynolds expressed concern for cattle grazing land being used for rearing African type animals which might contaminate the premises and jeopardize future cattle production on the same premises. Dr. Reynolds also presented some excerpts from material prepared by Dr. Werner Heuschele for this committee including the fact that 50 clinical cases of MCF have occurred in the San Diego facilities since 1974.

A resolution expressing concern for MCF was prepared prior to the meeting in response to a request from The Texas Animal Health Commission. We were informed this resolution had already passed in the committee on zoological Animals and had been accepted by the Committee on Resolutions, but nevertheless a motion was made, seconded, and passed, for this committee to co-sponsor the resolution.

Dr. Frank Hayes called attention to an effort being made to bring African animals onto St. Katbern Island off the coast of Georgia.
Dr. Al Smith presented a most informative paper on Caliciviruses in a wide range of marine and terrestrial animals. He expressed concern for restrictions on the movements of animals and products due to their pathogens.

Dr. Frank Hayes presented evidence for drawing attention to the danger posed by wild animals to domestic animals and visa versa. Wild swine on the west coast might be the avenue for calicivirus entry onto the mainland and likewise wild swine in parts of Texan bordering on Mexico could serve as hosts for the entry of hog cholera from Mexico.

Dr. Hayes' vivid portrayal of the confusion and conflicts that arose between people with different interest toward wildlife during an effort to alleviate suffering in deer when the Florida Everglades flooded was well received. Dr. H. L. Meyers moved the committee express a vote of thanks to Dr. Hayes for paving the way for cooperation between wildlife and domestic animal interest. A standing ovation for Dr. Hayes conveyed the overwhelming expression of gratitude.

Dr. William Buisch summarized activities and accomplishments in Emergency Programs during 1982. Although many problems were dissolved during a test exercise, this was the purpose of the exercise. During this exercise the most pressing problems disclosed concerned legal actions whereas in earlier test exercises problems were more technical. Dr. Buisch reported progress was being made toward eradication of African Swine Fever in Haiti. Approximately 87,000 swine have been depopulated out of an estimated population of 300,000. Depopulation is expected to be completed by May or June 1983. Plans and funds for repopulating swine in Dominican Republic and Haiti need more attention. Dr. C. D. Van Howelling, speaking for the National Pork Producers expressed encouragement for the repopulation of both countries, but also mentioned that all problems with international agencies had not been resolved.

Dr. Norvan Meyers explained that APHIS has 99 employees stationed outside the U.S. More positions are being added for veterinarians.
REPORT OF THE COMMITTEE ON FOOD
ANIMAL HYGIENE

Chairman: D. M. Bedell, Tifton, GA

Vice Chairman: P. J. Friedman, Richmond, VA

A. F. Bailey, OK; J. A. Bell, NC; L. G. Billingsley, CA; J. L. Blair, VA; A. D. Bond, D.C.; D.C. Breeden, NB; W. H. Dubbert, VA; G. B. Estes, VA; T. M. Gustafson, NB; R. E. Hall, WI; C. S. Johnson, TX; J. C. Leighty, MD; T. E. Liner, TX; J. L. McMillian, CA; H. O. Miller, IL; C. S. McCain, WA; J. K. Payne, D.C.

The Food Animal Hygiene Committee heard reports on

1. The Cooperative Residue Avoidance Program presented by Dr. Wayne Weber, Residue Evaluation Planning Staff, USDA.

2. Report on the Codex Alimentarius Committee on Meat Hygiene by Dr. W. H. Dubbert, FSIS, USDA.

3. Report on problems of meat rabbit processors was presented by Mr. Fred Martin, Prairieville, LA., and Mr. Eugene McMorris, Summit, MS.

Dr. Weber discussed the Cooperative Extension Service involvement in the Residue Avoidance Program. The main thrust of the program at this time is the seeking of data and education of the producer. Thirty-eight (38) states are involved in the program.

Dr. Dubbert discussed the Codex Committees code of practices for the slaughter of game animals and the code of practice for ante mortem and post mortem inspection of domestic animals.

The meat rabbit industry representatives are concerned about the competition of uninspected rabbit meat being imported from China. Purported differences in quality are points of concern influencing buyer acceptance of domestic inspected product.

The committee discussed the possible impact of including domestically produced rabbit meat under the Wholesome Meat Act as well as requiring imported rabbit meat to be subject to equal inspection. The Food Animal Hygiene Committee feels that more information on the volume of this young industry is needed before a position can be determined. The industry will be invited to supply more information on the subject for review by the committee.

I move that this report be referred to the Executive Committee for approval.
REPORT OF THE COMMITTEE ON INFECTIOUS DISEASES OF HORSES

Chairman: C. L. Campbell, Tallahassee, FL
Vice Chairman: R. C. Knowles, Silver Spring, MD

J. B. Anderson, TN; C. E. Boyd, SC; G. C. Cilley, NH; Jesus Castaneda G., Venezuela; LeRoy Coggins, NC; C. S. Duncan, NY; W. W. Clark, TX; G. B. Estes, VA; P. M. Epple, AL; R. C. Goulding, CA; A.M. Gallina, WA; J. B. Healy, CA; F. M. Jones, APO, Miami, FL; M. J. Kemen, NY; W. O. Kester, CO; W. W. Kirkham, IN; M. J. Nolan, D.C.; S. R. Nusbaum, NJ; M. A. Owen, MA; W. E. Pace, FL; D. D. Philson, FL; Victor Schroeder, DF; John Smiley, ME; J. D. Smith, KY; M. B. Tiegland, FL; C. D. Vail, CO; T. E. Walton, CO

The Committee on Infectious Diseases of Horses convened in Nashville, Tennessee, on November 8, 1982, with approximately 30 committee members and guests present. Topics of note covered by this committee were equine encephalitis, equine infectious anemia, contagious equine metritis and salmonellosis problems in horses.

Dr. James Pearson summarized the Venezuelan Equine Encephalitis surveillance activities carried out by the National Veterinary Services Laboratories (NVSL) for the calendar year 1981 and 1982. In 1981 samples were examined from 803 horses clinically exhibiting signs of encephalitis. Among these, 328 were confirmed as western encephalitis, 42 were confirmed as eastern encephalitis; No cases were confirmed of the Venezuelan type. In 1982 the summary of encephalitis cases confirmed at NVSL were 26 western encephalitis and 46 eastern encephalitis among 454 samples submitted to the laboratory during the period of January 1—September 30 of that year. No Venezuelan encephalitis was confirmed in 1982.

Equine Infectious Anemia (EIA) official test results were reported for Fiscal Year 1982 (October 1, 1981—September 30, 1982). Among 709,108 tests conducted, 4,234 positive tests were revealed, which represents approximately 2,117 infected horses—since nearly every positive coggins test is repeated to ensure the states of the positive animal. (See exhibit #1)

Dr. Ramsey Burdette summarized the E.I.A. control program that is underway at the Warm Springs Indian Reservation in Oregon. In 1981, 2,808 horses were tested revealing 734 positive animals. In 1982, initial and retested animals totaled 1,274 revealing 10 positive animals. Testing is continuing on this Indian Reservation and in an adjacent Bureau of Land Management herd to eliminate EIA.

Dr. James Pearson presented his experience with the Pitman-Moore developed enzyme-linked immunosorbent assay (ELISA) test (directed at detecting antibodies) as applied to E.I.A. Investigation of the potential of the use of the ELISA test have been principally limited to laboratory evaluation of this test, especially side by side with the A.G.I.D. test. At
this time, in Dr. Pearson’s experience and view, the ELISA test has these features:

1. Awkward, in that it requires 80% of one technician’s time for 2½ hours to run 30 samples—as compared to the average of 200 AGID tests performed in one day.
2. Reading is visualization of the reaction but a spectrophotometer can be used.
3. Appears to be not more sensitive than the AGID test (in general, ELISA tests are expected to be highly sensitive).
4. More work needs to be completed before field application would be practical.
5. It may be possible to automate this test to perform 500 tests daily.

Contagious Equine Metritis (CEM) continues to spread in certain parts of Europe. The latest country to be reported as affected since our 1981 report in Sweden. This makes a total number of 12 countries reported to be affected with CEM.

Certain states have become concerned with the practices presently employed in surveillance and precautionary measures that are being taken to prevent the spread of CEM.

An item entitled CEM alert was read to the committee as follows:

CEM Alert

1. Equine Industry is being falsely assured that clean up program on imported stallions will protect against CEM. Racing stallions that come in are not cleaned up in all cases and yet may breed 2-3 mares before racing.
2. Imported horses declared to be coming in for racing purposes are immediately advertised for stud services i.e. ($45,000 live foal). Horses can be diverted from racing with no U.S. follow up.
3. Import certificates do not always say what the animal will be used for.
4. If a restricted mare comes into State A and then shipped on to State B, State B does not know this was a restricted animal and have all necessary clean up tests been made. Infection may be in other parts of the body.
5. Many veterinarians do not believe that just removing the clitoris and clitoral fossa assures that all infection from the mare may have been eliminated.
6. Foreign import shippers are laughing at U.S. requirements. They say they know some racing stallions have bred mares before coming to U.S.
7. V.S. traceback on suspicious animals, because of a U.S. infected
animal, is very frustrating. Some lists are received at 3 different times.

8. We are not getting necessary information to follow up on movements from infected animals. No documentation of test results, epidemiology and present status of investigations are being received from V.S. or State involved. A multi-million dollar horse breeding farm will not cooperate if they aren't given documentation justifying culturing and breeding. States are put in a very tenable position and possibly open to lawsuits because of not advising the industry and other State officials.

Certain members of this committee are concerned that the present measures being taken to prevent spread of CEM in the domestic horse population are fraught with danger and such measures should be reviewed. We recommend that the Committee on Infectious Diseases of Horses of the USAHA take the initiative to gather the scientific/regulatory community together to meet on CEM to assemble data of pertinence on this disease—then arrange a meeting with the horse industry of the United States to chart the course that should be taken in dealing with CEM.

The committee wishes to admonish the U.S.D.A. Veterinary Services for not having an official representative present at this committee meeting to inform members on the status of infectious disease problems of domestic horses and contingencies related to the importation of horses into the United States. While we appreciate Dr. James Pearson's participation, many other items covered by the committee were not within the purview of his work.

The matter of Salmonella infections in horses was discussed and a Salmonella Subcommittee was appointed to define these problems and make recommendations to the Infectious Diseases of Horses Committee.
INTRODUCTION:

There is general agreement on the need for a national animal identification system for regulatory and census purposes. There is not general agreement, however, that the benefits derived would be worth the cost and bother of such a system at the present time. It is thought also, that temperature monitoring coupled to the identification system might increase the benefits of such a system. The body temperature of animals is one of the most useful measurable parameters to determine the physiologic and health status of animals. However, the cost-effectiveness and reliability of the remote monitoring of temperature is still in question.

We are presently studying body temperatures of dairy cattle using an active radiotelemetric system. I would like to present some of our data and discuss our results as they relate to livestock management and to the design and use of a passive temperature monitoring system.

The study we present could have been done in other species of livestock in any environment but our interest is in dairy cattle, particularly on large farms in hot climates. I'm sure many of you are familiar with the trend in the dairy industry of the last 3 decades to fewer but larger dairies, particularly in the southern U.S. This trend has been especially strong in Hawaii where the average herd size is over 600 cows per dairy. The large dairies have increased problems of management, which are particularly aggravated by the hot climate in the southern U.S., Puerto Rico, Hawaii, and much of the tropical and subtropical world. Reproductive problems, for instance, become much more acute in large dairies in hot climates. I believe that the situation of the large dairies in hot climates has come upon us so fast that we have not had time to react and to have all the answers on the proper management of livestock in this type of environment.

The active temperature monitoring system is being used to study the temperature patterns of cattle to develop strategies for the management of livestock, particularly in hot climates. We submit that these studies are necessary for the design and use of any future electronic (passive or active) temperature monitoring system to be linked to automated systems for management of livestock.
METHODS:

These studies were conducted on the University of Hawaii's Animal Science Research Farm located on the North Shore of Oahu. Holstein-Friesian cows from the 100-cow dairy were housed primarily in cement drylot pens, and were fed silage made from chopped pineapple plants and grain concentrates.

The telemetry equipment used was a computerized system for data collection, storage, and analysis, as previously described (Araki et al., 1983). Vaginal transmitters (Telonics, Inc., Mesa, Arizona) were used to monitor animal temperatures and proved to be highly reliable.

RESULTS:

Various aspects of these studies can be presented in excerpt form:

1. Effect of lactation on cattle temperatures.

   In a comparison of cattle in different states of lactation, vaginal temperatures were obtained from three groups (5 animals per group) of cows. In Figure 1, the daily mean temperatures of the three groups are presented: (A) early lactating animals, less than 100 days after calving, (B) late lactating cows, greater than 240 days post-partum, and (C) dry cows. Lines D and E represent daily mean environmental temperatures, blackglobe and drybulb temperatures, respectively. It can be seen that lactating cows had consistently higher temperatures than dry cows and that these differences were especially pronounced on hotter days.

   The differences among groups can be better defined by looking at the underlying rhythm which gave rise to the daily mean temperature for a group and comparing patterns between groups. For example, temperature patterns of the early and dry groups can be compared and analyzed by t-tests at each sampling time and over the entire 17 day period of this experiment (Figure 2). The dots represent each time in a 24-hour period that the early group temperature was found to be significantly higher than the mean temperature of the dry group. These results indicate that: (1) when there were significant differences in temperatures between the two groups, the temperatures of the early lactating group was always higher, (2) there was a clustering of significant differences around certain times of the afternoon and (3) higher temperatures of the early lactation group persisted throughout the night time hours. It must be noted that this early lactating group with the highest temperatures, is the very group of animals that are being bred in most dairy operations. The higher body temperatures of this group may contribute to the difficulty in breeding cows in hot climates.

2. Diurnal temperature patterns.

   Further examination of diurnal temperature patterns shows the highest temperatures in the afternoon with a gradual drop during
the evening. The lowest temperatures were seen in the early morning hours with temperatures again rising during the mid to late morning. In examining temperature patterns of lactating cows, a dip in temperature was seen 30-60 minutes following milking which suggests that cattle may be cooled efficiently during milking.

3. Cattle cooling experiments.

To determine how effectively cattle may be cooled during milking, modifications were made to the milking parlor and the milking routine. Window fans and garden misters were installed in the pre-milking holding area. Fans were directed on cows in the milking stalls, and fans and garden sprinklers were used to cool cows in the post-milking holding area. Figures 3 and 4 represent the temperature pattern of 2 cows that were cooled (lower tracing) and 2 control cows that were not cooled (upper temperature tracing) during milking. The hatched lines indicate the cooling period. Precipitous drops of up to 1.5°C in temperature could be seen in cooled cows which were not seen in control cows.

4. Figure 5 represents the temperature pattern of a cow selected to depict estrus. The points represent vaginal temperatures recorded every 15 minutes for nearly 3 days; (A) the day before estrus, (B) the day of estrus, and (C) the day after estrus. Temperatures on the day of estrus began rising at 0300 hrs., peak temperatures at about 1200 hrs. were 0.5°C higher than normal, and gradually descended to normal temperatures at 2200 hrs. This pattern was easily distinguished from the normal non-estrus diurnal pattern seen in dairy cattle.

5. The temperature pattern of a cow during parturition is shown in Figure 6. The upper tracing is of the temperatures recorded on the 5th and 4th days prior to parturition. The lower tracing shows temperatures on the day before and the day of parturition. The vaginal temperature started to fall at 1400 hrs. on the day before calving and gradually descended till 0800 hours of the day of calving. A steady rise was then seen until parturition which occurred at 1730 hrs. (arrow).

DISCUSSION AND SUMMARY:

Temperature telemetry is an exciting tool which adds a new dimension in animal studies for the development of new strategies for livestock management. I'd like to summarize by reiterating our results in terms of management, list some other ways in which temperature telemetry can be used in livestock management, and cite the implication of our results to the use of a passive temperature monitoring system.

1. The results of our studies indicate that it may be advantageous to milk the high lactating cows, the group being bred, at the hottest times of the day since they appear most susceptible to heat stress and the milking procedure provides some cooling effect. Our data
also suggest the strategy of maximally utilizing the milking house to cool cattle in hot climates. Milking areas could be redesigned with larger cow holding areas equipped with cooling before and after milking for better production and reproduction in hot climates.

2. Estrus detection by telemetry, if practical, would be very beneficial to dairy husbandry particularly in hot climates where the estrus may be shortened and less pronounced. It may also find application in beef cattle and swine operations. Monitoring parturition by temperature can be useful in horses, cattle, and swine.

3. Some perturbations of the temperature patterns may have been due to feeding. Time of feeding and the type of ration (roughage and grain concentrates) may have important effects on animal temperatures and the animal's ability to handle heat stress. If so, alterations in feeding during hot weather could significantly reduce the level of stress in animals.

4. Early detection of disease may be an important use of temperature telemetry. For example, earlier detection and treatment of mastitis in dairy cattle may reduce damage to mammary tissue and decrease the permanent effect on lowered production seen so often following mastitis. Another area often neglected is the early post-partum metritis frequently accompanied by cervicitis and salpingitis. The difficulty of differentiating post-partum metritis from normal involution results in the postponement of detection and treatment till 30 days after parturition. This neglect may seriously impair reproduction and perhaps permanently reduce an animal's reproductive efficiency. In dairy cattle, a high rate of endometrial infection and poor reproductive efficiency is known to be more severe in hot climates.

5. It will be of practical and academic interest to equate temperature changes with changes in hormonal levels in reproductive events such as ovulation, estrus and parturition. A critical evaluation of the hormonal responses to heat stress is also of interest in hot climates. Studies of this nature are being contemplated in our laboratory.

6. Studies of temperature patterns of dairy cattle in hot climates may eventually lead to the identification of criteria for selection of heat adaptable cattle. Heat tolerant cattle, capable of good reproduction and adequate production, would be desirable for use in hot climates of the world.

7. Telemetry studies may be expanded to include beef cattle and swine. Ear canal transmitters may also be more useful in these studies. Future studies may also include other measurable parameters, such as heart rate, respiratory rate, and uterine motility to better define the effects of management on livestock.

Our results indicate that if reliable animal body temperatures can be obtained, there is substantial commercial application in the management
of livestock. But because its usefulness depends upon recognizing an abnormal temperature, more work is required to define the normal temperature patterns of livestock and to determine what kind of perturbations are caused by different animal conditions. If a passive "temperature on demand" system is to be used commercially, normal temperature patterns of cattle and their perturbations due to various physiologic and pathologic conditions must first be established using a system which more continuously monitors animal temperature. In all probability this requires the active telemetric system. This phase of study must precede and be ongoing during use of any passive system in order to interpret periodically recorded body temperatures with the passive system.

I think that more extensive computerization, especially of large dairy farms, is inevitable. Automation of farms may then come close on the heels of computerization. Electronic animal identification would be a necessary component of automation. Automatic recording of animal weight, daily milk production, and body temperatures would be important parameters for automated farms for greater efficiency of breeding, feeding, culling, treatment and general management of livestock.

LITERATURE CITED

1. Figure 1. The daily mean temperatures of the three groups of cattle; (A) Early lactating cows, (B) late lactating cows, and (C) dry cows. Environmental conditions are represented by (D) Black Globe and (E) dry bulb temperatures.
2. Figure 2. Comparison of mean temperatures of the early lactating versus dry cows at each sampling time of the day over the entire 17-day period of the trial. The dots indicate those comparisons where the early lactating cows had significantly higher temperatures than dry cows.
3. Figure 3. A one-day temperature tracing of a cooled (open circles) and a control (closed circles) cow. The hatched line indicates the period of operation of cooling procedures in the milking parlor.
4. Figure 4. A one-day temperature tracing of a cooled (open circles) and a control (closed circles) cow. The hatched line indicates the period of operation of cooling procedures in the milking parlor.
5. Figure 5. Temperature tracing of a cow on the (A) day before estrus, (B) the day of estrus, and the (C) day after estrus.
6. Figure 6. Temperature tracing of a cow on the 5th and 4th days before parturition (upper tracing) and on the day before and day of parturition (lower tracing). Arrow indicates the time of parturition.
REPORT OF COMMITTEE ON LIVESTOCK IDENTIFICATION

Chairman: Harold Mindermann, West Des Moines, IA
Vice Chairman: R.E. Nelson, Brattleboro, VT

J.B. Ashcraft, CO; J.H. Baldwin, NY; D.R. Bridgewater, CO; H.F. Embry, IL; G.B. Estes, VA; Robert Gadd, SD; Bill Gallagher, SD; H.E. Goldstein, OH; Tom Haas, KY; J.N. Huff, CO; G.M. Jones, NM; Dee Likes, KS; D.A. Mitchell, WA; L.R. Olson, SD; N.F. Powers, Jr., NY; E.C. Roukema, VA; F.L. Seawright, NM; Raymond Schnell, ND; G.R. Snyder, VA; W.E. Stemler, IL; F.E. Sterner, CO; J.R. Taylor, TX; J.E. Thomas, NV; Mark Trask, SD.

Dr. I. T. Rhodes outlined progress in finding an alternative to the slap tattoo as required by regulation in identifying sows and boars. He recognized the disappointment in the T Tagger and received the project outline for development of a device in cooperation with ARS. He reiterated the problems with back tags and button tags emphasizing the desperate need for something different.

Dr. Granville Frye reported that a 1981 survey revealed that 48% of sows and boars were skinned. However, the percent ranged from 8.3% in the western region to 92.1% in the South Central region. 80% of the boars were skinned while 42% of the sows were skinned.

Calvin Campbell reviewed the progress being made with the Brucellosis information system with 7 states to be added to the 6 on the system with 18 more to be added in fiscal 83.

The problems associated with the use of a second eartag were described and the problems of reading tags were emphasized by the practitioners.

Following a report by Richard Nelson that Livestock Conservation Institute should continue the National Livestock Electronic Identification Board. Representatives of Identification Systems gave a presentation on a new electronic identifying system.

Dr. Gary Seawright of Los Alamos Scientific Laboratory reviewed the status of Electronic Identification with special attention to the results of the data being collected at the Jornada Range station where 39 transponders have been recording identification and body temperature as body weight was being recorded since January 1979.

Professor R. M. Nakomura of the University of Hawaii reviewed the data being collected electronically on body temperature in the dairy herd. The evidence was suggesting the need to cool high producing cows at milking time. The evidence showed that the temperature of high lactating cows was very significantly greater than that of dry cows.

Dr. Max Van Bruskerk reviewed the mandatory swine identification regulations in Pennsylvania. While adopted in May, with cautious implementation this fall, full enforcement will now go into effect. These
LIVESTOCK IDENTIFICATION

regulations call for eartag, tattoo or other acceptable ID so the animal can be traced to the herd of origin.

The Committee Report recommends that USDA-APHIS review part 71.18 CFR that has to do with breeding animals moving interstate commerce to determine if it does in fact prohibit tampering with or removal of official identification at point of destination and if deficient, propose appropriate rule making to correct the deficiency.

In other action, the committee recommends that the UM&R for Brucellosis and Eradication Control include a requirement that each state prohibit tampering with or removing official identification.

Report on APHIS Identification Research and other identification activities—Dr. Irvin T. Rhodes, Chief Staff Veterinarian, Animal Identification and Surveillance, Veterinary Services.

1. Report on the “T” tagger development. This instrument is a product of Dennison Manufacturing Company, Framingham, Massachusetts. The instrument is used to attach a plastic “T” type tag to clothing.

Walter Jones, Animal Health Technician, made mechanical modifications to the instrument trying to adapt it to use in identifying unrestrained sows and boars. He developed a touch/triggering mechanism that actuated the air-pressured injector, passing the tag through a large-guage hypodermic needle. There are functional problems.

Recently the Company has stated that all development is being held in obeyance pending additional interest in the instrumentation by other agencies and industry. There is not sufficient profit potential for the Company to continue development.

2. Agricultural Research Service participation is a mutually benefiting project to research and develop a swine identification method and/or device with Veterinary Services participation in the implementation of the method for programmed disease eradication activities.

This is now in the developmental stage.


The statistical data is attached.

4. Vinyl backtag and special glue project for identifying sows and boars.

The vinyl tags were tried on sows and boars in Georgia and Minnesota. The tags are moisture proof and, therefore, do not curl or disintegrate when swine go under sprinklers. This was an improvement over the presently used paper backtags. The glue material used previously is water soluble; therefore, the percentage of loss was high. Veterinary Services is working with a company that specializes in adhesives. We hope to find a non-water soluble glue that will bond with the vinyl tag and
swine hair coat, even if oily and wet. We have not, as yet, tested a glue product we are satisfied with.

5. Button Tags.

This may be a desperation effort. The idea is to get going with a swine identification device that is available and field distributed now. This is the button portion of the bangle tag and is coded. Cost estimate is 15 cents if produced on ribbon in sequence. The idea is to slap this button subcutaneous behind the ear with a backtag applicator stick (demonstration). The problem is that MPIP objects to non-sterile injection. This problem would exist with the “T” tagger also.

Pennsylvania Mandatory Swine Identification Rules

Consideration of swine health programs for the regulation and control of avian tuberculosis and pseudorabies in Pennsylvania swine herds revealed the necessity to establish identification rules that would permit control of movement of swine and epidemiologic investigation of infected herds. Revised swine health rules were subsequently promulgated and adopted May 20, 1982.

Specific references to swine identification requirements are made in Chapters 3, 5 and 10 of the Pennsylvania Department of Agriculture Title VII Regulations Administered by the Bureau of Animal Industry which regulate intrastate and interstate movement, livestock auctions and pseudorabies control respectively.

Interstate Movement: “All swine shall be permanently identified by an ear tag, tattoo or other acceptable identification approved by the Director so swine can be traced to the farm of origin. This identification shall be in place prior to the animal entering this Commonwealth.”

Intrastate Movement: “All swine in intrastate movement shall be individually identified with an ear tag, tattoo, or other reliable identification approved by the Director to permit traceback to herd of origin.”

Livestock Auctions: “Owners and operators of community or other sales places and livestock dealers shall maintain accurate records of all livestock (swine) transactions for two years, which records may be examined at any time by a representative of the Bureau. Records of Sales shall include identification of swine bought, sold or handled.”

Pseudorabies: “All swine moved intrastate or interstate and all pseudorabies-tested swine shall be identified by permanent identification such as ear tag, tattoo or other acceptable identification approved by the Director so the swine can be traced to the farm of origin.” “Records of swine identification, movement and ownership shall be kept by the consignor, auction or dealers for two years after swine are moved so that they can be traced to herd of origin. These records shall be made available to the Director or his representative on request.”

The Bureau considers ear tattoo of swine to be the most practical and reliable form of identification available at this time. Producers of swine
are being urged to ear tattoo all baby pigs with a registered herd number. Slap tattoo of older swine with the herd number is an option as are ear tags. Ear tags are less desirable because they are frequently lost from the ear. Back tags and ear bangle tags may be utilized to identify finished hogs moving to market.

Swine producers and feeders are being urged to apply to the Bureau for a registered herd identification number. This number is a combination of four letters and/or numbers which will be unique for each swine farm. All feeder pigs and finished hogs should be marked by ear tattoo or slap tattoo with this number. Breeding animals must be individually identified and this may be accomplished by ear notch of animals bearing the herd identification tattoo, by an additional unique tattoo or by unique ear tag. Feedlot operators marketing finished hogs originating from other farms must identify these animals to the feedlot. Slap tattoo of these hogs with a registered herd (feedlot) number is preferred although any reliable method of identification is accepted.

Breeding swine imported into Pennsylvania must be individually identified. Feeder pigs and slaughter hogs must be identified to the state of origin. The Bureau is recommending that out-of-state consignors ear tattoo their feeder pigs with the state code number (the prefix number on official cattle ear tags).

Identification of imported swine must be established prior to entry into Pennsylvania. Identification of swine in intrastate movement must be established at the point of transfer of ownership and prior to commingling with other swine. Persons having possession or custody of swine are responsible for the identity of those swine and must maintain records that will permit tracing the movement of those swine. Swine presented to marketing agents or slaughter establishments must have been identified by the consignor; however, the consignee has the option to provide identification to the consignor for the consignor in lieu of refusing to accept the swine.

The most convenient and effective place to monitor compliance with identification requirements is at the marketplace and slaughter establishment. It is unfortunate that regulatory officials must focus their attention upon marketing agents since this activity may impede the flow of livestock through these channels. In order to minimize interference with the flow of livestock in marketing channels, the Pennsylvania Department of Agriculture will stress application of a registered herd number to swine before leaving the farm of origin.
REPORT OF THE COMMITTEE

SOW AND BOAR SLAUGHTER

(Based on April 1981 MPIP Plant Survey and September 1981 MPIP Slaughter Report)

Granville Frye, D.V.M.
Hyattsville, MD.

### NATIONAL

<table>
<thead>
<tr>
<th>Category</th>
<th>Total Slaughter</th>
<th>No. Skinned</th>
<th>Percent Skinned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sow Slaughter</td>
<td>457,245</td>
<td>193,641</td>
<td>42.3</td>
</tr>
<tr>
<td>Boar Slaughter</td>
<td>102,704</td>
<td>80,260</td>
<td>78.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Category</th>
<th>Total Slaughter</th>
<th>No. Skinned</th>
<th>Percent Skinned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sow Slaughter</td>
<td>145,033</td>
<td>68,123</td>
<td>46.9</td>
</tr>
<tr>
<td>Boar Slaughter</td>
<td>64,587</td>
<td>47,984</td>
<td>74.3</td>
</tr>
</tbody>
</table>

- No. Plants Slaughtering Sows and Boars: 654
- No. Plants Skinning Sows and Boars: 301
- Percent Skinning Sows and Boars: 46

No. plants slaughtering Boars: 186

### NORTHERN REGION

<table>
<thead>
<tr>
<th>Category</th>
<th>Total Slaughter</th>
<th>No. Skinned</th>
<th>Percent Skinned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sow Slaughter</td>
<td>145,033</td>
<td>68,123</td>
<td>46.9</td>
</tr>
<tr>
<td>Boar Slaughter</td>
<td>64,587</td>
<td>47,984</td>
<td>74.3</td>
</tr>
</tbody>
</table>

- No. Plants Slaughtering Sows and Boars: 243
- No. Plants Skinning Sows and Boars: 127
- Percent Skinning Sows and Boars: 52.3

No. Plants Slaughtering Boars: 83
### SOUTHEAST REGION

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Slaughter</td>
<td>87,458</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. Skinned</td>
<td>67,636</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent Skinned</td>
<td>68.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sow Slaughter</td>
<td>78,114</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. Skinned</td>
<td>61,222</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent Skinned</td>
<td>78.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boar Slaughter</td>
<td>9,344</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. Skinned</td>
<td>6,414</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent Skinned</td>
<td>68.6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| No. Plants Slaughtering Sows and Boars | 123 |
| No. Plants Skinning Sows and Boars    | 67  |
| Percent Skinning Sows and Boars       | 54.4|

| No. Plants Slaughtering Boars | 17 |

### SOUTH CENTRAL REGION

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Slaughter</td>
<td>28,766</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. Skinned</td>
<td>26,498</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent Skinned</td>
<td>92.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sow Slaughter</td>
<td>26,687</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. Skinned</td>
<td>25,326</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent Skinned</td>
<td>94.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boar Slaughter</td>
<td>2,079</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. Skinned</td>
<td>1,172</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent Skinned</td>
<td>56.4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| No. Plants Slaughtering Sows and Boars | 63 |
| No. Plants Skinning Sows and Boars    | 20 |
| Percent Skinning Sows and Boars       | 31.7|

| No Plants Slaughtering Boars | 15 |
### NORTH CENTRAL REGION

<table>
<thead>
<tr>
<th></th>
<th>Total Slaughter</th>
<th>No. Skinned</th>
<th>Percent Skinned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Slaughter</td>
<td>230,882</td>
<td>63,292</td>
<td>27.4</td>
</tr>
<tr>
<td>Sow Slaughter</td>
<td>204,623</td>
<td>38,791</td>
<td>18.9</td>
</tr>
<tr>
<td>Boar Slaughter</td>
<td>26,259</td>
<td>24,501</td>
<td>93.3</td>
</tr>
</tbody>
</table>

| No. Plants Slaughtering Sows and Boars | 154 |
| No. Plants Skinning Sows and Boars    | 83  |
| Percent Skinning Sows and Boars       | 53.9|

### WESTERN

<table>
<thead>
<tr>
<th></th>
<th>Total Slaughter</th>
<th>No. Skinned</th>
<th>Percent Skinned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Slaughter</td>
<td>3,223</td>
<td>268</td>
<td>8.3</td>
</tr>
<tr>
<td>Sow Slaughter</td>
<td>2,788</td>
<td>79</td>
<td>2.8</td>
</tr>
<tr>
<td>Boar Slaughter</td>
<td>435</td>
<td>189</td>
<td>43.4</td>
</tr>
</tbody>
</table>

| No. Plants Slaughtering Sows and Boars | 71  |
| No. Plants Skinning Sows and Boars    | 4   |
| Percent Skinning Sows and Boars       | 5.6 |

| No. Plants Slaughtering Boars         | 30  |
LIVESTOCK IDENTIFICATION

BOAR SLAUGHTER

<table>
<thead>
<tr>
<th>Rank</th>
<th>State</th>
<th>No. Boars Slaughtered</th>
<th>No. Skinned</th>
<th>% Skinned</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Illinois</td>
<td>25,396</td>
<td>23,707</td>
<td>93.3</td>
</tr>
<tr>
<td>2</td>
<td>Pennsylvania</td>
<td>15,915</td>
<td>1,566</td>
<td>9.8</td>
</tr>
<tr>
<td>3</td>
<td>Iowa</td>
<td>15,826</td>
<td>14,648</td>
<td>92.5</td>
</tr>
<tr>
<td>4</td>
<td>Missouri</td>
<td>9,294</td>
<td>9,283</td>
<td>99.9</td>
</tr>
<tr>
<td>5</td>
<td>Michigan</td>
<td>9,240</td>
<td>9,240</td>
<td>100.</td>
</tr>
<tr>
<td>6</td>
<td>Minnesota</td>
<td>7,956</td>
<td>7,730</td>
<td>97.1</td>
</tr>
<tr>
<td>TOTAL</td>
<td></td>
<td>83,627</td>
<td>66,174</td>
<td>79.1</td>
</tr>
</tbody>
</table>

83 percent of the national boar slaughter in September 1961 occurred in the 6 States listed.
### Plants Slaughtering Sows and Boars and Type of Slaughter

Based on April 1981 MPIP Plant Survey

and September 1981 MPI Slaughter Printout

<table>
<thead>
<tr>
<th>State</th>
<th>Number of Plants</th>
<th>Number Skinning</th>
<th>Percent Skinning</th>
<th>Total Males</th>
<th>Number Skinned</th>
<th>Percent Skinned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Connecticut</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>98</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Delaware</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Illinois</td>
<td>12</td>
<td>6</td>
<td>50</td>
<td>233</td>
<td>1,378</td>
<td>44.3</td>
</tr>
<tr>
<td>Maine</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Maryland</td>
<td>6</td>
<td>1</td>
<td>17</td>
<td>6</td>
<td>11</td>
<td>18</td>
</tr>
<tr>
<td>Massachusetts</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>104</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Michigan</td>
<td>3</td>
<td>2</td>
<td>100</td>
<td>16,511</td>
<td>16,511</td>
<td>100</td>
</tr>
<tr>
<td>Minnesota</td>
<td>22</td>
<td>15</td>
<td>68</td>
<td>21,890</td>
<td>10,697</td>
<td>49.5</td>
</tr>
<tr>
<td>New Hampshire</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>New Jersey</td>
<td>5</td>
<td>3</td>
<td>60</td>
<td>328</td>
<td>125</td>
<td>38.1</td>
</tr>
<tr>
<td>New York</td>
<td>34</td>
<td>16</td>
<td>47</td>
<td>409</td>
<td>166</td>
<td>40.6</td>
</tr>
<tr>
<td>Ohio</td>
<td>14</td>
<td>9</td>
<td>64</td>
<td>15,926</td>
<td>15,926</td>
<td>96.6</td>
</tr>
<tr>
<td>Pennsylvania</td>
<td>93</td>
<td>59</td>
<td>63</td>
<td>28,540</td>
<td>11,546</td>
<td>43.2</td>
</tr>
<tr>
<td>Rhode Island</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Vermont</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Virginia</td>
<td>16</td>
<td>8</td>
<td>50</td>
<td>9,760</td>
<td>6,369</td>
<td>64.8</td>
</tr>
<tr>
<td>West Virginia</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Wisconsin</td>
<td>5</td>
<td>2</td>
<td>40</td>
<td>16,401</td>
<td>13,193</td>
<td>80.4</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>243</td>
<td>127</td>
<td>52.3</td>
<td>209,620</td>
<td>116,107</td>
<td>55.4</td>
</tr>
<tr>
<td>Oklahoma</td>
<td>9</td>
<td>6</td>
<td>64</td>
<td>15,393</td>
<td>9,961</td>
<td>38.6</td>
</tr>
<tr>
<td>Florida</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Georgia</td>
<td>25</td>
<td>11</td>
<td>44</td>
<td>3,652</td>
<td>1,172</td>
<td>86.9</td>
</tr>
<tr>
<td>Kansas</td>
<td>28</td>
<td>12</td>
<td>43</td>
<td>19,889</td>
<td>16,087</td>
<td>92.4</td>
</tr>
<tr>
<td>Louisiana</td>
<td>5</td>
<td>4</td>
<td>80</td>
<td>530</td>
<td>668</td>
<td>88.3</td>
</tr>
<tr>
<td>North Carolina</td>
<td>15</td>
<td>9</td>
<td>60</td>
<td>10,597</td>
<td>10,124</td>
<td>95.5</td>
</tr>
<tr>
<td>South Carolina</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tennessee</td>
<td>43</td>
<td>24</td>
<td>56</td>
<td>32,477</td>
<td>25,866</td>
<td>79.0</td>
</tr>
<tr>
<td>Virgin Islands</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>123</td>
<td>67</td>
<td>53.5</td>
<td>87,458</td>
<td>67,636</td>
<td>78.6</td>
</tr>
<tr>
<td>Arkansas</td>
<td>30</td>
<td>3</td>
<td>10</td>
<td>6,762</td>
<td>4,472</td>
<td>65.7</td>
</tr>
<tr>
<td>Louisiana</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>New Mexico</td>
<td>3</td>
<td>1</td>
<td>33</td>
<td>51</td>
<td>51</td>
<td>1</td>
</tr>
<tr>
<td>South Dakota</td>
<td>9</td>
<td>6</td>
<td>66</td>
<td>5,504</td>
<td>5,353</td>
<td>97.3</td>
</tr>
<tr>
<td>North Dakota</td>
<td>20</td>
<td>10</td>
<td>50</td>
<td>16,448</td>
<td>14,672</td>
<td>89.2</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>63</td>
<td>20</td>
<td>15.9</td>
<td>28,766</td>
<td>26,498</td>
<td>92.1</td>
</tr>
<tr>
<td>South Dakota</td>
<td>23</td>
<td>16</td>
<td>70</td>
<td>2,222</td>
<td>967</td>
<td>42.6</td>
</tr>
<tr>
<td>Montana</td>
<td>4</td>
<td>3</td>
<td>75</td>
<td>7,286</td>
<td>7,256</td>
<td>96.8</td>
</tr>
<tr>
<td>Wyoming</td>
<td>19</td>
<td>7</td>
<td>37</td>
<td>767</td>
<td>162</td>
<td>19.0</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>34</td>
<td>24</td>
<td>41</td>
<td>34,212</td>
<td>3,246</td>
<td>94.9</td>
</tr>
<tr>
<td>North Dakota</td>
<td>23</td>
<td>21</td>
<td>91</td>
<td>1,922</td>
<td>1,607</td>
<td>83.5</td>
</tr>
<tr>
<td>Arizona</td>
<td>14</td>
<td>12</td>
<td>86</td>
<td>547</td>
<td>62</td>
<td>11.3</td>
</tr>
<tr>
<td>Utah</td>
<td>5</td>
<td>3</td>
<td>20</td>
<td>38,988</td>
<td>5</td>
<td>0.1</td>
</tr>
<tr>
<td>Wyoming</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>154</td>
<td>83</td>
<td>53.9</td>
<td>230,887</td>
<td>63,292</td>
<td>27.4</td>
</tr>
<tr>
<td>Alaska</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Arizona</td>
<td>14</td>
<td>2</td>
<td>14</td>
<td>1,764</td>
<td>249</td>
<td>14.1</td>
</tr>
<tr>
<td>California</td>
<td>17</td>
<td>16</td>
<td>94</td>
<td>3699</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Idaho</td>
<td>17</td>
<td>16</td>
<td>94</td>
<td>3699</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Nevada</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Oregon</td>
<td>24</td>
<td>13</td>
<td>50</td>
<td>816</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Washington</td>
<td>14</td>
<td>1</td>
<td>8</td>
<td>256</td>
<td>2</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>71</td>
<td>4</td>
<td>5.6</td>
<td>3,223</td>
<td>268</td>
<td>8.3</td>
</tr>
<tr>
<td><strong>National TOTAL</strong></td>
<td>654</td>
<td>301</td>
<td>46</td>
<td>559,949</td>
<td>273,901</td>
<td>48.9</td>
</tr>
</tbody>
</table>
COOPERATING SLAUGHTER ESTABLISHMENT - Boar Testing Field Trial - June 21 - Nov. 1, 1982

<table>
<thead>
<tr>
<th></th>
<th>Form A</th>
<th>Form B</th>
<th>Trace Attempts</th>
<th>Successful Traces</th>
<th>Total Tests</th>
<th>Reactors</th>
<th>Reactors traced to Herd/Origin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. Tags Applied</td>
<td>No. Tags Retrieved</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ill.</td>
<td>1,045</td>
<td>64</td>
<td>20</td>
<td>17</td>
<td>18,669</td>
<td>3</td>
<td>None</td>
</tr>
<tr>
<td>Ind.</td>
<td>363</td>
<td>36</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>97</td>
<td></td>
<td>4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Conclusions

1. Low incidence in the population that was tested;
2. Trace attempts involved only short haul situations;
3. Statistics support other data but do not enable conclusion that the boar is a reliable sentinel animal for locating brucellosis infected source herds;
4. Number of source herds involved in the test = approx. 590;
5. Small boars (probably not breeders) and sows that were identified were tested. No titred samples found among any of these;
6. All titred samples from large boars were not identified.
The USAHA Committee on Professional Oversight met at 1:30 p.m., Thursday, November 11, 1982. The following subjects were considered by the Committee:

1. All items contained in last year's report were reviewed.
   a. The Committee at its past three meetings expressed concern regarding the implied warranty involved in signing health certificates. Statements on certain health certificates could place the issuing veterinarian in unwarranted jeopardy. At this year's meeting, a resolution addressing this problem has been submitted by another committee for consideration.

   It was noted the AVMA Professional Liability Insurance Trust is in the process of reviewing official health certificates. This review was initiated because of increasing lawsuits pertaining to health certification.

   It is again this Committee's recommendation that states modify signature statements on their official health certificates.

   b. The Committee reviewed the progress resulting from Resolution No. 17 endorsed at the 1981 annual meeting. Two members of the Professional Oversight Committee served on a task force which was appointed to evaluate ARS/APHIS Research and Diagnostic Programs. The recommendations of the special task force were totally compatible with the goals and objectives of the USAHA. A recommendation was made that the President of the USAHA communicate with the Secretary of Agriculture expressing appreciation for the expediency with which the administrators of ARS and APHIS responded to the task force recommendations.

2. The Swine Brucellosis Subcommittee was unable to complete its report in time for review and approval of the Brucellosis Committee. Therefore, the Subcommittee report was not included in the Brucellosis Committee report presented on Wednesday, November 10. Dr. Paul Doby, Chairman of the Subcommittee on Swine Brucellosis, presented the report to the Committee on Professional Oversight. The report was reviewed and passed to the Executive Committee without objection.

3. The problem of committee meeting scheduling was discussed. Many USAHA members are unable to attend committees of interest because of the large number scheduled on Monday, Tuesday, and Wednesday of the proceedings. It was suggested that some
meetings could be rescheduled for Thursday to provide a better distribution. One of the problems with heavy scheduling on Thursday is the short deadlines to complete reports in time for the Executive Committee’s review. President John Ragan stated committee scheduling was creating problems for the American Association of Veterinary Laboratory Diagnosticians (AAVLD). Committee meetings held on Monday and Tuesday affect attendance at the AAVLD general sessions. Dr. Ragan advised the Committee he would give this problem area priority consideration prior to next year’s meeting.

4. One resolution concerning the personal liability of federal and state employees engaged in carrying out their official duties was moved to the Resolutions Committee.
REPORT OF THE COMMITTEE ON PARASITIC DISEASES AND PARASITICIDES

Chairman: R. L. Pyles, New Mexico
Vice Chairman: John H. Gray, Texas

J. H. Bailey, SD; L. G. Beihl, IL; A. R. Burgess, WY; S. E. Christy, IL; R. O. Drummond, TX; B. Gallagher, SD; S. C. Gartman, TX; J. F. Hudelson, CO; N. W. Kruse, NE; M. H. Lang, IA; R. P. McDonald, TX; C. H. Miranda, SC; J. H. Niemi, SD; J. E. Novy, TX; J. R. Olson, SD; J. R. Pemberton, IA; R. L. Rissler, MD; G. O. Schubert, MD; M. G. Scroggs, OH; G. F. Slonka, TX; P. O. Smith, CA; R. K. Strickland, IA; W. W. Utterback, CA; D. E. Zinter, MD.

The committee met on Monday, November 8, 1982, with 12 members and 17 guests present.

Dr. I. R. Reid, Animal Health Division, H of A, Ottawa, Ontario, Canada, presented to this committee an update on a field trial using Ivermectin on a rather large outbreak of Sarcoptic mange in cattle.

MANGE IN CANADA

Mange is a reportable disease under the ADP Act and Regulations. In practice, only the following are quarantined and treated under supervision:

- Sarcoptic mange of cattle, sheep, goats and horses.
- Psoroptic mange of cattle, sheep and horses.

Psoroptic mange is the principal mange in the USA, primarily in the south western states but is not known to exist in Canada. Cattle imported from the USA are re-examined after 30 to 60 days for Psoroptic mange.

In Canada, Sarcoptic mange of cattle occurs only in British Columbia (north) and Alberta (scattered). In recent years, an average of 12 cases per year were uncovered. In FY 1981-82, 70 herds were positively diagnosed, 44 of which had been in contact through a community pasture. Three of these were dairy herds.

The number of spray-dip machines available in these two provinces was inadequate to treat all of those herds with the classical double treatment using Toxaphene or Lindane.

Ivermectin Field Trial—Conducted with the cooperation of Health and Welfare Canada and Merck, Sharp and Dohme. The field work was carried out by Food Production and Inspection Branch personnel.

65 herds were treated with Ivermectin (50 in B.C. and 15 in Alberta). A total of 13,000 cattle were treated with one subcutaneous injection. (1% solution — 1 ml/100 lbs body weight). All animals were treated, regardless of age, with new born calves being treated at one week of age. No lactating dairy cattle were treated with Ivermectin.

Of the 13,000 cattle treated, 2,088 cattle exhibited lesions. 746 of which
were classified as medium to extensive. Some of the herds were also infested with Chorioptic and Psorergatic mange and biting and sucking lice.

Results of the treatment: 100 percent clinical recovery. Adverse effects: insignificant. Favorable side-effects: improved general condition; noticeable weight gains; and delighted ranchers.

A series of slides were shown of 12 animals with severe lesions showing pre-treatment, 15 days post treatment and 31 days post treatment.

Dr. Glen O. Schubert, VS, APHIS, USDA, reviewed for the committee the RPAR decision on toxaphene usage and restrictions that was released on October 18, 1982. It may be used for the treatment of cattle and sheep scabies in a vat or spray-dip machine. It must be applied by certified applicators who must use respirators and other protective gear. Disposal must meet criteria for hazardous wastes. The sale of toxaphene will continue until supplies are exhausted or until December 31, 1986, whichever occurs first.

Dr. Schubert also presented the following figures on the Puerto Rican Tick Program and the Cattle Scabies Program:

**TICK PROGRAM IN PUERTO RICO — FY 1982**

<table>
<thead>
<tr>
<th></th>
<th>B. microplus</th>
<th>A. variegatum</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. Herds Treated:</td>
<td>45,080</td>
<td>2,652</td>
<td>47,721</td>
</tr>
<tr>
<td>No. Animals Treated:</td>
<td>797,833</td>
<td>269,477</td>
<td>1,067,310</td>
</tr>
<tr>
<td>Premises Freed:</td>
<td>1,803 less 331 reinfested; total premises freed 1,472 (Boophilus)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**CATTLE SCABIES — FY 1982**

There has been a total of 115 reported psoroptic cattle scabies outbreaks which have been confirmed in the United States in FY 1982. These outbreaks have been reported from the following States:

- California — 4
- Colorado — 39
- Iowa — 7
- Kansas — 15
- Minnesota — 1
- Nebraska — 13
- Nevada — 1
- New Mexico — 8
- Oklahoma — 4
- Texas — 18
- Wyoming — 1

As of October 29, 1982, there have been scabies outbreaks reported for 76 consecutive months.

Dr. John H. Gray, VS, APHIS, USDA, Austin, Texas, presented the following report on the Texas Tick Program and statistics on the National Tick Surveillance Program.
REPORT OF THE COMMITTEE

STATUS OF THE UNITED STATES - TEXAS TICK PROGRAM
(BOOPHILUS ANNULATUS AND BOOPHILUS MICROPLUS)
IN FISCAL YEAR 1982

NEW INFESTATION:

<table>
<thead>
<tr>
<th>Description</th>
<th>TEQA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free Area</td>
<td>15</td>
</tr>
<tr>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

The following number of animals were apprehended along the Rio Grande:

Mexico to USA
- 34 cattle, 14 were tick infested
- 47 horses, 0 were tick infested

USA to Mexico
- 38 cattle, none infested
- 52 horses, none infested

STATUS OF WILDLIFE INVOLVEMENT:

The Webb County tick infestation, previously reported, appears to have been eradicated. We have a new Boophilus annulatus tick infestation in Maverick County. Epidemiological data indicates the white-tailed deer have maintained the tick infestation.

STATUS OF THE NATIONAL TICK SURVEILLANCE PROGRAM:

The collection and submission of ticks from native and imported animals plus plant and animal material is continuing its downhill trend. Somehow, we must stimulate animal health regulatory personnel to collect and submit ticks.

The southern states are at risk to the introduction and establishment of ticks from areas of the world where African swine fever, Heartwater and other such diseases are common. A fast look at the National Tick Surveillance Program results for the last four years shows we are not doing well.

NATIONAL TICK SURVEILLANCE PROGRAM
TICK COLLECTIONS

<table>
<thead>
<tr>
<th>CATTLE</th>
<th>ALL HOSTS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>States</td>
</tr>
<tr>
<td>States¹</td>
<td></td>
</tr>
<tr>
<td>CY 78 8,191</td>
<td>7684</td>
</tr>
<tr>
<td>CY 79 7,789</td>
<td>7318</td>
</tr>
<tr>
<td>CY 80 6,460</td>
<td>6099</td>
</tr>
<tr>
<td>CY 81 8,010</td>
<td>7721</td>
</tr>
</tbody>
</table>
ALL HOST TICK COLLECTION, POSSIBLE HIGH RISK STATES

<table>
<thead>
<tr>
<th>States</th>
<th>CY 78</th>
<th>CY 79</th>
<th>CY 80</th>
<th>CY 81</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alabama</td>
<td>32</td>
<td>26</td>
<td>20</td>
<td>24</td>
</tr>
<tr>
<td>California</td>
<td>5</td>
<td>6</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Florida</td>
<td>265</td>
<td>304</td>
<td>303</td>
<td>286</td>
</tr>
<tr>
<td>Georgia</td>
<td>91</td>
<td>91</td>
<td>127</td>
<td>58</td>
</tr>
<tr>
<td>Hawaii</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Louisiana</td>
<td>5</td>
<td>5</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Mississippi</td>
<td>6</td>
<td>34</td>
<td>21</td>
<td>13</td>
</tr>
<tr>
<td>South Carolina</td>
<td>24</td>
<td>19</td>
<td>59</td>
<td>54</td>
</tr>
</tbody>
</table>

States¹ = 50 states plus Puerto Rico and the U.S. Virgin Islands
Texas² and Puerto Rico are involved in Tick Eradication Programs

Perhaps this Committee should review the National Tick Surveillance Program for effectiveness in relationship to the reasons for having this program. Perhaps priority areas of emphasis should be established for tick collections. Areas of priority may be states at risk for possible establishment of tick population, or areas at risk for introduction.

The “WHYS” for collecting ticks were reported in the 1980-1981 report, National Tick Surveillance Program. They are reprinted here for your information.

WHY COLLECT TICKS?

1. The Southern States are vulnerable to Boophilus spp. because of the threat from Mexico and the Caribbean Islands.
2. Tick collections provide an index of surveillance activity.
3. Tick collections provide information on the changing incidence and distribution of native ticks.
4. Tick collections provide an early warning to the introduction of exotic ticks.
5. Tick collections provide a measure of the interest and commitment of supervisions and employees to the goal of tick eradication.
6. The conscientious collection of ticks helps develop increased rancher/producer confidence in all livestock disease prevention and eradication programs.

Dr. R. D. Drummond, US Livestock Insects Laboratory, SEA,AR, USDA, updated the committee on “Recent Advances in Veterinary Entomology.” Subject matter covered new products for the control of biting flies, cattle grubs, nosebots, ticks and mites and new methods of application such as boluses, leg bands and tail tags.
REPORT OF THE COMMITTEE ON PUBLIC HEALTH
AND ENVIRONMENTAL QUALITY

Chairman: A.J. Roth, Richmond, VA
Vice Chairman: R.H. Singer, Winchester, KY

R.K. Anderson, MN; A.W. Bailey, OK; C.R. Bartz, MD; L.M. Boyer, VA; C.R. Dorn, OH; R.P. Crawford, TX; S.L. Hendricks, MN; W.T. Hubbert, MD; W.E. Jennings, TX; J.C. Leighty, MD; M.R. Levy, NJ; V.M. Loomis, MD; W.R. Miller, AL; E.V. Morse, IN; C.J. Nelson, CO; Paul Nicoletti, FL; R.L. Parker, SC; W.C. Patterson, GA; J.E. Pearson, IA; J.C. Prucha, MD; L.W. Schnurrenberger, MD; D.F. Schwindaman, MD; T.B. Snodgrass, TX; J.H. Steele, TX; C.D. Stumpff, KS; H.M. Trabosh, MD.

The Committee on Public Health and Environmental Quality met at 1:30 PM, Wednesday, November 10, 1982 as scheduled. A total of 13 members and 15 guests were in attendance.

Hazards occurring to animal and public health as a result of private ownership of wild and exotic animals was presented to the Committee by Dr. Stanley Diesch, University of Minnesota. It was encouraged that legislation be considered by the states controlling the private ownership of wild and exotic animals. An example of a bill of this nature that is being considered in Minnesota is attached hereto. The presentation provided by Dr. Diesch is attached hereto. It was recommended by the Committee that the United States Animal Health Association reaffirm at this time a resolution passed in 1980 as follows: "The problem of wild animal/pet associated rabies is an increasing hazard to both man and animals, and whereas no feasible means exist for assuming that wild animal pets especially foxes, skunks, and raccoons are immune to rabies, be it resolved that the U. S. A. H. A. requests that the U. S. D. A. and U. S. P. H. S. together develop rules to prohibit the interstate traffic in skunks, foxes and raccoons."

A method of calculating the wind dispersion of toxicants occurring from industrial exhaust systems was presented by Dr. Ernest D. King, Oklahoma University College of Health.

Bovine tuberculosis in man was presented by Dr. Charles D. Stumpff, Veterinary Services, U. S. D. A. in which he discussed the incidence of tuberculosis caused by Mycobacterium tuberculosis and M. bovis in man and cattle from the late 1800s to the present time.

Evaluation of public health effects on animals and human beings living near high voltage DC transmission lines was presented by Dr. Frank Martin, University of Minnesota. He stated that the health effect of proximity to high voltage power lines is becoming a fresh issue in this country as the new technology of direct current D.C. transmission becomes more widely applied. In 1980 a ± 400 volt DC power line was activated in Minnesota, then the largest in the country. Meanwhile other
states, notably New York, are planning even higher voltage DC lines. Large amounts of air ionization is possible from DC transmission and may create a health effect issue not present in AC power transmission. The State of Minnesota has funded a retrospective study, undertaken by the College of Veterinary Medicine, into the productivity of Holstein dairy cows as recorded in DHIA files collected prior to and following energizing of the power line. A report of the Subcommittee on Economic Loss to Industry and in Human Productivity due to Major Zoonoses was presented by its Subcommittee Chairman, Dr. Erskine Morse, Purdue University. He suggested that a subcommittee of the main committee, 1) Explore and document the animal health economic aspects as well as the loss in human productivity of the following zoonoses: (a) Ornithosis, (b) Brucellosis, (c) Salmonellosis, (d) Vesicular stomatitis, (e) Tuberculosis, (f) Toxoplasmosis.

A report of the Subcommittee on Public Health Aspects of Brucellosis Reactors in Slaughter Channels by Subcommittee Chairman, Dick Crawford, Texas A&M. He stated that conditions that help to alleviate this problem would be improve air flow through the plant, improve personal hygiene of the plant personnel and continuing educational program on the hazards of brucellosis. Employees with flu symptoms for 3 days may be a red flag for the employee to see his doctor.

A Report of the Subcommittee on Future Challenges and Concerns in Public Health was given by Dr. William Hubbert, APHIS.

He pointed out that the minimum standards for local health departments being evaluated in certain states. These states are: California, Illinois, Michigan, Minnesota, North Carolina, and Ohio, have either adopted or are in the process of developing minimum standards for the operation of local public health departments.

The future role of the veterinarian in local health departments and the future contributions of the veterinary profession in public health will be greatly affected by the content of these minimum standards. Veterinarians should become involved in the development, evaluation and implementation of these standards in their states.

Report of the Subcommittee on Hazardous Waste Causing Serious Health Problems to Farm Animals and Man was provided by Dr. W. R. Miller, Auburn University. It is agreed that hazardous waste does indeed pose a present as well as a potential threat to the health of farm animals as well as man. In addition to the generally recognized problem of landfill effluents into the ground water supply and the atmosphere, there also exists the problem of dumping of hazardous materials at sites other than landfills.

An important factor associated with the problem of hazardous waste is the lack of knowledge concerning its magnitude. There appears to be no central repository for information concerning farm animal morbidity and mortality from hazardous waste.
In order to initiate interest in the hazard waste problems and to begin the development of epidemiologic information the Subcommittee makes the following recommendations:

1. That Dr. Robert H. Singer, Winchester, Kentucky, be asked to present a paper on the hazardous waste problem at a general session of the 1983 meeting of the U.S. Animal Health Association.

2. That a survey instrument be developed by the Committee to be submitted to State Veterinarians, Diagnostic Laboratories, and others in an effort to gain epidemiologic information on morbidity and mortality of farm animals resulting from exposure to hazardous waste.

This Committee will further evaluate survey methods.

A report was presented by Dr. Jack C. Leighty on Toxoplasmosis, The Modes of Infection to Man and to Domestic Animals Entering the Food Chain; a copy of which is attached to this report.

TOXOPLASMOSIS

Jack C. Leighty, D.V.M.

*Toxoplasma gondii* is an obligate, intracellular, coccidian parasite. Like other coccidia it has an enteroepithelia (sexual) cycle in a specific host. Current knowledge indicates that the specific host must be a member of the cat (Felidae) family. However, unlike most other coccidia this organism has a cycle that can occur as a systemic infection in mammals and birds with invasion and multiplication in any cell with the exception of non-nucleated erythrocytes. The organism exists in three principal forms: the oocysts that are excreted in the feces of acutely infected cats are the product of the enteroepithelial cycle; the tachyzoites which are the rapidly multiplying forms that occur in acute infections in the tissues of cats as well as non-felines; and the bradyzoites that are the slow multiplying encysted forms that persist inside tissue cells as immunity develops. Each of these forms is capable of initiating an infection under appropriate circumstances.

**OOCYST PRODUCTION**

The enteroepithelial multiplicative stage is initiated in felines with the ingestion of sporulated oocysts, tachyzoites or bradyzoites. The prepatent period (time from ingestion of the infective form to the excretion of oocysts) is 20-24 days after ingestion of sporulated oocysts, 5-10 days after ingestion of tachyzoites and 3-5 days after ingestion of encysted bradyzoites. Cats may excrete up to 10 million oocysts in a single day, and excretion may continue for two weeks. Immunity to the intestinal stage in cats is relative. Renewed oocyst production seldom occurs with exposure to the original infecting strain, but may occur and persist for a few days upon reinfection by a different strain of the organism. The oocysts are not infective in freshly passed cat feces. They
sporulate and become infective within one to four days under aerobic conditions at suitable temperatures. Each infective oocyst contains two sporocysts and each sporocyst contains four sporozoites. The oocysts are approximately 10-12 microns in diameter. They are very resistant to acids, alkalies, and common disinfectants, but are killed by ammonia and exposure to 55° C. temperature. They survive for months in warm, moist soils but have also been shown to survive under natural conditions in Kansas soils resisting the winter temperatures. Their low specific gravity tends to move them toward the upper areas of moist soils. Movement to the soil surface also results from earthworm ingestion. Coprophagic insects such as filth flies and cockroaches can transport infective oocysts directly to food or to points where the probability of human or animal infection is increased.

THE TACHYZOITE

The extra intestinal cycle, which occurs in all species but constitutes the entire cycle in the non-feline host, usually begins after ingestion of sporulated oocysts, tachyzoites or encysted bradyzoites. However, infection can also be initiated by the introduction of tachyzoites or bradyzoites into the body through the abraded skin, the mucosa, the lungs, by blood transfusion, organ transplant or transplacentally. Transmission by blood sucking arthropods has neither been conclusively eliminated nor confirmed.

Infection occurs when tachyzoites penetrate host cells and there, protected from immunological mechanisms, divert host cell metabolites to themselves. Within the host cells, by a process known as endodyogeny, two new tachyzoites are formed with the loss of the original tachyzoite. This process continues until the host cell becomes distended and ruptures releasing 16 or more tachyzoites. They spread from cell to cell and are disseminated throughout the body, to infect new cells by the blood and lymph circulation in macrophages, lymphocytes, and granulocytes, as well as in free form. The level reached by the infection depends upon the virulence of the infecting strain for the particular species. Tissues having a greater accessibility via lymphatics or blood vessels, and tissues which are most suitable for tachyzoite replication, as are lymph nodes, are most severely infected. Those that do not regenerate well, such as brain and retina, are most significantly damaged while those that do regenerate quickly, such as lymphoid tissue and intestinal epithelium, suffer less damage.

The tachyzoites are the least resistant of the three forms being highly susceptible to drying, osmotic forces, proteolytic digestive enzymes, the hosts antibody defenses and temperature extremes.

The parasitemia persists until the hosts immune system makes the extracellular environment unfavorable for the tachyzoites. As host resistance rises the parasitemia declines and the formation of intracellular cysts containing bradyzoites begins.
THE BRADYZOITE

Encysted bradyzoites develop intracellularly, particularly in neurons, retinal cells and cardiac and skeletal muscle. The cysts increase in size as the bradyzoites within multiply. A cyst measuring 50 microns may contain between 1,000 and 3,000 bradyzoites.

The cysts, formed following an acute infection, may persist for the life of the host. The occasional release of bradyzoites stimulates the production of antibody creating a condition known as infection immunity or premunition. However, if for some reason immunity wanes or is suppressed, rapid proliferation of the organism sometimes occurs with consequent damage to new cells, and with new cyst formation, if immunity returns. The cyst wall isolates its contents from the host preventing an inflammatory reaction unless it is ruptured.

The cysts are resistant to acidified pepsin and trypsin digestion. The freezing of meat markedly reduces the number of infectious cysts, but is not a sure way of killing all organisms. Heating meat to 66° C. (150° F.) throughout kills all Toxoplasma.

THE PATHOLOGICAL EFFECT

Injury to the host occurs via two mechanisms. The proliferation of the organism in the host’s cells, with subsequent destruction of the cell, accounts for much of the pathology. However, injury through hypersensitivity reactions is believed to occur, also. By this mechanism, in chronically infected, hypersensitive hosts the rupture of a Toxoplasma cyst may result in a reaction that causes the death of adjacent, uninfected cells.

THE HUMAN HEALTH PROBLEM

“Man is infected with Toxoplasma by the cyst stage from meat, the oocyst stage from cats and soil, or with tachyzoites transplacentally.” In the best studied regions of the world, Europe and the United States, the overall prevalence of toxoplasmosis in humans is 20% to 30% of the general population. Throughout the world approximately one half billion people may be affected. Prevalence in Africans and Latin Americans appears to be 50% to 70%. Data from Japan, Korea and a few Southeast Asian countries indicate rates of between 5% and 20%. Cultures that are isolated by geographic or dietary habits from infectious sources, such as Eskimoes and certain Pacific Islanders, show 0% to very low prevalence rates. Prevalence varies a great deal among local populations within any country. Climate, dietary habits, cultural hygiene practices, animal populations and many other factors seem to influence the relative risk of infection within any group. Serological studies of meat eaters and vegetarians have shown comparable infection rates indicating that intimate contact with other infection sources, such as contaminated soil, may play an important part in transmission.

In general it is true that while infection is very common, clinically
evident infection is not. Although morbidity data on this disease is poor it appears that a number of factors may contribute to determining whether an infection is sufficiently severe to produce clinical manifestations. These may include the infective dose, the virulence of the infecting organisms, concomitant infections, extremes of age, exposure, poor nutrition, pregnancy, lactation, blood disease, malignancies, and immunosuppressive drugs. In consideration of significant differences that may occur, Toxoplasma infections are usually classified as congenital and acquired.

**ACQUIRED TOXOPLASMOSIS**

It appears that most toxoplasmosis is acquired after birth. Incidence rates have been calculated for different age groups. However, the relative number of infections that occur in the different age groups appears to vary from place to place based on factors related to individual exposure.

There are three principal manifestations of acute acquired toxoplasmosis. They are simple lymphadenopathy, lymphadenopathy with involvement of another organ, and generalized toxoplasmosis. Lymphadenopathy is by far the most common form. Any node or group of nodes may be affected and the course of the disease may vary from rather severe, with a long recovery period, to a mild condition involving a single node. Children and young adults are most often affected. Occasionally lymphadenopathic toxoplasmosis involves other organs such as the brain, heart, lung, liver or skeletal muscles. Symptoms then relate to the diseased organs. Generalized toxoplasmosis is usually associated with immunological deficiency states that may have a variety of causes such as tumors, organ transplants or immunosuppressive therapy. The disease produced is often severe and sometimes fatal.

**CONGENITAL TOXOPLASMOSIS**

Infection of the fetus is a frequent result of an acute toxoplasmosis infection of the mother during pregnancy. In 30% to 40% of the women who become infected during their first pregnancy, transplacental infection of the baby will occur. In utero infections of the fetus in chronically infected mothers appears to occur with less frequency. However, the supporting data on the latter situation is less abundant.

The infection rate for fetuses born to acutely infected mothers varies with the trimester during which the mother becomes infected. The rate is approximately 17% for the first trimester, 24% for the second trimester and 62% for the third trimester.

Congenital toxoplasmosis infections in the United States are estimated to occur in from 1.1 to 2.7 in 1,000 live births. Applying this to 3.3 million live births per year, we can calculate an approximate annual incidence of 3,630 to 8,910. Studies in France indicate the fate of these babies as follows:
5%-15% will die
8%-10% have severe brain and eye damage
10%-13% will have moderate to severe visual handicaps
58%-72% will be asymptomatic at birth with a proportion developing active retinochoroiditis as children or young adults.

Related studies indicate that toxoplasmosis appears to be a potent cause of minimal brain damage which may be overlooked in early infancy and yet be expressed clinically years later.

In an effort to estimate the potential value of preventative programs for congenital toxoplasmosis only, Wilson and Remington used the following figures:

The average lifetime cost per individual for services, including aid to totally disabled, special schooling for visually handicapped, special schooling for moderately retarded, institutional or state-supported foster care for severely retarded, and yearly ophthalmologic follow-up care, is $67,246. The cost, if 3,630 affected babies are born annually, would be $244.1 million.

MAJOR FOOD ANIMAL SPECIES

The first report of spontaneous toxoplasmosis in swine was from R. L. Farrell et al at the Ohio State University College of Veterinary Medicine in 1952. Serological evidence of infection has since been reported from the U.S.A., Surinam, Netherlands, Switzerland, Norway, Germany, Czechoslovakia, Denmark, Singapore, Japan, Brazil, Taiwan, Phillipines, Italy, Egypt and India in percentages between 12 and 60. As in man, the disease in swine is usually subclinical. Toxoplasmosis is reportedly one of the most important causes of ovine abortion in England, Australia and New Zealand. The disease has been described in sheep in most parts of the world.

Following the first report of clinical toxoplasmosis in cattle by Sanger at Ohio State University in 1953, evidence of Toxoplasma infection has been found in cattle in most countries where investigations have been conducted but with less frequency than in other food animal species.

Meat bearing Toxoplasma cysts have been found to be a significant source of human infections in certain cultures. Braveny conducted a survey in Germany between 1968 and 1971 that involved 30,000 serum specimens and questionnaires which indicated that the eating of raw meat was the most important source of human toxoplasmosis infection. The risk of infection from animal contacts, including cats, was small. A study of possible infectious sources for pregnant women in the same country also found that raw meat was a greater infectious hazard than a household cat. A serological survey of 1,050 persons in South Kalimantan, Indonesia, and their associated animals, indicated that in spite of the presence of many infected cats, the principle source of human infection was undercooked goat meat.
Clinical symptoms seem to seldom be associated with acquired toxoplasmosis in otherwise healthy adults. However, clinical cases associated with undercooked meat have been reported. Disease may not follow infection acquired from the oocyst state of *T. gondii* as frequently as infection from the cyst because of the difference in dose of infecting organisms (eight sporozoites per oocyst compared to hundreds or thousands of bradyzoites per cyst). In one notable case that occurred in New York City in 1968, five medical students separately ate rare hamburger at the same place on the same night and all developed acute, clinically recognizable toxoplasmosis.

Humans who handle raw meat have been found to have higher prevalence of antibody. Riemann (1975) studied 144 workers in a slaughterhouse in Brazil and found 72% serologically positive for *T. gondii* antibodies. The highest prevalence, 92%, was among meat inspectors. The prevalence in boning and sausage department employees was 80% and 79%. The lowest prevalence was in kill-floor employees and corral workers which were 60% and 65%, respectively. Ten percent of the cattle tested had *T. gondii* antibodies. Similar results were found in two surveys in Japan with the highest prevalence among those who handle swine.

**DIAGNOSIS**

*Toxoplasma* infection can be diagnosed by histologic examination, isolation of the parasite, serology, skin test and by demonstration invitro of antigen-specific lymphocyte recognition. Histological techniques may seek forms of the organism as well as characteristic lesions. Isolation usually requires inoculation of suspect material into mice but tissue culture inoculation is also used. Pepsin and trypsin digestions are often used to separate the organisms from tissue. The serological tests used in the United States have included the Sabin-Feldman dye test, the indirect fluorescent antibody test, the complement-fixation test, the indirect hemagglutination test, and the enzyme immunoassay test for both antibody and antigen. The skin test measures the development of cell mediated immunity but, since it becomes positive weeks or months after infection, is of little value in the diagnosis of acute infection. The in-vitro lymphocyte test can be used to detect both lymphocyte transformation and the production, by lymphocytes, of a macrophage migration inhibition factor.

**TREATMENT**

A synergic combination of sulfonimidines and pyrimethamine has been used in experimental animal infections and in human infections. Spiramycin is effective in animals. While of value in certain clinical situations, both treatments have distinct limitations. Pyrimethamine is teratogenic and can cause a folic acid deficiency.
BREAKING THE INFECTION CYCLE

From the foregoing information, steps required for prevention of animal and human infections become apparent. Direct or indirect contact with the feces of cats that may contain infective oocysts should be avoided. The consumption of meat containing viable tachyzoites or encysted bradyzoites should be prevented.

Since the cat is key to the direct and indirect infection of humans by its contamination of the environment, including animal feeds, with infective oocysts one could expect that control of the infection in cats would be of great value. Our knowledge of the immune response of cats to infection would indicate that a vaccine may be possible. It would be highly desirable to produce immunity in cats, by vaccination, to any Toxoplasma organism that might be encountered and to have such immunity persist for a reasonable period of time—perhaps at least a year.

It would also be desirable to achieve this immunity without actually infecting the cat with viable organisms that would cause shedding of oocysts and a premunition state. Rather sophisticated techniques are available today for the identification and isolation of the antigenically important components of microorganisms. An acceptable vaccine should not be beyond the realm of possibility.

The center for Disease Control has reported that in 1981 rabid cats outnumbered rabid dogs by approximately 20%. In Iowa, for example, 74% of the bite-associated rabies exposures in 1981 were caused by cats. Some communities have already made the vaccination of cats for rabies mandatory. Since most communities have required registration, rabies vaccination and control of stray dogs for many years, the systems necessary to institute similar programs for cats are already in place. One might reasonably expect to see a proliferation of requirements for the registration and rabies vaccination of cats throughout this country in the near future.

A feline Toxoplasma vaccine might find a significant place in such programs. The public health implications of the disease are a persuasive argument for preventative programs that protect the cat, its owners, their neighbors and the food animals that they own. Interrupting the disease cycle by reducing the contamination of our environment by the only form of the organism that occurs free in nature could prove to be the most feasible cost effective approach significantly reducing human and animal health problems caused by this disease.

(REVISOR) XX/RH 81-2082

Introduced by Reif, D. Carlson, Blatz, B. Anderson, Welch
March 16th, 1981
Ref. to Com. on Health & Welfare
Companion S.F. No. 902
Ref. to S. Com. on
Reproduced by PHILLIPS LEGISLATIVE SERVICE, INC.
A bill for an act

relating to health; prohibiting the possession of skunks; setting a penalty;
proposing new law coded in Minnesota Statutes, Chapter 145.

BE IT ENACTED BY THE LEGISLATURE OF THE STATE OF MINNESOTA

Section 1. (145.365) (POSSESSION OF SKUNKS)

  Subdivision 1. (PROHIBITION.) In order to protect the public health and prevent human and domestic animal exposure to rabies, it shall be unlawful to:

  (a) Import into this state any skunk, for sale, barter, exchange or gift for use as a personal pet;

  (b) Acquire, sell, barter, exchange, give, purchase or (possess) any skunks.

  Subd. 2. (LIMITATION.) The provisions of subdivision 1 do not apply to the importation or possession of any skunk by a publicly or privately owned zoological park or circus or any other show where a skunk is exhibited but is not in physical contact with the public, or by scientific or educational institutions for research or educational purposes.

  Subd. 3. (PENALTY.) Violation of subdivision 1 is a misdemeanor.
THE HAZARDS OCCURRING BY THE PRIVATE OWNERSHIP OF WILD AND EXOTIC ANIMALS TO ANIMAL AND PUBLIC HEALTH

By Dr. Stanley L. Diesch*

During the last four decades an increase has occurred in private ownership of wild and exotic animals as pets. Wild animals are indigenous species such as skunks, raccoons and foxes which have not been domesticated. Exotic animals are foreign animals such as lions, leopard, ocelots, monkeys and snakes.

Most wild and exotic animals do not make reliable companion animals. They cause problems for owners, practicing veterinarians, practicing physicians, state and federal governmental agencies, including public health and regulatory officials.

Bites or other trauma should be of major concern for the owner. Injuries from minor to death of a human or another animal have been reported. Following a bite by a wild or exotic animal, public health officials will usually recommend that it be handled as a wild animal rabies suspect and be euthanized for rabies examination.

In 1981, the members of the American Veterinary Medical Association Council on Public Health and Regulatory Veterinary Medicine conducted an extensive survey on injuries or mental anguish associated with the keeping of wild and exotic animals as pets. Since tabulated reports did not exist, the Council requested information by letter from the U.S. Department of Interior, Centers for Disease Control, Association of Zoo Veterinarians, State Health Departments, State Veterinary Medical Associations, State Game Departments, Veterinary Diagnostic Laboratories, Colleges of Veterinary Medicine, Veterinary Science Departments and State Regulatory Agencies. They also requested these agencies to suggest other sources of information.

Reports of injuries were received from throughout the United States, 72 reports were selected and tabulated. These ranged from slight injuries to death and were associated with 25 species of wild-exotic animals. (1) The American Veterinary Medical Association (AVMA) strongly opposes the keeping of exotic or wild animals as pets. The AVMA has repeatedly addressed this problem in 1973, 1978, 1979 and 1981 and urged that states not having prohibitive laws should enact legislation.

The Centers for Disease Control, USPHS strongly recommends that wild animals should not be kept for pets and encourages states to make it unlawful to retain, sell or ship wild animals as pets, especially those captured from the wild. Their major concern is rabies and the handling of potential rabies due to exposure.

* Dr. Stanley L. Diesch, Professor and Division Head of Veterinary Epidemiology, Food Hygiene and Public Health, College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota 55108.
An increasing number of states are enacting legislation prohibiting the interstate shipment of wild-exotic animals for the purpose of becoming pets. Minnesota and several other states have allowed the raising of skunks as pets and the interstate shipment of skunks. These required health certificates signed by a practicing veterinarian. For example in fiscal year 1980 Minnesota shipped 2483 skunks from 10 farms to other states for sale as pets. (2) In 1980 in Minnesota the United States Department of Agriculture (USDA) Veterinary Services licensed 10 skunk farms/brokers. State regulatory officials approved interstate health certificates signed by private veterinary practitioners. Health certificates indicate the animals are clinically free of infectious diseases.

In 1982 Minnesota passed legislation that will phase out the rearing and interstate shipment of skunks for pets in 1985. However, the regulation still allows the right of an individual to own a skunk as a pet.

Approximately 40 states have legislation pertaining to wild animal laws. In examining several laws, extensive variation was found to exist. The following are selected examples of human injuries including death associated with wild and exotic animals which were submitted to the AVMA Council in Public Health and Regulatory Veterinary Medicine.

CASE REPORTS (1)

Procyonidae
Reports: raccoons 6
Case:
A four month old girl was severely mauled by a one year old pet raccoon that crawled into her bed. The raccoon tore off her scalp, chewed off part of her nose and mouth and bit the fingers off one hand. The child died the following day.

Mustelidae
Reports: skunks 20, ferrets 6
Case:
A ferret attacked a five month old infant and inflicted multiple bites and loss of 40% of her ears.

Canidae
Reports: timber wolf 3, coyote 1
Case:
A pet timber wolf bit a two year old boy inflicting 41 bite and chew wounds. The father initially refused to allow the state to kill the wolf for a rabies examination. Later he allowed the wolf to be destroyed.

Ursidae
Reports: black bear 4, Malayan bear 1
Case:
A black bear mauled the stomach and leg of a 52 year old lady.
REPORT OF THE COMMITTEE

Rodentia
Reports: woodchuck 1, hamster 1

Felidae
Reports: bobcat 1, couger 3, African lion 7, tiger 4, leopard 2

Case:
An African lion attacked a 28 year old female which resulted in death.

Primates
Reports: marmoset 1, African Red 1, chimpanzee 1, capuchin 1, macaque 1

Case:
A chimpanzee attacked a 25 year old human male who suffered a fractured skull, loss of a finger and a broken arm.

Reptilia
Reports: turtle 1, cobra 1, python 2, diamondback 2, rattler 2, boa constrictor 1

Case:
A python attacked and strangled to death a 33 year old male. Accidents and problems associated with wild and exotic animals continue to be reported (3).

In South Carolina a pet raccoon not only became a financial liability for the state but also affected the lives of at least 25 people. A family picked up some young raccoon kits along the side of the road. Later one of the raccoons (that attained adult size) subsequently developed rabies. Retrospective investigation indicated that at least 16 people had been exposed to this raccoon and another rabid one. The estimated cost of the exposure to the single raccoon was approximately $10,000 (4).

Outside of progressive restrictive action by individual states and municipalities, little recent action has been taken by the federal government.

As evidenced by media reports, owners continue to have difficulty in keeping and handling wild-exotic animals. There is no licensed rabies vaccines for wild or exotic animals available in the United States. Nutritional requirements for keeping wild and exotic animals as domestic animals are not adequately defined. These animals, many recently brought from the wild do not become domesticated nor is their behavior predictable as compared to cats and dogs which have lived in a domestic environment for generations and look to people as protectors.

In 1980 the United States Animal Health Association passed a resolution that stated: "The problem of wild animal/pet associated rabies is an increasing hazard to both man and animals, and whereas no feasible means exist for assuming that wild animal pets especially foxes, skunks, and raccoons are immune to rabies, be it resolved the USAHA requests that the USDA and USPHS together develop rules to prohibit the interstate traffic in skunks, foxes and raccoons." (5)
An alternative to total ban of wild or exotic as pets is that stringent requirements be implemented for keeping of such pets. Contributions to keeping and raising of wild and exotic animals have been made by knowledgeable, lay people.

Education, legislation and enforcement can reduce the number of households keeping wild or exotic animals as pets. Continued concern and action by organizations concerned with animal and human health, such as the United States Animal Health Association can hopefully assist in controlling this increasing problem.

REFERENCES


2. Olson, H.: Personal communication.


REPORT OF THE STATE FEDERAL RELATIONS COMMITTEE

Chairman: J. R. Ragan, Nashville, TN

H. E. Goldstein, OH; B. W. Hawkins, OR; L. W. Hinchman, IN; N. W. Kruse, NB; F. E. Mann, Jr., TX; J. O. Pearce, Jr., FL; G. B. Rea, OR; J. C. Shook, MD; D. U. Walker, VT

The State-Federal Relations Committee of the U.S. Animal Health Association met in College Park, Maryland on January 25-29, 1982. Staff reports and consultations were presented by APHIS, ARS, FSIS and the Office of Transportation from USDA and by the Bureau of Veterinary Medicine, FDA.

Adequate programs in animal health must be maintained in order to control the loss of productivity in domestic livestock production due to disease, to prevent or eliminate incursions of foreign animal diseases, and to preserve a credible animal health basis for international commerce in livestock and livestock products.

The Committee restates its conviction that control and eradication programs for animal diseases should be assigned priority based upon:

1. The relative threat to human health.
2. The level of overall cost of the disease to the livestock industry.
3. Most favorable cost/benefit ratio.

We recognize that funding for needed animal health activities is particularly difficult to generate in the current economic climate. We further understand that budget adjustments will likely be required, and that increased responsibility for support of national programs may have to come from state funding and industry elements closest to the service and benefit.

It is imperative in this situation that truly cooperative program judgements be made with meaningful input from state and federal regulatory sources, practicing veterinarians, and livestock industry representatives.

It is our view that program economies of significant magnitude can be achieved without crippling or dismantling critical, major programs. To do this, every resource involved must be reevaluated and the most cost effective combinations molded. These resources include state personnel, federal personnel, fee basis veterinary services, and the numerous contributions of livestock producers and the marketing industry. Decentralization of decision making and regional program flexibility must be given high priority, but not to the point of losing minimal national program standards. Administrative overhead must not be allowed to consume resources more critically needed in field performance.

It goes without saying that new programs should not be initiated until adequate funding provisions can be made for them. Further, if justifica-
tion and the resource availability are not adequate to carry on active programs in any area, disruptive restrictions should not be placed or maintained on commerce in the industry until the situation changes.

BRUCELLOSIS

The progress of the Brucellosis Program over the last two years has been tremendous, therefore, we urge USDA, APHIS, to do everything possible to keep the program moving ahead.

This committee continues to support the comprehensive past recommendations of the USAHA and charges APHIS staff to pursue all avenues of maintaining the eradication effort.

BLUETONGUE

Continued research commitment to answer some of the perplexing problems in the area of vector-borne diseases is needed.

We are in total agreement with combining the effort on Bluetongue and Rift Valley Fever research at the Denver facility. It should prove to be very cost effective.

This Committee appreciates the cooperation that was demonstrated between ARS and APHIS in this area.

ANAPLASMOSIS

The move of the Anaplasmosis research from Beltsville to Moscow and Pullman Universities from a cost standpoint is certainly sound and from the research point of view the endemic areas in the Northwest is where the work can best produce results, especially in work on vectors.

TUBERCULOSIS

Tuberculosis is still a threat to human health. However, the infection rate has been reduced to a very small percentage in cattle in the United States.

We feel that tuberculosis should be treated as an exotic disease. It is an exotic disease to many states that are accredited free and to those states who are reaching that status.

Such a designation will allow a task-force effort to deal with outbreaks and insure adequate regulatory control.

The outbreak in elk raised in confinement in South Dakota for the production of antlers to be exported to foreign countries for human consumption is a definite threat to our cattle industry.

The Committee feels that serious consideration was not given to the permanent identification of Mexican cattle this past year, and that this effort must be pursued.

SCREWWORM

We support the strong position taken by the USDA, APHIS, in dealing
with the Mexican labor problem in the screwworm plant at Tuxtla Gutierrez.

With the dissension in the Mexico Screwworm Plant as it is, we urge USDA, APHIS, to keep the plant in Mission in such a position that it could be used as a backup plant to keep the Screwworm Program moving South.

**BIOLOGICS**

Agricultural industry requires and is entitled to a continuing supply of safe and efficacious biologics.

The Veterinary Biologics Staff has indicated as a major problem the continued growth of the unlicensed biologics industry, especially with the advent of mobile production laboratories. Lack of efficacy of products produced in unlicensed laboratories is a continuing problem.

The Committee again recommends that APHIS fund a study by the National Research Council into the regulation of both production and sale of animal biologics. Production facilities and product effectiveness should be considered. Enforceable standards for the industry should be established. Subsequently, legislation based on those standards should be developed to guarantee safety and efficacy of biologic products sold in commerce.

The Committee urges that Veterinary Biologics exercise all necessary steps to ensure adequate regulatory control, including licensing and inspection of those firms engaged in the developing field of genetic engineering. High standards in both facilities and expertise are required rather than restrictive controls over product development. Standards for efficacy and safety in this area are of extreme importance to the agricultural industry.

The Committee applauds the cooperation and flexibility exhibited by the Veterinary Biologics staff presentation this year.

**POULTRY**

Continued effort and cooperation between APHIS and the various states is required in order that all states qualify as "Pullorum-Typhoid Clean". The two diseases could then be declared exotic. Industry-government cooperation through NPIP continues to be outstanding. However, particular emphasis must be placed on seeking out and testing small, back-yard flocks where most foci of infection are disclosed and which pose the greatest threat to the commercial poultry industry.

The Committee recommends that a cooperative effort be undertaken, utilizing all necessary means by USDA and the states to eliminate foci of *S. gallinarum* through flock depopulation. Live *S. gallinarum* vaccine or birds vaccinated with such vaccine should be prohibited entry into any state.

Avian Mycoplasmosis in multiple-age laying flocks continues to extract
significant losses from the commercial poultry industry. It appears that such losses could be prevented through a largely self-supporting disease control program. It is also apparent that state laboratories lack sufficient program and diagnostic expertise to control Mycoplasmosis in present circumstances.

The Committee recommends that APHIS and ARS cooperate in a study which, if feasible, would lead to recommendations for establishment of a self-supporting program to control or eradicate *Mycoplasma gallisepticum* in laying flocks. Presumably, APHIS would supply epidemiological support, where necessary, to insure its success.

**EMERGENCY PROGRAMS**

The Committee appreciates the valuable training that is acquired for emergency programs staff and support personnel through the continuing outbreaks of VVND; but it is not very cost effective.

Some way must be found to prevent this continuing drain on tax dollars. Suggestions are:

1. A meeting of all commercially licensed pet bird dealers should be held, indicating the threat to future imports and soliciting their advice and help.
2. Permanent identification of imported birds.
3. Encourage the domestic production on breeder farms.
4. The licensing of every pet bird dealer.
5. Increasing prosecution and fines of smugglers.
6. Strong efforts should be made to prevent a premium for the diagnosis of VVND in an overall attempt to discourage the introduction of this disease.

The computerization of emergency animal disease reporting will go a long way toward reducing the time needed to arrive at the initiating of action to an emergency situation.

Training for emergency disease control should be continued and expanded to include Veterinary Colleges, especially the faculty. This involvement plus the profile gathering of movements and numbers in and out of countries will be most time saving in times of an emergency.

In times of budgetary constraints and prioritization of items receiving funding, this Committee feels strongly that the protection of our domestic livestock population from exotic disease should continue to receive the highest level of concern.

**SWINE DISEASES**

The Committee was disappointed that the scope of swine diseases presented was funneled into discussions of only Pseudorabies and garbage control; therefore it strongly urges APHIS to develop a more
pertinent program of swine disease control that will allow the staff the capabilities of producing viable programs that will be beneficial to the swine industry. It also urges that the ARS continue to involve the programs of research into the priority areas where the economic impact on the swine industry results in unwarranted losses.

Pseudorabies continues to be a major concern of the swine industry in the major hog producing states and evidence indicates that the concern is being exhibited through activities of organizations representing the industry. Steps are being taken to establish a more meaningful rapport between the industry and APHIS, by establishing an advisory committee of producers to assist in modifying sections of the regulations which appear inadequate.

It is gratifying to note that five states have offered to participate in a pseudorabies control model and that proposals for the necessary monies are in place. The necessity for a supplemental budget of at least $1,500,000 is considered to be valid as a step phase development of control features in different socio-geographic environment in the various states even in the face of budget frugality apparent at this time.

The Committee strongly urges the continuation of in-depth research by ARS and the development of practical guidelines by revised regulations in the control phase of this disease by APHIS.

NATIONAL VETERINARY SERVICE LABORATORIES

The Committee appreciated the report on the activities and responsibilities being pursued by the N.V.S.L. in the areas of biologics, antigen production and improved diagnostic procedures. It recognizes the concern that many states are misusing the laboratory’s availability of diagnostic procedures which could be (and should be) conducted on the local level; a process which dilutes the laboratory cadre’s capabilities for necessary developmental diagnostic procedures.

The Committee recommends that a study be conducted to consider the establishment of users fees in the necessary areas to allow the designated purposes of the laboratory to be adequately pursued. Such a procedure would enable the laboratory to furnish both the diagnostic agents, educational programs and procedural surveillance at a level that will allow the state-federal approved laboratories to conduct adequate diagnostic services to their animal industries.

The Committee reiterates its support of the construction of facilities which will allow the consolidation of laboratory processes in a more effective manner.

IMPORT-EXPORT

A review of these programs revealed three important items. (1) A study to determine how the Harry S. Truman Import Station at Fleming Key can most fully and economically be utilized. (2) How the lag-time in processing tourist, immigrants and agricultural products can be reduced
without sacrificing the safety of our livestock population. (3) How tests, inoculations and other treatments and facilities presently being developed may best be applied to enhance our exports to foreign countries.

If all, or even part of these are accomplished, great strides will have been made toward enhancing animal agriculture and thus greatly contributing to the gross national product.

ANIMAL WELFARE

The USAHA continues to be a sounding board for the various individuals, agencies, and organizations who have concerns in the area of animal welfare. We believe that we are providing a forum for the discussion of both the real and the suspected mistreatment of our animal population. The results of the discussions by the Animal Welfare Committee are recorded for advice and counsel to the administration of the Horse Protection Act and the Animal Welfare Act by Veterinary Services.

Resolutions that result from these deliberations and as they are approved by the Executive Committee are recommended to Veterinary Services for implementation. Even though presently there is a wide variation of thought as pertains to this subject, increased interest as shown by requests to participate in these discussions cannot but result in realistic and practical recommendations for the prevention of mismanagement and exploitation of our animal population.

Certain research projects sponsored by these deliberations will separate fact from fiction and will be the basis for needed change in animal husbandry practices.

SCABIES

Scabies continues to persist in the midwest and western states and is a threat to the entire cattle industry. It is a very costly disease to the industry, however it seems to travel mainly to the states with large numbers of feeding operations.

With the introduction of Ivermectin as a treatment for scabies, this Committee feels it will lessen the dipping load for industry and that the scabies regulations should be carefully reviewed and updated. We also recommend that Veterinary Services continue to secure exemption for toxapene from E.P.A for its use in the treatment of scabies. The exemption allows deviation from the label.

The largest problem with scabies eradication is the lack of resources.

PROFESSIONAL DEVELOPMENT

Continued training and development of competent regulatory professional and technical staff is most important. It is necessary that this training continue to be afforded to state personnel since few if any state animal health agencies have the facilities to provide this service. It is
essential that training schedules be provided to all concerned as soon as possible and further that state officials be notified in advance when cooperating personnel are assigned to such schedules.

We reiterate that scheduling of these training sessions on a regional basis will reduce travel costs and make such training available to a greater number of state personnel.

AGRICULTURE RESEARCH SERVICE

The State-Federal Relations Committee of the USAHA met with Dr. T. B. Kinney and ARS Staff members. The Committee was afforded the criteria for evaluating research funding and programming. The criteria for formulating research programs is predicated upon: (a) importance (b) economic relevance, and (c) national and political relevance. The Committee was apprised that the approach for decision making is predicated upon scientific merit and probability of success; the sources must be suitable and available; the results must be practical and have scientific relevance.

It is still quite apparent that funding for animal disease research is in need of constant review in direct relationship to the immediate needs as well as the long range needs to the regulatory and industry requests. For example, this Committee was made aware of a breakthrough involving production of monoclonal antibody which provides the opportunity to differentiate between strain 19 and field brucellosis infection. This Committee most emphatically requests that ARS give top priority to this paramount need for the brucellosis control or eradication effort.

This Committee was made aware that ARS personnel have made a concerted effort to enhance communications with APHIS and FSIS and that this cooperative communication effort will continue.

This Committee is aware of the special study committee appointed jointly by ARS and APHIS Administrators to study the total aspects related to all federal funds involved with animal disease research, resulting from Resolution #17 adopted by USAHA at St. Louis.

This Committee is encouraged that such an undertaking should result in a positive effort to provide much needed animal disease research.

The integrated reproductive research project was discussed and the committee was made aware that the program was totally integrated with extension and teaching to provide efficacy. This Committee commends ARS for this innovative approach.

FOOD SAFETY INSPECTION SERVICES

This Committee was most appreciative and commends the staff of FSIS for providing a complete report in a most informative manner prior to the January 1982 meeting.

It was apparent to the Committee that FSIS Administrators are encouraging existing state meat inspection programs to remain intact. In
order to accomplish the continued maintenance of state meat inspection programs, it is recommended that USDA officials provide the legislative process necessary to provide the state inspected "equal to" products the ability to move in interstate commerce.

The Committee was most encouraged with the FSIS effort in providing animal disease morbidity and mortality data. It is recommended that all effort be maintained to coordinate this program with APHIS, CDC and the respective states through the USAHA Committee on Morbidity and Mortality to implement a viable disease reporting program.

This Committee continues to recommend the implementation of administrative and/or procedural changes which will provide adequate consumer protection at a practical, reasonable expenditure of public funds.

F.D.A.

This Committee was gratified that the Bureau of Veterinary Medicine responded in a positive way to the USAHA resolution regarding the approval of minor use drugs. The proposed regulation to permit use of these drugs is a significant breakthrough. In addition a B. V. M. Joint Standing Committee on the minor use of drugs will be available to confer with interested parties.

It appears the suggestion in our resolution to extrapolate data from drug use in other ruminants as well as data from foreign countries using these drugs have been given favorable consideration.

The final answer to Resolution #16 urging FDA to convene a hearing to finalize a decision regarding the sale of certified raw milk in interstate commerce will be forthcoming. There appears to be some question regarding this interpretation of present requirements as they appear in the Federal Register.

We support those portions of pending legislation which would amend the zero tolerance provisions of the Delaney Amendment and provide for reasonable evaluation of risk/benefit ratios in considering approval of all new drug applications.
REPORT OF THE COMMITTEE ON WILDLIFE DISEASES

Chairman: E. Tom Thorne, Wyoming
Vice Chairman: V. F. Nettles, Georgia

W. D. Bolton, VT; D. R. Cassidy, IA; G. Erickson, IA; M. A. Essey, CA; J. B. Finley, Jr., TX; D. J. Gilhooley, HI; J. H. Gray, TX; F. A. Hayes, GA; D. A. Jessup, GA; D. C. Johnson, GA; R. J. Lee, VA; H. A. McDaniel, MD; E. V. Morse, IN; L. M. Siegfried, WI; J. S. Smith, NV; C. D. Stumpff, KS; G. S. Trevino, TX; W. G. Winkler, GA.

The Committee on Wildlife Diseases convened at 1:30 PM, October 8, 1982. Twelve committee members and eight visitors were present. The first order of business was to review the Committee Report of 1981 and reflect on the status of each item previously recommended for further consideration. Items with apparent need for carry-over from Old and New Business of the 1981 Report herein are considered Old Business. Summary statements with a synopsis of action taken to date are cited as follows.

OLD BUSINESS

1. Memorandum of Understanding and Cooperative Agreement Between APHIS, USDA and State Fish and Wildlife Agencies.

On October 18, 1977, the Wildlife Diseases Committee recognized that the major disease threat to domestic livestock and wildlife of this nation was not from within but from without in the form of a devastating contagion from foreign shores. On October 30, 1978, the Committee proposed that a Memorandum of Understanding between APHIS and state fish and wildlife agencies be established. The purpose of this memorandum was to provide for cooperation in the control, prevention, and eradication of diseases and parasites that now exist or could become active in the United States and may spread among domestic livestock and poultry and wild animals and birds. In 1979, this committee recommended that APHIS work with the Fish and Wildlife Health Committee of the International Association of Fish and Wildlife Agencies in developing this memorandum. Wording was agreed upon in 1980 and the agreements were distributed for signature, beginning in the Southeast. In the 1981 Report, this committee recommended that completion of signing of this document be pursued with diligence and the goal of complete participation be realized as soon as possible.

During this year's meeting of the Wildlife Diseases Committee, Dr. W. W. Buisch reviewed the history of the memorandum and progress in the past year. As of November 5, 1982, 22 states have completed Memorandums of Understanding, 6 memorandums have state signatures and are pending APHIS signature, and remaining states have received documents for signature. If all progresses well, all 50 states could have memorandums in effect by January 1, 1983.

58
WILDLIFE\_DISEASES

RECOMMENDED ACTION: That APHIS, USDA and signatory state fish and wildlife agencies be commended for their foresight and participation, and that APHIS and nonsignatory state fish and wildlife agencies be encouraged to work cooperatively toward completion of this endeavor.

2. Compensation for Relocation of Wildlife in the Event of Depopulation as an Essential Measure for Preventing Spread of a Dangerous Contagious Disease.

In the 1981 Report of this committee, it was recommended that Emergency Programs of Veterinary Services, APHIS, revitalize efforts in seeking the previously proposed memorandum with each state fish and wildlife agency of the United States. This item and subsequent recommendations favoring a reimbursement for relocation agreement between state and Federal agencies has been on the agenda of this committee since 1975. In 1975, legal counsel of APHIS noted that Congress had not provided authority for relocation cost associated with wildlife depopulation during an emergency. State fish and wildlife agencies continue to assume a position of skepticism about depopulating wild animals as part of an animal disease program.

Dr. W. W. Buisch reported to the committee that APHIS is again asking their Office of General Counsel to review this question and, especially, to examine any consideration that might be given during an extraordinary emergency. Although legislation to provide such authority has been proposed in the past by APHIS, success in gaining such authority, nevertheless, has not been achieved.

RECOMMENDED ACTION: That Emergency Programs, APHIS complete research into legalities of Federal reimbursement to state fish and wildlife agencies for relocation costs in the event wildlife depopulation is necessitated by eradication of a communicable disease; furthermore, it is recommended that if it is ruled that such reimbursement is not legally possible, legislative changes making such reimbursement possible be pursued.

3. Foreign Animal Disease Protection Through the U.S. Customs Service

The 1981 Report of this Committee urged careful consideration be given to adopting a resolution relating to a European style “red and green” door policy of voluntary declaration at international ports of entry that had been proposed to U.S. Customs Service. In a report to the committee, Dr. W. W. Buish discussed the background of that resolution and related events since 1981.

In 1980, passengers returning to the United States from foreign travel had increased in numbers to a demonstrated new high. In addition, Congress was actively involved in the issue of expediting passage of passengers through ports of entry. In fact, the “red door-green door” concept of processing passengers was being seriously considered. The
livestock and wildlife industries promptly indicated their concern; and, therefore, a test of alternate procedures at 2 ports of entry was requested by Congress before new procedures would be implemented nationwide. These tests keyed on-use of profiles of individuals suspect of carrying contraband for prioritizing which passengers should be thoroughly inspected. This was in addition to the combined Customs-Immigration referrals from the Customs Declaration Form, interview procedures, and hand carried luggage search. These tests demonstrated a significant improvement in expediting passenger clearance and a significant increase in contraband seized. During 1982, Customs again proposed changes in clearance procedures. Initially, Agriculture and Immigration were not advised of the changes affecting their particular areas of jurisdiction. However, at this time all agencies affected are involved and working together to assure each of their respective concerns are met.

RECOMMENDED ACTION: That APHIS be commended for resisting relaxation of control that is necessary to prevent introduction of a foreign animal disease which could endanger this country’s livestock and wildlife.

4. Bovine Tuberculosis in Pen-raised Elk

In 1981, this committee urged consideration by USAHA of a resolution denoting bovine tuberculosis in certain herds of pen-raised elk.

Dr. Charles D. Stumpff, Veterinary Services, reported to the committee that an outbreak of bovine tuberculosis in captive elk occurred in the fall of 1980 and spring and summer of 1981. In South Dakota, 2 herds were involved. Both herds had experienced death loss, and infection with Mycobacterium bovis was confirmed bacteriologically. Epidemiologic tracing involved possible exposed and source herds and resulted in 10 herds of elk tested for tuberculosis in 7 states. There were 2 herds of cattle which were possibly exposed; but as of this date, spread to cattle has not been confirmed. Two elk herds have been completely destroyed. One herd remains in existence with 10 of 56 animals remaining. Suggested procedures to control such outbreaks and tuberculosis testing techniques were discussed by Dr. Stumpff.

RECOMMENDED ACTION: Veterinary Services and state livestock, fish and wildlife, and public health agencies should be commended for investigating and controlling this outbreak of bovine tuberculosis in captive elk.

5. Epizootic Hemorrhagic Disease and Bluetongue in White-tailed Deer

Dr. Victor F. Nettles reported on hemorrhagic disease in white-tailed deer during the past year.

White-tailed deer often develop severe illness when infected by either of two viruses, bluetongue virus (BT) or epizootic hemorrhagic disease virus (EHD). Collectively, this disease syndrome is termed hemorrhagic disease (HD) because the illnesses are clinically and pathologically in-
distinguishable. Hemorrhagic Disease has occurred in sporadic outbreaks each summer or fall since 1971.

In the summer and fall of 1982, HD infections were infrequent in the Southeast and mortality was noted only in South Georgia and southern South Carolina. Several viral isolates have been sent to the National Veterinary Services Laboratory (NVSL) in Ames, Iowa, for identification and typing. Hemorrhagic disease has not been confirmed in states outside the Southeast in 1982.

In September, 1982, a memorandum was sent from the Southeastern Cooperative Wildlife Disease Study to state and Federal wildlife management personnel in 16 southeastern states regarding procedures for diagnosing HD in white-tailed deer. This action was taken in order to expedite the diagnosis of HD outbreaks through greater collaboration between field wildlife biologists and foreign animal disease diagnosticians of Veterinary Services, USDA. Although these sporadic outbreaks of HD are relatively common, complacency toward diagnosis is dangerous since many lesions of HD resemble those of foot-and-mouth disease or rinderpest.

RECOMMENDED ACTION: The Wildlife Disease Committee views the present cooperation of Veterinary Services and the National Veterinary Services Laboratories with wildlife agencies as highly commendable. The committee encourages APHIS to continue their collaborative role in surveillance and applied research of BT and EHD in wild ruminants.


At last year's meeting the committee commended the U.S. Fish and Wildlife Service (USFWS) for initiating a DVE survey in waterfowl. The goal in this work was to determine if DVE was well entrenched in free-flying populations. To date, the USFWS has surveyed mallards in the Central and Mississippi Flyways and a large sample from Lake Andes National Wildlife Refuge in South Dakota. Virus was not detected in either flyway or on Lake Andes. Statistically, the sample size was adequate to detect an infection rate of 0.5 percent with 90 percent confidence.

The USFWS's National Wildlife Health Laboratory states that the work to date does not answer the question of whether or not DVE is enzootic in migratory waterfowl. However, their interpretation of the results was that the data suggest that DVE virus in these flyways was below levels reflective of an enzootic situation.

RECOMMENDED ACTION: The USFWS should be praised for taking these steps toward finding the source of DVE and is encouraged to continue with surveillance in other species and locations. Furthermore, since many DVE outbreaks have occurred in widely separated small municipal flocks of semi-tame ducks in settings where wild waterfowl
could become exposed, it would be wise to survey such birds for virus as well as wild waterfowl.

NEW BUSINESS


Dr. Frank A. Hayes reported on the circumstances surrounding an emergency deer depopulation on Conservation Area 3 in the Florida Everglades in July, 1982. Until June, the area had been under drought conditions and an estimated 6,000 deer roamed over 500,000 acres of sawgrass savannah habitat. Two tropical depressions resulted in heavy rainfall and water control activities led to the flooding of the area with 2 to 3 feet of water. Flooding reduced the available deer habitat to 5,000 acres, 1 percent of the prior area. Because of this flooding and habitat reduction, the deer quickly became victims of malnutrition, stress, and extreme parasitism. The Florida Game and Fresh Water Fish Commission quickly made plans for an emergency hunt; however, well meaning but misinformed citizens opposed the hunt. Subsequent court restraints were sought by deer protectionists in state and Federal Courts and this controversy attracted nationwide news coverage.

A compromise decision was reached whereby only part of the area would be hunted. Deer rescue attempts were allowed in the rest of the area; however, they proved futile. The net result was that deer in the unhunted area died in greater numbers and in a far more inhumane manner than the hunted population.

RECOMMENDED ACTION: State and Federal Agricultural Agencies should recognize the recent public opposition to killing deer in the Everglades as indicative of future public relations problems regarding possible depopulation of wildlife in foreign animal disease eradication or control programs.

2. *Heartwater Disease in the Caribbean.*

The discovery of heartwater infections in Guadaloupe and the presence of the bont tick, *Amblyoma variegatum*, on several Caribbean islands should be viewed as a significant potential wildlife disease problem. In the historic heartwater regions of Africa, some species of wild ruminants act as natural asymptomatic hosts whereas others can be clinically infected. Thus, it is reasonable to predict that white-tailed deer populations, which virtually blanket the tropical and subtropical portions of the southeastern United States, would readily become an important factor if heartwater were introduced.

Furthermore, white-tailed deer in the southeast are important hosts for native species of *Amblyomma* spp. ticks including the Gulf Coast tick, *A. maculatum*. Experimental studies have shown that *A. maculatum* can transmit heartwater and one heartwater authority suspects that the ubiquitous Lone Star tick, *A. americanum*, also is capable. These potential problems could be further compounded by the likelihood that
the exotic bont tick, *A. variegatum*, probably would find deer a suitable host.

The committee was informed that experimental infections of heartwater in white-tailed deer are being planned through the mutual efforts of APHIS, the Plum Island Animal Disease Center, and the Southeastern Cooperative Wildlife Disease Study. Additionally, tick surveillance in white-tailed deer has been initiated in Florida to monitor potential bont tick introduction.

RECOMMENDED ACTION: The Committee commends the USDA for their foresight in seeking this much needed information.


Dr. Erskine V. Morse reported on a survey that was conducted in Tippecanoe County, Indiana during 1979–80 to ascertain the prevalence of *Salmonella* spp. in raccoons. Feces and mesenteric lymph nodes from 106 raccoons were examined and 43 *Salmonella* isolations were made from 33 (33.1%). Twelve serotypes were isolated. Two raccoons harbored 3 serotypes, while 6 others were colonized by 2 serotypes. Salmonellae were cultured from the mesenteric lymph nodes more frequently than from fecal samples. The animals did not give evidence of clinical salmonellosis, and pathologic changes directly attributable to salmonellosis were not observed.

Raccoons of urban origin were 30.8% positive; rural animals were 34.7% positive. Young were 33.3% positive and salmonellae were isolated from 29.6% of the adults. *Salmonella typhimurium* and its variant Copenhagen were the most frequently cultured salmonellae (35%). The antimicrobial resistance of the isolates was minimal to 12 commonly used drugs in animal or human medicine. Raccoons may serve as a reservoir of *Salmonella* infection for other wildlife, livestock, pets, and human beings.

RECOMMENDED ACTION: None required.

4. *Vesicular Stomatitis* Outbreaks in the Southwestern and Rocky Mountain States

At the request of the Committee, Dr. James S. Smith reported on the extensive outbreak of vesicular stomatitis (VS) during the past summer and fall. Dr. Smith reported that a major epizootic of New Jersey strain VS began in May, 1982 and involved about 750 premises in 10 states. Cattle, horses, sheep, goats, bison, dogs, and humans were found to be infected by either virus isolation or serology.

To date, the extent of wildlife involvement in this large-scale epizootic is unknown. Clinical disease was not reported in wild species, but an extensive search was not made. Serologic tests conducted in Colorado have demonstrated exposure of pronghorn antelope to VS virus, and trial inoculation of VS virus into pronghorns is planned in Wyoming.

RECOMMENDED ACTION: Because vesicular stomatitis is clinically
indistinguishable from foot-and-mouth disease and because vesicular stomatitis may have serious effects upon free-ranging wild animals, Veterinary Services and state livestock and fish and wildlife agencies should be encouraged to work cooperatively to continue surveillance for vesicular stomatitis and to promulgate research into the epidemiology of epizootic and enzootic vesicular stomatitis in domestic and wild animals.


The successful restoration of wild turkey populations has been based primarily on translocation of wild-caught adult birds from well established flocks into new areas of suitable habitat. As a result, most states having wild turkeys also have range expansion programs that involve bird relocation by state fish and wildlife agencies. Overall these projects have been a credit to wildlife management and have accounted for substantial increases in wild turkey numbers in previously void areas.

As the popularity of wild turkey translocation has increased, interstate movements of birds has become more frequent. Disease and parasite monitoring of wild turkeys has become more detailed and translocated birds are being given much deserved attention. Of interest to the Wildlife Disease Committee is the question of Mycoplasma gallisepticum (MG) infections in wild turkeys. The status of this disease in wild turkeys is unknown, but several investigators have independently reported evidence of MG. Serologic positive wild turkeys have been found in several states, but there is some confusion over possible false positives caused by using frozen serum for the plate agglutination test.

Mycoplasma gallisepticum has been isolated from wild turkeys in California and Georgia and an undetermined species of Mycoplasma has been recovered from a declining population of wild turkeys in Colorado. In these instances, however, the birds were in close association with farm yard poultry or were captive wild birds. Unclassified Mycoplasma also have been cultured from wild turkeys in Texas and Virginia.

The significance of MG to the domestic poultry industry is well known. Whether true wild turkeys harbor the organism is doubtful, but the question must be considered. According to APHIS Poultry Disease Epidemiologist, Dr. Darrell Johnson, backyard poultry and game birds are considered reservoirs for M. gallisepticum. The use of artificial feeding of wild turkeys in the proximity of domestic birds should be discouraged. Furthermore, release of pen-reared "wild" turkeys still is a popular practice among private citizens, hunting clubs, and shooting preserves and may be a problem of mutual concern to wildlife and agricultural interests.

RECOMMENDED ACTION: It is recommended that the Wildlife Disease Committee of the USAHA encourage investigation of Mycoplasma in wild turkeys. Committee members should help state agricultural and state wildlife officials work together to assess this
potential problem by methods that will not impede the progress of wild
turkey restoration.


Throughout the United States, wildlife mortality and morbidity
problems often are investigated by wildlife veterinarians employed by
state or Federal wildlife agencies. These persons are responsible for the
accurate diagnosis in situations where the investigators are hampered by
inadequate histories, meager pathologic descriptions, and partially
decomposed specimens. Thus, early recognition of diseases in wild
species that are of significance to domestic livestock or poultry is not a
simple task.

Because many foreign animal diseases could infect our native wildlife
species and because foreign animal disease could enter wildlife
populations prior to their detection in domestic animals, wildlife
veterinarians should be trained in the recognition of major foreign animal
diseases. Few wildlife veterinarians have seen foreign animal diseases;
and therefore, our nation's defenses against one avenue of foreign animal
disease is not well fortified. The Committee on Wildlife Diseases sent a
resolution to be considered by the Resolutions Committee.

8. Support for APHIS's Programs to Protect the Domestic Livestock
   Industry and Wildlife Resources of this Nation.

Dr. Frank Hayes presented a resolution to the Committee supporting
the maintenance of APHIS's posture on the high priority of import
regulations in the prevention of foreign animal diseases. This resolution
will be introduced via the Import-Export Committee and the Zoological
Committee also will be a co-sponsor. The Wildlife Diseases Committee
agreed unanimously to also co-sponsor a resolution sent to the
Resolutions Committee.

Mr. Chairman:

This report has been respectfully submitted with a motion for its
acceptance.

E. Tom Thorne
Chairman
ECONOMIC AND EPIDEMIOLOGICAL IMPLICATIONS OF ANAPLASMOSIS IN TEXAS CATTLE HERDS

F. J. Alderink
Veterinary Services APHIS USDA
Hyattsville, Maryland

R. A. Dietrich
Department of Agricultural Economics
Texas A&M University
College Station, Texas

In 1979 the US Animal Health Association's committee on anaplasmosis stated there was a need to know the incidence of anaplasmosis in the United States. In 1980 the committee stated that research on vector identification should be accelerated and field studies be conducted to evaluate methods of anaplasmosis control and to determine which methods of prevention were used in herds of high risk.

The incidence of clinical cases reported previously within anaplasmosis affected herds was 1 to 2% in 2 California beef herds and in 1 Montana beef herd. This paper provides estimates on the incidence of clinical anaplasmosis in Texas derived from a definitive survey of producers. It adds evidence difficult to refute to the long held suspicion that the winter tick is a natural vector of Anaplasma marginale. This Texas study incriminated horseflies as the principal vector during the summer, as does other research reported previously.

Prevention of disease may be accomplished by (1) increasing the resistance of the host, (2) decreasing the infectivity of the disease agent, or (3) preventing transmission of the disease agent from the source to the susceptible host. All three methods were used by Texas producers to prevent clinical cases of anaplasmosis. The principal methods used in 1980 were (1) vaccination of cattle, (2) feeding a low level of chlorotetracycline to cattle and thereby reducing A marginale's ability to cause disease, and (3) control of ectoparasites. These 3 methods of anaplasmosis control when used in herds of high risk indicated they were all cost beneficial.

A $10 million loss due to anaplasmosis was reported for Texas in 1968. This estimate was based on the opinion of an expert knowledgeable about the disease. An estimate of a $5,244,318 cost of anaplasmosis to California beef cattle producers during 1976 was based on survey results. The California study divided the cost of anaplasmosis into its components of death, reduced calf crop, increased cull rate, veterinary fees, and treatment costs. The Texas study reported here provides detail on anaplasmosis caused losses and extrapolates the direct costs of anaplasmosis to the state as a whole.

SOURCE OF DATA

Two mail questionnaires, 1 to practicing veterinarians and 1 to beef cattle producers, were designed to obtain the necessary data for this
study. A random sample of 307 veterinarians was selected by using a random number table. A random sample of 2,297 producers was drawn in cooperation with the Texas Department of Agriculture and the USDA. The questionnaires were designed to include only cattle 2 years old and older, since cattle under 2 years of age do not show clinical anaplasmosis or only mild disease with minimum loss.

Eighty-three veterinarians and 499 producers returned usable questionnaires for response rates of 27% and 21.7% respectively.

The producer responses were divided into 2 categories: (1) herds with a recent history of anaplasmosis, and (2) herds with no recent history of anaplasmosis. Herds with a recent history were defined as herds with clinical cases during one or more of the years 1978, 1979, and 1980. However, annual estimates of incidence, losses and economic costs were based on the calendar year 1980.

EPIDEMIOLOGICAL RESULTS

Incidence

Incidence\textsuperscript{a} of clinical cases\textsuperscript{b} of anaplasmosis was estimated from the producer's responses. Fig 1 depicts the marked variation in incidence between different geographic areas of Texas. Area 3, the cross hatched parts of Fig 1, was the area where over 80% of the clinical cases reported by survey respondents occurred. This area is divided into the 4 subareas; 3a, 3b, 3c and 3d with respective incidences of 2.2, 0.6, 0.9, and 1.0\textsuperscript{\textcircled{a}} (Table 1).

The gray part of Fig 1, area 2, shows the area of Texas that had an average incidence of less than 0.1\% (Table 1).

The non-shaded parts of the map in Fig 1 were designated area 1. No clinical cases were reported by the survey respondents from this area except for the 2 small localities indicated by gray in West Texas. One ranch in the Panhandle and 3 or 4 ranches in the Trans Pecos region of Texas had a minor problem with anaplasmosis. In West Texas the disease appeared to be restricted to these 2 localities.

The extrapolation of the incidence for area 2 and the subareas of area 3 (Table 1) onto the cattle population at risk for each area, provided an estimate of 15,015 clinical cases of anaplasmosis occurring in Texas during 1980. The 4 cases reported from West Texas were not extrapolated onto the cattle population of subarea 1a. The population at risk was the number of "cows and heifers that have calved" in Texas from the 1978 Agricultural Census\textsuperscript{\textcircled{a}} adjusted for bulls. The adjustment for bulls was 1 bull added for each 25 "cows and heifers that have calved."

\textsuperscript{a} Incidence as used in this paper is the percent of the cattle over 2 years of age diagnosed as becoming clinically affected with anaplasmosis during the period of 1 year.

\textsuperscript{b} Clinical cases were determined by the veterinarians in their survey and by the producer's veterinarian or the producer in the producer's survey by whatever methods they used to arrive at a diagnosis.
Fig 1. Geographic areas of incidence of anaplasmosis in Texas as determined in 1980.  

- Area 1 where no clinical cases or cases were rarely reported.  
- Area 2 where 0.076 cases/100 cattle at risk were reported.  
- Area 3 where 0.6 to 2.2 cases/100 cattle at risk were reported.
ANAPLASMOSIS IN TEXAS CATTLE

Table 1. Number of Beef Herds and Cattle, and Clinical Cases of Anaplasmosis from a 1980 Random Survey of Texas Beef Producers by Geographic Area of Incidence

<table>
<thead>
<tr>
<th>Area</th>
<th>Number of Herds</th>
<th>Number of Cattle</th>
<th>Number of Clinical Cases</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Area 1</td>
<td>101</td>
<td>25,365</td>
<td>4</td>
<td>0.0158</td>
</tr>
<tr>
<td>Subarea 1a</td>
<td>73</td>
<td>20,745</td>
<td>4</td>
<td>0.0193</td>
</tr>
<tr>
<td>Subarea 1b</td>
<td>28</td>
<td>4,620</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Area 2</td>
<td>286</td>
<td>50,153</td>
<td>38</td>
<td>0.0758</td>
</tr>
<tr>
<td>Area 3</td>
<td>112</td>
<td>15,385</td>
<td>189</td>
<td>1.228</td>
</tr>
<tr>
<td>Subarea 3a</td>
<td>28</td>
<td>4,110</td>
<td>91</td>
<td>2.214</td>
</tr>
<tr>
<td>Subarea 3b</td>
<td>21</td>
<td>3,622</td>
<td>23</td>
<td>0.6350</td>
</tr>
<tr>
<td>Subarea 3c</td>
<td>52</td>
<td>5,055</td>
<td>47</td>
<td>0.930</td>
</tr>
<tr>
<td>Subarea 3d</td>
<td>11</td>
<td>2,598</td>
<td>28</td>
<td>1.078</td>
</tr>
<tr>
<td>Total Survey</td>
<td>499</td>
<td>90,903</td>
<td>231</td>
<td>0.254</td>
</tr>
</tbody>
</table>

aGeographic areas of incidence as delineated in Fig 1.

bClinical cases were determined by the producer's veterinarian or the producer in the producer survey by whatever methods they used to arrive at a diagnosis.

cIncidence is the percent of the cattle over 2 years of age diagnosed as becoming clinically affected with anaplasmosis during the period of 1 year.

Seasonal Occurrence and Vectors

The veterinarian respondents reported 1,290 cases during a typical year. Of the 1,290, 663 or 51.4% were diagnosed during the 3 months of July, August and September with horseflies designated as the principal vector. Only 175 cases were reported to occur during the winter months of December, January, February and March. However, 58.9% (103) of these winter cases were reported from the Edward's Plateau. By contrast, the Plateau accounted for only 6.6% of the summer cases. The winter tick, Dermacentor albipictus was incriminated by the veterinarians as the winter vector, a fact that is difficult to refute, as it is the only vector (except for man) that is active at that time of the year.

ECONOMIC RESULTS

The direct costs of anaplasmosis to Texas beef cattle producers were estimated by applying 1980 market prices12 for cattle to the anaplasmosis caused losses the producer survey respondents reported.

Physical Losses

The producer respondents reported 231 clinical cases of anaplasmosis in 1980 (Table 1). The physical losses associated with these clinical cases as determined from the producer survey and their dollar cost are tabulated in Table 2. The 83 deaths compose 35.9% of the 231 clinical cases. The average weight lost by the 148 surviving clinical cases was 86
kg (190 pounds). The proportionate weight lost by bulls was 136 kg (300 pounds).

Forty-two of the 148 survivors or 28.3% became chronic cases (Table 2). Cattle convalescing from anaplasmosis have a recovery period of 1 to 3 months while replacing lost erythrocytes and regaining their weight. Cattle that did not regain their weight or had a longer recovery period were classed as chronic cases. The estimated loss due to chronic cases was determined from the difference in value between an average brood cow or bull and a thin cow or bull of lighter weight and lower price per pound; both a direct result of the animal becoming a chronic case.

Thirty calves were lost due to anaplasmosis induced abortion from 112 surviving pregnant cows. Fourteen of the 148 survivors were bulls, 22 of the 134 cows were not pregnant (Texas beef cows have an 84% calf crop)*3, which leaves 112 pregnant cows. A 50:50 sex ratio was assumed for the 30 calves (Table 2).

Cost of Treatment

The average cost of treatment per clinical case of $65.44 was composed of $34.54 for drugs and/or veterinary service and $30.90 for labor and extra husbandry required to handle the case.

The sum of the losses sustained by the producer respondents during 1980 due to death, weight loss, chronic cases, abortions and cost of treatment and labor is estimated to be $97,984 for 231 cases of anaplasmosis (Table 2). This is an average cost of $424.17 per clinical case. The 15,015 total annual cases estimated for Texas in the Incidence section of this report times $424.17 resulted in a total estimated annual cost of anaplasmosis caused losses to Texas beef producers of $6,368,912.

Cost of Preventive Measures

Only data from herds with a reported anaplasmosis problem were used to evaluate the different control methods. Herds were assigned to the principal control method utilized on them. According to the producer survey, no preventive measures were used in area 1 (Fig 1).

The herd owners who relied predominantly on vaccination as a control method protected 3,593 head of cattle for a vaccine and labor cost of $6,225 which computes to an average annual per head cost of $1.73. The vaccination interval used by Texas producers, was equivalent to vaccinating the herd once every 2.4 years. If the manufacturer's recommendations of vaccinating every 2 years were followed, the average per head cost would have been $2.11. The total cost of all producer respondents during 1980 for vaccine and the labor to administer it was $9,035.

*3 "Anaplaz" produced by Fort Dodge Laboratories was the only vaccine used by the producers surveyed.
Table 2. Number of Clinical Cases of Anaplasmosis, by Type of Physical Loss and Associated Dollar Loss and Total Dollar Loss, and Treatment Cost, Random Survey of Beef Producers, Texas, 1980

<table>
<thead>
<tr>
<th>Item</th>
<th>Number of Clinical Cases and Physical Losses</th>
<th>Value Per Case or Physical Loss</th>
<th>Total Survey Losses and Costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical cases</td>
<td>231</td>
<td>424.17</td>
<td>97,984</td>
</tr>
<tr>
<td>Death loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cows</td>
<td>75</td>
<td>575.00</td>
<td>43,125</td>
</tr>
<tr>
<td>Bulls</td>
<td>8</td>
<td>1,250.00</td>
<td>10,000</td>
</tr>
<tr>
<td>Totals</td>
<td>83</td>
<td></td>
<td>53,125</td>
</tr>
<tr>
<td>Survivors</td>
<td>148</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cows</td>
<td>133.8</td>
<td>81.70</td>
<td>10,931</td>
</tr>
<tr>
<td>Bulls</td>
<td>14.2</td>
<td>174.00</td>
<td>2,471</td>
</tr>
<tr>
<td>Totals</td>
<td>148.0</td>
<td></td>
<td>13,402</td>
</tr>
<tr>
<td>Chronic cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cows</td>
<td>38</td>
<td>131.00</td>
<td>4,978</td>
</tr>
<tr>
<td>Bulls</td>
<td>6</td>
<td>637.50</td>
<td>2,550</td>
</tr>
<tr>
<td>Totals</td>
<td>42</td>
<td></td>
<td>7,528</td>
</tr>
<tr>
<td>Abortions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heifer calves</td>
<td>15</td>
<td>241.50</td>
<td>3,622</td>
</tr>
<tr>
<td>Steer calves</td>
<td>15</td>
<td>346.00</td>
<td>5,190</td>
</tr>
<tr>
<td>Totals</td>
<td>30</td>
<td></td>
<td>8,812</td>
</tr>
<tr>
<td>Cost of treatment and labor</td>
<td>231</td>
<td>65.44</td>
<td>15,117</td>
</tr>
<tr>
<td>Total losses and costs</td>
<td></td>
<td></td>
<td>97,984</td>
</tr>
</tbody>
</table>

The producers feeding a low level of chlortetracycline as the primary method of control fed it to 3,824 head at an average annual cost of $2.88 per head. The total cost of chlortetracycline for all respondents using it was $12,499.

Vector control was the principal control method used on 2,794 head of cattle at an average annual cost of $4.53 per head. The total expenditure for vector control reported by the survey respondents to prevent anaplasmosis was $12,650.

The use of injections of oxytetracycline was of minor importance as a control method. The producers surveyed reported using $3,175 worth of oxytetracycline along with a labor cost of $1,365 for administering the drug, for a total of $4,450.

The total expenditure by Texas cattle producers for preventing anaplasmosis was estimated by multiplying the survey respondent's
expenditure by an expansion factor. The expansion factor was the cattle population at risk (see *Incidence* section) in areas 2 and 3 (Fig 1) divided by the cattle population surveyed in areas 2 and 3 (Table 1). This total expenditure by Texas producers during 1980 was $2,587,411.

*Total Cost of Anaplasmosis in Texas*

The sum of the losses due to anaplasmosis and cost of treatment ($6,374,882) and the prevention costs ($2,589,411) resulted in an estimated total cost to Texas beef cattle producers equal to almost $9.0 million during 1980.

**BENEFIT/COST RATIOS AND NET BENEFITS**

Herds from area 3 (Fig 1), the regions of Texas with a high incidence of clinical cases, were used to estimate the benefit/cost ratio and net benefits of three control methods. Herds were selected in which the control measure had been used more than 1 year and the no control group of herds belonged to producers who had lived with anaplasmosis more than 1 year. Herds in which one of the control methods was used were compared to the non-control group of herds. The incidence of clinical cases within herds was found to vary with herd size. For example, the incidence within herds of 300 head or less in the no-control group was 3.57, while herds over 300 head on which no control was used had an incidence of 1.44. To reduce this effect of herd size on the incidence of clinical cases within the herds using the control measures, cost benefit analyses were performed on 2 herd sizes. The 122 head herd was the average size of the herds with 300 head or less, and the 553 head herd was the average size of the herds larger than 300 head (Table 3).

The incidence determined from the survey for herds of 300 head and less for each preventive category was applied to the 122 head herd and similarly the incidence of the larger herds was applied to the 553 head herd to estimate the number of clinical cases in each. The same proportion of losses and costs determined from the producer respondents (Table 2) were used on the number of clinical cases resulting under each control method to arrive at the cost benefit analyses summarized in Table 3.

In the 122 head herd, low level chlortetracycline and vector control had similar returns of $4.02 and $3.84 for each dollar of cost for the control program. However, vector control had a $500 advantage in net benefit over low level chlortetracycline because the higher cost of vector control over feeding chlortetracycline had less effect on net benefit than on the benefit/cost ratio. In the 553 head herd, vaccination had a higher benefit/cost ratio compared to low level chlortetracycline, $3.20 compared to $2.16, for each dollar spent on each control method. Vaccination also had a $900 advantage in net benefit. The results of this cost benefit analysis favors vaccination over the other 2 preventive methods. A consideration not accounted for in this analysis is that vector control has the benefit of reducing blood loss, irritation, and worry of cattle by ectoparasites in
ANAPLASMOSIS IN TEXAS CATTLE


<table>
<thead>
<tr>
<th>Control Method</th>
<th>Reduction of Production Costs</th>
<th>Increase in Total Value of Product</th>
<th>Returns to Control Method</th>
<th>Cost of Control Method</th>
<th>Benefit/Cost Ratio</th>
<th>Net Benefit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaccination</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low level chlortetracycline</td>
<td>951.09</td>
<td>461.22</td>
<td>1,412.31</td>
<td>351.36</td>
<td>4.02</td>
<td>1,060.95</td>
</tr>
<tr>
<td>Vector control</td>
<td>1,370.46</td>
<td>750.51</td>
<td>2,121.95</td>
<td>552.66</td>
<td>3.84</td>
<td>1,569.31</td>
</tr>
<tr>
<td>Vaccination</td>
<td>2,499.88</td>
<td>1,237.11</td>
<td>3,736.99</td>
<td>956.69</td>
<td>3.91</td>
<td>2,780.30</td>
</tr>
<tr>
<td>Low level chlortetracycline</td>
<td>2,321.07</td>
<td>1,125.38</td>
<td>3,446.45</td>
<td>1,592.64</td>
<td>2.16</td>
<td>1,853.81</td>
</tr>
<tr>
<td>Vector control</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

\*Production costs include losses due to death loss, weight loss, chronic cases, and the cost for veterinary service and labor used on the clinical cases.

\*Increase in Total Value Product = increase in feeder calves sold as a result of reduced abortion rate and reduced replacement calves required to replenish losses due to death and culled chronic cases.

\*Benefit/Cost Ratio = Returns to Control Method/Cost of Control Method.

\*Net Benefit = Returns to Control Method - Cost of Control Method.

No producer with a herd size < 300 head (average 122 head) reported using vaccination as the principal anaplasmosis control method in Area 3.

No producer with a herd size > 300 head (average 553 head) reported using vector control as the principal anaplasmosis control method in Area 3.

In addition to reducing the incidence of clinical cases of anaplasmosis. Vaccination and feeding a low level of chlortetracycline have no or very little benefit to the producer other than reducing the incidence of clinical anaplasmosis.

DISCUSSION

The necessary requirements for clinical cases of anaplasmosis to occur are: (1) the disease agent, *A. marginale*, (2) susceptible hosts, and (3) vectors to transmit the agent.

The variation in the incidence of clinical cases of anaplasmosis between different geographical areas of Texas is probably due to a difference in
the numbers and activity of vectors between the areas (requirement 3). Horsefly numbers attain high levels in parts of area 2, but these high levels are not constant throughout the summer season as they seem to be in subareas 3b, c, and d. The winter tick range includes much of Texas, but the surveys indicate this tick is much more of a problem in the Edwards Plateau than in other areas of the state.

Requirement 1 is satisfied as it is very unlikely the areas of Texas with no or very few clinical cases of anaplasmosis do not have cattle infected with and carrying *A. marginale* as a reservoir of the disease agent. The frequent moving of cattle between areas through constant buying and selling and ranchers moving cattle between widely separated land holdings is assurance no large area of Texas remains without carrier cattle as a source of infection.

With regard to requirement 2, the presence of susceptible hosts, we are not aware of evidence of any herd becoming 100% infected through natural transmission so that all the animals are carriers and thereby, resistant to becoming a clinical case. Consequently, non-vaccinated herds can be expected to contain cattle susceptible to anaplasmosis.

The total estimated annual cost of anaplasmosis caused losses in Texas of almost $6.4 million is conservative. Many veterinarians observed the number of clinical cases of anaplasmosis during the summer of 1980 was relatively low compared to many previous years because of the dry summer and reduced number of vectors and their activity. It follows that if the number of cases was less, the losses and economic costs would also be less.

Data on preventive methods obtained from producers were largely economic. The cost of the control methods was requested in the questionnaire but not the level of chlortetracycline fed nor its timing with the anaplasmosis season. Likewise, the insecticide used for vector control and method of application were not requested. However, the results from the producer survey indicated vector control at an annual cost of less than $3.00 per head for insecticide did not satisfactorily reduce the clinical incidence of anaplasmosis.

As would be expected, the economic benefits from control programs were greater where anaplasmosis losses were higher. The net benefits calculated were for herds in area 3 where herds with an anaplasmosis problem had a higher within herd incidence than in area 2. In area 2, net benefits from control programs were much less in smaller herds than those listed in Table 3 for smaller herds in area 3. Returns to anaplasmosis control programs in larger herds in area 2 were negative. Owners of these herds were using anaplasmosis preventive measures as insurance against large potential losses rather than as a productive input to increase profit.
ANAPLASMOSIS IN TEXAS CATTLE

REFERENCES


15. Teel PD: Seasonal and Geographical Distribution of Ticks Parasitizing Cattle in Texas. Unpublished paper. Dept of Entomology, Texas A&M Univ, College Station, TX, 1981.
ULTRASTRUCTURE AND DEVELOPMENT OF COLONIES OF ANAPLASMA MARGINALE IN THE GUT OF INCUBATED DERMACENTOR ANDERSONI

Katherine M. Kocan, PhD; Donald Holbert, PhD; S. A. Ewing, DVM, PhD; Jakie A. Hair, PhD; Selwyn J. Barron, BVSc.

From the Department of Veterinary Research (Kocan, Barron) and the Department of Parasitology, Microbiology and Public Health (Ewing), College of Veterinary Medicine; the Department of Statistics (Holbert); and the Department of Entomology (Hair), Oklahoma State University, Stillwater, Oklahoma, 74078.

Supported by USDA-SEA, Animal Health Program, Grant No. 5-5-23820 and Oklahoma Agricultural Experiment Station Project Numbers: 2-1-11480 and 2-1-11441.

Professional paper 1253 of the Oklahoma Agricultural Experiment Station, Stillwater, Oklahoma, 74078.

The authors thank Wanda Edwards, June Willis and Gayle Arbaugh for technical assistance and Nancy Stratton for clerical assistance.


SUMMARY

Colonies of Anaplasma marginale Theiler were studied in midgut epithelial cells of adult Dermacentor andersoni Stiles that were infected as nymphs. The colonies were categorized by light microscopy into 5 morphologic types that appear to represent stages in a developmental sequence. Representatives of each colony type were selected with the light microscope and sectioned for examination by electron microscopy. The morphology of individual A. marginale organisms within colony types varied and included: small electron-dense forms, larger reticulated forms, pleomorphic reticulated forms and small particles.

Three splenectomized dairy calves were inoculated with a Virginia isolate of Anaplasma marginale and served as an infective source for laboratory-reared Dermacentor andersoni nymphs. One month after molting, groups of adult ticks were incubated at 37 C for 0, 1.5, 2.5 and 7 days. Tissue sections of gut were prepared from ticks in each incubation period in 3 trials and examined by light microscopy for colony density, diameter and type of colonies of A. marginale. The mean colony density for all trials of incubated ticks ranged from 2.23 to 7.32 colonies per .001 mm² gut tissue. While the range of colony density was quite wide, sec-
tions of gut from 345 of 346 ticks examined contained colonies. Both male and female ticks were infected in all experimental groups. Although variation of colony density among incubation groups of each trial was not statistically significant, the number of colonies per .001 mm² gut tissue was greatest in the 7-day group in Trial 1 and in the 2.5-day group in Trials 2 and 3. The colonies were classified according to morphologic characteristics (colony types 1-5) and all colony types were present in each incubation group from the 3 trials. Although no distinct pattern of distribution of colony type was apparent, there were fewer Type 1 colonies in the non-incubated groups of each trial than in any others; and Type 1 colonies were most frequently observed in 7-day groups, especially in male ticks. In all 3 trials Type 3 colonies were the most frequently observed type in ticks that were incubated 2.5 days. The mean diameter of each colony type was progressively larger in all 3 trials, ranging from 5.63 μm (for Type 1) to 13.33 μm (for Type 5). In most groups, the average diameter of colonies of all types was larger in male ticks than in female ticks.

INTRODUCTION

*Anaplasma marginale* Theiler was recently described by light and electron microscopy in midgut epithelial cells of adult *Dermacentor andersoni* Stiles and *Dermacentor variabilis* (Say) that were infected as nymphs(1). Fluorescent antibody (FA) studies of *A. marginale* in *D. andersoni* demonstrated positive fluorescence of gut tissues from feeding adult ticks(2). Ferritin-labeled antibody studies confirmed the identity of *A. marginale* in similar tissues with the electron microscope(3).

Colonies of *A. marginale* were recently described in midgut epithelial cells of unfed, adult *D. andersoni* that were incubated at 37 °C for 2.5 days(1). Light microscope studies using FA and electron microscope studies using peroxidase-antiperoxidase (PAP) technique confirmed the identity of *A. marginale* within the colonies(4,5).

Ultrastructural studies of *A. marginale* in ticks have revealed organisms in feeding adult ticks that are very similar morphologically to organisms comprising marginal bodies of infected bovine erythrocytes (1). However, organisms that developed in unfed adult ticks that were incubated were different from those observed in erythrocytes. The organisms developed in rather large colonies in tick gut cells and were, individually, often electron-dense and pleomorphic with respect to both size and shape. Their diameter was between 0.21 and 0.56 μm and the shape ranged from round or concave to elongate with protoplasmic extensions. A small, electron-dense particle was also described within colonies, suggesting a mode of reproduction in addition to binary fission. The small membrane-bound particles appeared to form by sub-particulation of the internal cellular components on the organism.

The life cycle of *A. marginale* has not been determined in either the vertebrate or invertebrate hosts. This prokaryotic organism is presently classified in the Order Rickettsiales, Family Anaplasmataceae(6,7). This
classification is based upon characteristics of *A. marginale* observed in bovine erythrocytes in which the organism is an obligate intracellular parasite. The morphology is similar to that of the members of the Rickettsiales, and the organism appears to divide by binary fission.

Recent studies on the influence of elevated temperature on *A. marginale* in incubated *Dermacentor andersoni* have shown that experimental calves experienced the shortest prepatent periods when inoculated with gut homogenates made from ticks that had been incubated for 2.5 days at 37°C(8). Histologic sections of gut from ticks used in the incubation experiments contained colonies of several morphologic types. The purpose of the present study was to classify these colony types with the light microscope, to study the organisms within each type by electron microscopy, and to determine the colony density, size, type and distribution of the parasite in adult *D. andersoni* that had been incubated at 37°C for varying periods. The data are compared with the calf transmission study reported previously(8).

**MATERIALS AND METHODS**

**Animals**—Donor calves were infected with *A. marginale* and monitored for infection as described previously(8). Calf No. 184 had a peak parasitemia of 34.5%; Calf No. 204 had a peak parasitemia of 31.5%; and Calf No. 195 had a peak parasitemia of 50.6% during tick feeding.

**Laboratory Propagation and Infection of Ticks**—*Dermacentor andersoni* was reared and maintained at the Oklahoma State University Department of Entomology, Tick Laboratory(9). Larvae were fed on rabbits and were allowed to develop to the nymphal stage. Nymphal ticks were infected as described previously(8). Adult ticks were maintained in the laboratory at room temperature for 1 month and then used for histologic studies. Trial 1 was done in Spring 1980, Trial 2 was done in late Summer 1980, and Trial 3 was done in Fall of the same year.

**Collection of Tissues and Light and Electron Microscopy**—Gut tissues were collected from ticks in 3 trials of incubation experiments. For each trial, all ticks were infected (fed) on 1 calf. Groups of ticks were incubated at 37°C for 0, 1.5, 2.5 and 7.0 days. In each trial, 15 female and 15 male ticks were dissected for each of the 4 incubation times, the gut removed and processed individually for electron microscopy as described previously(1). Thick sections (1 μm) were stained with Mallory's stain(10) for 2 minutes at 60°C, and were examined by light microscopy. The area of the gut cross section was measured with a calibrated ocular grid and the number and type of colonies within the cross section was recorded. The number, diameter, and type of colonies per unit area of gut was determined, and the data were analyzed statistically. The results of the morphology studies were compared with the calf transmission study reported previously(8). Ultrathin (silver-reflective) sections were cut with
an ultramicrotome\textsuperscript{a} and a diamond knife.\textsuperscript{a} The sections were collected on 300-mesh copper grids, stained with uranyl acetate and lead citrate\textsuperscript{11} and observed and photographed in an electron microscope\textsuperscript{b} (operated at 60 kV).

Characterization of Colonies of \textit{A. marginale}: Typing and Electron Microscopy—Thick sections of gut tissue were examined by light microscopy for the presence of colonies. Each colony was assigned a type number (1-5) based upon its morphologic characteristics. Colonies representing each type were located with the light microscope, fine-sectioned and studied with the electron microscope to characterize further each colony type and organisms within.

RESULTS

\textit{Light Microscopy Studies}—Colonies of \textit{A. marginale} in midgut epithelial cells of adult \textit{D. andersoni} that were infected as nymphs were observed to conform to one of 5 distinct types (Fig. 1 a-f). These colonies were most frequently in the basal portion of the epithelial cells near the basement membrane; occasionally they were seen on the coelomic side of the basement membrane or near the luminal surface of the gut cells. No colonies were observed in sections of gut tissues from control ticks.

The morphologic characteristics of each colony type are as follows:

\textbf{Type 1}—These colonies were round, very compact and stained intensely with Mallory's stain. Although individual darkly-stained organisms within the colony could be seen with the light microscope, they did not appear to be separate from one another. There was little or no empty space in the Type 1 colonies (Fig. 1a).

\textbf{Type 2}—Type 2 colonies were similar to but slightly less dense in staining characteristics than Type 1 colonies. The major difference was that the mass of organisms appeared to have separated from the distinct limiting membrane of the colony, forming an open area or "halo" (Fig. 1b).

\textbf{Type 3}—Type 3 colonies contained masses of organisms similar in staining characteristics to those in Type 1 and 2 colonies. The organisms were aggregated into clumps, however, leaving open areas between them. The Organisms within the clumps appeared to be closely associated, almost as if adhered to one another. It was difficult to discern individual, discrete organisms in Type 3 colonies (Fig. 1c).

\textbf{Type 4}—Type 4 colonies contained many separate organisms. The colonies often were large and somewhat irregular in shape (Fig. 1d).

\textsuperscript{a}Sorval MT-2 and Dupont Diamond Knife, Dupont Instruments, Wilmington Delaware.

\textsuperscript{b}Philips EM-200, Philips Electronic Instruments, Mount Vernon, New York.
Type 5—These colonies were also often irregularly shaped. They contained separate individual organisms that were often larger and more densely stained than the organisms in Type 4 colonies. The number of organisms in Type 5 colonies was fewer than in Type 4 and typically there was an appearance of considerable empty space (Fig. 1e). Some Type 5 colonies contained very large, amorphous particles along with the typical densely-staining organisms (Fig. 1f).

Electron Microscopy Studies—For morphologically distinguishable forms were recognized with colonies. The four morphologic forms were: electron-dense forms, reticulated forms, pleomorphic reticulated forms and small particles.

Type 1 Colonies—Small, round Type 1 colonies were most often near the basement membrane (Fig. 2a). The colonies contained electron-dense forms, often irregularly-shaped, ranging in size from 0.21 µm to 0.4 µm. The cell membranes were not as distinct as in the larger reticulated forms. The electron-dense forms were in close association with one another. The colonies were often near the host cell nucleus.

Type 2 Colonies—Type 2 colonies contained electron-dense forms similar to those described in Type 1 colonies (Fig. 2b). The organisms appeared to be separated slightly from one another, a characteristic which was not evident at the light microscope level. The mass of organisms was separated from the limiting membrane forming an open area or “halo”, a feature that was clearly visible by light microscopy. Type 2 colonies were also generally near the host cell nucleus and the basement membrane.

Type 3 Colonies—Type 3 colonies contained both electron-dense forms and larger, less densely staining reticulated forms (Fig. 2c and d). A limiting membrane was often evident but sometimes absent. When the limiting membrane was present, it was often irregular. The organisms were also somewhat separated from one another, a feature not detectable with the light microscope. Type 3 colonies also contained a very small particle which was outside of the parasite membrane. These particles were approximately 0.06 µm and were generally not found in Type 1 and 2 colonies (Fig. 2c and d).

Type 4 Colonies—Type 4 colonies contained many reticulated forms that were separate from one another and ranged in size from 0.37 µm to 0.61 µm. The reticulated forms within these colonies often had small particles (0.06 µm) within the cell membrane of the organism (Fig. 3b and d). Some Type 4 colonies contained membrane whorls around the reticulated forms, suggesting early development of large amorphous clusters of reticulated forms like those in Type 5 colonies (Fig. 3c and d). The limiting membrane of Type 4 colonies was often disrupted or appeared to be missing entirely.

Type 5 Colonies—Type 5 colonies contained fewer well defined reticulated forms than did Type 4 colonies (Fig. 4a) and sometimes
contained large pleomorphic reticulated forms which often formed large clusters (Fig. 4b-d). The pleomorphic reticulated forms were very irregular in size and shape and often appeared to be connected to one another. Some pleomorphic reticulated forms were surrounded by membrane whorls (Fig. 4b and d).

A. marginale Parasitemia of Donor Calves During Tick Feeding — The A. marginale parasitemia of donor calves during feeding of nymphal ticks are listed in Table 1.

Transmission studies that were done with the same groups of incubated ticks have been reported previously(8). The prepatent periods of calves inoculated with gut homogenates of ticks that were either not incubated or incubated for 1.5, 2.5 or 7 days are superimposed over the graph depicting the density of the colonies to facilitate comparison (Figure 5).

**Number of Colonies per .001 mm² of Gut Tissue** — The mean numbers of colonies per .001 mm² gut tissue and the range in the 3 trials of incubated and unincubated ticks are listed in Table 2 and depicted graphically in Figure 5. The means for all trials of incubated ticks ranged from as few as 2.23 colonies up to 7.32 colonies per .001 mm² gut tissue. The range among individual ticks was quite wide; only 1 of 346 ticks examined, however, did not contain colonies. Both male and female ticks in each incubation group of all trials contained colonies. An analysis of variance was performed on these data and the results are listed in Table 3. The results for each trial are as follows:

**Trial 1** — The difference in density of colonies among incubation groups in Trial 1 was not statistically significant when averaged across both sexes. The significant interaction of incubation time x sex indicated that the effect of incubation period of colony density was not consistent across sexes. While colony density generally increased in males increasing incubation time, the density of colonies in sections of female gut peaked at 2.5 days of incubation (Figure 5, Table 2). The combined average colony density (combined for males & females) decreased slightly at 1.5 days of incubation and increased at 2.5 and again at 7 days of incubation.

**Trial 2** — No statistically significant differences were observed in the density of colonies present in gut tissues from ticks incubated for the 4 experimental periods (Table 3). The colony density was lower at 1.5 days and peaked at 2.5 days of incubation. Both male and female ticks in each incubation group contained colonies. Experimental error (variation among ticks) was appreciably higher in this trial than Trials 1 and 2 (Table 3).
### TABLE 1: A. marginale Parasitemias of Donor Calves During Tick Feeding.

<table>
<thead>
<tr>
<th>Trial No.</th>
<th>Calf No.</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>184</td>
<td>5.6</td>
<td>7.8</td>
<td>16.0</td>
<td>18.8</td>
<td>NA</td>
<td>34.5</td>
</tr>
<tr>
<td>2</td>
<td>204</td>
<td>8.2</td>
<td>17.4</td>
<td>18.8</td>
<td>31.5</td>
<td>31.5</td>
<td>32.0</td>
</tr>
<tr>
<td>3</td>
<td>195</td>
<td>10.8</td>
<td>23.3</td>
<td>33.0</td>
<td>50.6</td>
<td>43.2</td>
<td>30.3</td>
</tr>
</tbody>
</table>

### TABLE 2: Mean Number of Colonies per .001 mm² of Gut Tissue from Adult Male and Female D. andersoni that were Incubated or Not Incubated (Trials 1 to 3).

<table>
<thead>
<tr>
<th>Trial No.</th>
<th>Incubation (Calf No.)</th>
<th>Incubation (nymphal ticks fed on)</th>
<th>No. of adult ticks (days)</th>
<th>Sex of Ticks per group</th>
<th>Mean No. Colonies per .001 mm² of Gut Tissue</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>TRIAL 1</td>
<td>0.0</td>
<td>184</td>
<td></td>
<td>Male</td>
<td>2.88</td>
<td>1.17- 9.70</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>4.17</td>
<td>0.98- 9.91</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>3.55</td>
<td>0.98- 9.91</td>
</tr>
<tr>
<td></td>
<td>1.5</td>
<td></td>
<td></td>
<td>Male</td>
<td>2.73</td>
<td>0.40- 8.91</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>4.31</td>
<td>1.09- 8.68</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>3.49</td>
<td>0.40- 8.91</td>
</tr>
<tr>
<td></td>
<td>2.5</td>
<td></td>
<td></td>
<td>Male</td>
<td>3.67</td>
<td>0.73- 8.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>4.72</td>
<td>1.76-10.94</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>4.19</td>
<td>0.73-10.94</td>
</tr>
<tr>
<td></td>
<td>7.0</td>
<td></td>
<td></td>
<td>Male</td>
<td>5.49</td>
<td>1.08-14.18</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>3.19</td>
<td>0.36- 6.25</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>4.30</td>
<td>0.36-14.18</td>
</tr>
<tr>
<td>TRIAL 2</td>
<td>0.0</td>
<td>204</td>
<td></td>
<td>Male</td>
<td>4.19</td>
<td>0.31- 8.82</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>5.31</td>
<td>0.95-17.24</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>4.75</td>
<td>0.31-17.24</td>
</tr>
<tr>
<td></td>
<td>1.5</td>
<td></td>
<td></td>
<td>Male</td>
<td>6.07</td>
<td>0.00-12.81</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>3.35</td>
<td>0.63- 7.12</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>4.71</td>
<td>0.00-12.81</td>
</tr>
<tr>
<td></td>
<td>2.5</td>
<td></td>
<td></td>
<td>Male</td>
<td>6.19</td>
<td>1.07-15.42</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>8.45</td>
<td>0.13-37.50</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>7.32</td>
<td>0.13-37.50</td>
</tr>
<tr>
<td></td>
<td>7.0</td>
<td></td>
<td></td>
<td>Male</td>
<td>5.69</td>
<td>1.04-11.92</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>5.59</td>
<td>0.81-14.74</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>5.64</td>
<td>0.81-14.74</td>
</tr>
<tr>
<td>TRIAL 3</td>
<td>0.0</td>
<td></td>
<td></td>
<td>Male</td>
<td>2.12</td>
<td>0.30- 7.12</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>2.36</td>
<td>0.42- 7.80</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Combined</td>
<td>2.23</td>
<td>0.30- 7.80</td>
</tr>
</tbody>
</table>
FIGURE 5: Comparison of the Mean Number of Colonies per .001 mm² of Gut Tissue from Adult Male and Female D. andersoni that were Incubated or Not Incubated (Trials 1 to 3).

Trial 3—The interaction effect of incubation time × sex was marginally significant in Trial 3. Both sexes showed an increasing colony density up to 2.5 days but a decrease at 7 days (Figure 5).

Colony Type Distribution per Incubation Time—Frequency of occurrence of the 5 types of colonies is listed in Table 4 and graphically displayed in Figure 6. Colonies of each type were present in ticks from each of 4 treatments in all 3 trials. The distribution of all 5 types of colonies varied considerably among the incubation periods of the 3 trials and although no consistent pattern of distribution was apparent, some trends were discernible. Few type 1 colonies were observed in ticks that were not incubated in comparison with numbers of type 1 colonies present in those from the 7-day incubation group; this trend was particularly marked in the male ticks in Trials 1 and 3. Type 3 colonies were the predominant type in all ticks from the 4 groups in Trial 2. Furthermore, the type 3 colony was the most frequently observed type in ticks from the 2.5-day group in all 3 trials.

Colony Size —The diameters of colonies according to type are listed in Table 5 and graphically presented in Figure 7. The mean diameter increased with colony type ranging from 5.63 μm (in type 1) to 13.33 μm (in type 5). The diameter of all colonies was slightly greater in male ticks than in females in 12 out of 15 groups. The exceptions were type 5 colonies in trial 1 and types 4 and 5 colonies in trial 3.

### TABLE 3: Analysis of Variance of the Number of Colonies per .001 mm² Gut Tissue Within Each Trial.

<table>
<thead>
<tr>
<th>Source of Variation</th>
<th>TRIAL 1</th>
<th>TRIAL 2</th>
<th>TRIAL 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>df</td>
<td>mean square</td>
<td>P value</td>
</tr>
<tr>
<td>Between Incubation Time</td>
<td>3</td>
<td>5.14</td>
<td>.5079</td>
</tr>
<tr>
<td>Between Sexes</td>
<td>1</td>
<td>4.57</td>
<td>.4052</td>
</tr>
<tr>
<td>Incubation time X Sex interaction</td>
<td>3</td>
<td>23.72</td>
<td>.0154</td>
</tr>
<tr>
<td>Experimental Error (among ticks within incubation time X sex)</td>
<td>107</td>
<td>6.55</td>
<td>111</td>
</tr>
</tbody>
</table>
# Development of Anaplasma marginale 85

## Table 4: Distribution of Colony Types (1-5) from Adult Male and Female D. andersoni that were Incubated or Not Incubated (Trials 1 to 3).

<table>
<thead>
<tr>
<th>Trial No.</th>
<th>Incubation Time</th>
<th>Sex of Tick</th>
<th>Colony Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Calf No. nymphal ticks fed on)</td>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td><strong>TRIAL 1</strong> (184)</td>
<td>0.0</td>
<td>Female</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>69</td>
</tr>
<tr>
<td></td>
<td>1.5</td>
<td>Female</td>
<td>64</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>44</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>108</td>
</tr>
<tr>
<td></td>
<td>2.5</td>
<td>Female</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>7.0</td>
<td>Female</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>160</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>212</td>
</tr>
<tr>
<td><strong>TRIAL 2</strong> (204)</td>
<td>0.0</td>
<td>Female</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td>1.5</td>
<td>Female</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>61</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>101</td>
</tr>
<tr>
<td></td>
<td>2.5</td>
<td>Female</td>
<td>68</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>72</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>140</td>
</tr>
<tr>
<td></td>
<td>7.0</td>
<td>Female</td>
<td>106</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>110</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>216</td>
</tr>
<tr>
<td><strong>TRIAL 3</strong> (195)</td>
<td>0.0</td>
<td>Female</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>1.5</td>
<td>Female</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>2.5</td>
<td>Female</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>134</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>165</td>
</tr>
<tr>
<td></td>
<td>7.0</td>
<td>Female</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>162</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined</td>
<td>167</td>
</tr>
</tbody>
</table>
FIGURE 6: Comparison of Distribution of Colony Types (1-5) from Adult Male and Female D. andersoni that were Incubated or Not Incubated (Trials 1 to 3).
TABLE 5: Average Diameters of the Five Types of Colonies of *A. marginale* in Adult Male and Female *D. andersoni* (Trials 1 to 3).

<table>
<thead>
<tr>
<th>Trial Number</th>
<th>Colony Type</th>
<th>No colonies Measured</th>
<th>Range</th>
<th>Average</th>
<th>SEM</th>
<th>Female</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>418</td>
<td>2-16</td>
<td>5.63</td>
<td>0.08</td>
<td>5.33</td>
<td>5.85</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>485</td>
<td>4-22</td>
<td>7.45</td>
<td>0.11</td>
<td>7.38</td>
<td>7.51</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>598</td>
<td>3-25</td>
<td>8.60</td>
<td>0.11</td>
<td>8.67</td>
<td>8.85</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>379</td>
<td>3-22</td>
<td>9.82</td>
<td>0.18</td>
<td>9.13</td>
<td>10.33</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>345</td>
<td>4-44</td>
<td>10.48</td>
<td>0.25</td>
<td>10.56</td>
<td>10.41</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>488</td>
<td>3-15</td>
<td>6.14</td>
<td>0.08</td>
<td>6.02</td>
<td>6.25</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>335</td>
<td>4-16</td>
<td>7.41</td>
<td>0.12</td>
<td>7.26</td>
<td>7.55</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>1372</td>
<td>1-25</td>
<td>8.52</td>
<td>0.07</td>
<td>8.36</td>
<td>8.64</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>427</td>
<td>4-50</td>
<td>12.21</td>
<td>0.25</td>
<td>11.80</td>
<td>12.66</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>317</td>
<td>5-40</td>
<td>13.08</td>
<td>0.29</td>
<td>13.02</td>
<td>13.11</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>411</td>
<td>1-15</td>
<td>6.19</td>
<td>0.09</td>
<td>6.10</td>
<td>6.21</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>136</td>
<td>4-14</td>
<td>7.40</td>
<td>0.17</td>
<td>7.35</td>
<td>7.42</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>629</td>
<td>4-27</td>
<td>8.66</td>
<td>0.10</td>
<td>8.64</td>
<td>8.67</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>474</td>
<td>5-27</td>
<td>10.82</td>
<td>0.17</td>
<td>11.06</td>
<td>10.64</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>452</td>
<td>5-55</td>
<td>13.33</td>
<td>0.28</td>
<td>13.78</td>
<td>12.93</td>
</tr>
</tbody>
</table>

*a*  
Standard error of the mean.
FIGURE 7: Comparison of Average Diameters of the Five Types of Colonies of *A. marginale* in Adult Male and Female *D. andersoni* (Trials 1 to 3).

**AVERAGE COLONY DIAMETER (µm)**
REFERENCES


Legends

Fig. 1 a-f: Light photomicrographs of *A. marginale* colony Types 1-5 in cross-sections of gut epithelial cells of unfed adult ticks that were infected as nymphs. 1 μm plastic sections stained with Mallory's stain. × 2500.

Fig. 1a — Type 1 colony (C).
Fig. 1b — Type 2 colony (C).
Fig. 1c — Type 3 colony (C).
Fig. 1d — Type 4 colony (C).
Fig. 1e and f — Type 5 colonies (C).

Fig. 2 a-d: Electron micrographs of colony Types 1-3 in midgut cells of adult ticks that were infected as nymphs.

Fig. 2a — Type 1 colony (C) containing electron-dense forms (EF); located near the basement membrane (BM) and host cell nucleus (Nu). × 22,500.
Fig. 2b — Type 2 colony (C) located near the basement membrane (BM). The colony has a well-defined limiting membrane (LM) and is near the host cell nucleus (Nu). × 10,620.
Fig. 2c — Type 3 colony (C) next to the basement membrane (BM). A limiting membrane (LM) is evident in a portion of the colony. × 12,000. The enclosed area is magnified in Fig. 2d.
Fig. 2d — High magnification of the enclosed area of Type 3 colony from Fig. 2c. The colony contains electron-dense forms (EF), reticulated forms (RF) and small particles (P). × 37,500.

Fig. 3 a-d: Electron micrographs of colony Type 4 in midgut epithelial cells of adult ticks that were infected as nymphs.

Fig. 3a — Low magnification of Type 4 colony (C). × 4,700. The enclosed area is magnified in Fig. 3b.
Fig. 3b — Higher magnification of enclosed area of Fig. 3a. Reticulated forms (RF) are present and some contain small particles (P) within the limiting membranes of the reticulated forms. × 18,000.
Fig. 3c — Electron micrograph of Type 4 colony (C). × 6,480. The enclosed area is magnified in Fig. 3d.
Fig. 3d — Higher magnification of the enclosed area of Fig. 3c. Colony contains reticulated forms (RF), some of which appear to be subparticulating into small particles (P). Membrane whorls (MW) surround some pleomorphic reticulated forms. × 18,750.

Fig. 4 a-d: Electron micrographs of colony Type 5 in midgut epithelial cells of adult ticks that were infected as nymphs.

Fig. 4a — Type 5 colony (C) that contains several reticulated forms (RF). × 7,650.
Fig. 4b — Type 5 colony (C) containing pleomorphic reticulated forms
Membrane whorls (MW) surround some of the pleomorphic reticulated forms. $\times 8,850$.

Fig. 4c—Type 5 colony (C) with limiting membrane (LM). $\times 3,008$. Enclosed area is magnified in Fig. 4d.

Fig. 4d—Higher magnification of the enclosed area of Fig. 4c. Notice numerous pleomorphic reticulated forms (PRF); some reticulated forms are surrounded by membrane whorls (MW). $\times 15,840$. 

*DEVELOPMENT OF* ANAPLASMA MARGINALE *91*
DEVELOPMENT OF *ANAPLASMA MARGINALE*
REPORT OF THE COMMITTEE ON ANAPLASMOSIS

Chairman: K. L. Kuttler, Moscow, ID
Vice Chairman: A. A. Cuthbertson, Elko, NV

J. Lee Alley, AL; J. F. Badger, MO; D. M. Bedell, GA; G. M. Brown, IA; G. M. Buening, MO; D. L. Brinkmeyer, IA; J. F. Cavanaugh, OH; W. B. Fairchild, LA; F. W. Frank, ID; R. I. Hail, KY; R. F. Hall, GA; R. L. Hartin, OK; T. J. Holt, MD; J. A. Howarth, CA; J. D. Huber, NV; M. M. Jochim, CO; R. A. Magonigle, CT; Duane Miksch, Ky; Dave Nash, ID; F. C. Neal, FL; W. G. Nelson, ID; V. F. Nettles, GA; E. J. Richey, OK; D. S. Roberts, KS; R. C. Searl, IA; N. R. Swan

The Anaplasmosis Committee met in open session at 1:30 p.m., Wednesday, November 10, 1982, in the Radisson Plaza Hotel, Nashville, Tennessee, with 31 in attendance.

Dr. Frederick Alderink, USDA-APHIS, College Station, Texas, made a presentation on the economic impact of anaplasmosis in Texas. Dr. Kathy Kocan summarized her paper on Anaplasma bodies in ticks. These papers have been presented to the general session and will be published in the proceedings.

A paper was read from Dr. Richard C. Searl, Ft. Dodge Laboratories, Ft. Dodge, Iowa which discussed the use of Anaplaz vaccine and reviewed its history regarding efficacy and the association with neo-natal isoerythrolysis (N.I.). In most instances, it was found that antigens “A”, “F”, and “V” were associated with N.I. by producing hemolytic antibody. Newer antigen production methods have eliminated donor cattle of the “V” antigen type. Antigens “A” and “F” are reduced to the extent possible in each production serial so as to reduce their antigen mass and hence, the antibody response in the recipient. Current recommendations are that the vaccine be administered only to open cattle whenever possible. Under the new schedule, two primary injections are to be administered at 4-week intervals, followed by a single booster a year after calving.

Even though the vaccine does retain the potential of producing N.I. if properly administered, such occurrences are rare.

Dr. Jerry L. Zaugg of the U.S. Department of Agriculture-ARS, Caldwell, Idaho, presented a report dealing with the immunological significance of Anaplasma colostral antibodies ingested by the neonate. This report suggests that the colostral antibody ingested by calves born to carrier cows did not induce a level of resistance or protection and was not responsible for an immune response in comparison to calves born to clean cows.

Dr. Kenneth L. Kuttler, USDA-ARS, Pullman, Washington, reported on efforts to remove Anaplasma infection in cattle by treating at various stages of the acute infection using LA-200. Treatment of acutely infected animals with 20 ml/kg LA-200 given 2 times intramuscularly at a 7-day interval was unsuccessful in eliminating the infection. Previous studies
using this same dosage in carrier intact animals does result in eliminating the infection. The persistence of residual immunity following treatment which clears the infection is significant and is currently being evaluated as to duration and relative level of protection.

Dr. A. A. Cuthbertson, Veterinary Practitioner from Elko, Nevada, reported the use of LA-200 in the treatment of anaplasmosis. Dr. Cuthbertson indicates that in his experience, the use of LA-200 was successful in moderating the course of acute infection and in aborting cases of possible anaplasmosis in a herd-treatment situation. One injection of LA-200 was usually sufficient in acute infection.

A report from Dr. John P. Maas, University of Idaho, Caldwell, Idaho, was given by Dr. Zaugg, in which he reported on the prevalence of Anaplasma infection in a closed herd held in an endemic zone over a 3-year period. Over this period of time, little or no transmission was detected in a herd where approximately 30% of the cattle were initially reacting to the complement fixation test. During this period of time, very few ticks were observed and no precautions were taken to prevent transmission.

A report from Dr. D. T. Henry, University of California, Davis, California, was read by Dr. Kuttler, in which he reported on the use of an attenuated Anaplasma vaccine in California. The attenuated vaccine has been in use sporadically throughout tropical countries, principally South and Central America since 1968. In recent years, a license was obtained to produce this vaccine in California, where it had been used in local cattle not destined for movement out of the state. All 50, eleven (11) month old beef heifers, when inoculated with the vaccine, became sero positive, showing only moderate clinical response. Severe reactions to this vaccine are possible, particularly in older or lactating dairy cattle. It is, therefore, recommended that it be used in young animals.

Anaplasmosis continues to be a persistent and costly disease of cattle, notwithstanding significant advances in our understanding of its transmission, pathogenesis, diagnosis, treatment, and prevention. New technology has enabled the researcher, the practitioner, and the regulatory veterinarian to make significant progress in our understanding of this costly disease.

The development of a control procedure that would completely eliminate the losses from anaplasmosis is an elusive goal which has yet to be accomplished. The vaccines discussed in the meeting, plus the newer formulations of oxytetracycline suggest means and procedures which hopefully will allow us to reach the goal of control and even possible eradication in some select areas. The committee urges continued support for research and field investigations of anaplasmosis in developing more effective control measures.
THE ROLE OF FDA IN THE REGULATION OF VETERINARY BIOLOGICS

Good morning. I wish to thank you for inviting me here today. I am pleased to have the opportunity to discuss with you FDA's future role in the regulation of the so-called "intrastate" veterinary biologics, that is, veterinary biologics unlicensed by the USDA because they are not shipped in interstate commerce, thus escaping jurisdiction under the Virus-Serum-Toxin Act. As a result of a recent court decision, FDA's authority to regulate veterinary biologics has been reconfirmed. The FDA does have authority to regulate animal biologics, particularly those not under the jurisdiction of the U.S. Department of Agriculture. In the past, producers of these veterinary biologics have not only been unlicensed by the USDA, but the FDA has not routinely exercised its authority either. This situation poses an unfair advantage over producers licensed with the USDA, but more importantly fails to provide uniform assurance of the safety, quality and purity of these products which are so important to the veterinary profession and animal health.

In 1913, Congress enacted the Virus-Serum-Toxin Act, which vested in the Secretary of Agriculture the responsibility for licensing and inspecting facilities producing veterinary biologics offered in interstate commerce. The U.S. Livestock Sanitary Association was vitally active in that legislation. Under this law the USDA does not have jurisdiction over animal biologics which do not enter interstate commerce. Under the Federal Food, Drug and Cosmetic Act, FDA's authority extends to drug products including biologics, whether or not shipped in interstate commerce simply on the basis that a component used in the manufacture of the drug moved in interstate commerce. Many court decisions support FDA's jurisdiction over products whose components have moved in interstate commerce. Although FDA has not taken a preplanned active role in regulating unlicensed animal biologics under the provisions of the Federal Food, Drug and Cosmetic Act, we have in fact taken regulatory actions against unlicensed veterinary biologics.

In 1972, the regulatory responsibility for human biologics under the old Division of Biologics Standards was transferred to the FDA. From that point on, human biological products would be regulated not only under the provisions of the Public Health Service Act, but also under the Federal Food, Drug, and Cosmetic Act. This became possible because biologics fall within the definition of a "drug" under the FD&C Act which permitted the jurisdictional authority. With regard to veterinary biologics, the Agency has never developed and implemented a compliance program to cover routine inspection of unlicensed veterinary biologics producers. In fact, a Compliance Policy Guide advises our field offices not to inspect firms manufacturing only animal biologics unless specifically directed by the Bureau of Veterinary Medicine. Thus, regulatory action against veterinary biologics has been taken in the past by the FDA when assisting USDA in regulating products for which USDA did not have an
appropriate enforcement remedy. Under the Virus-Serum-Toxin Act, USDA lacks authority for seizure or injunction. Rather, the VSTA provides licensing and inspectional authority and a prosecutorial remedy. The Federal Food, Drug and Cosmetic Act provides for additional enforcement activity such as seizure of products which are adulterated or misbranded and injunction to prohibit violations from occurring. For this reason, FDA's historical activity involving the veterinary biological industry has been due largely to requests for assistance from USDA—requests that reflected the statutory inability of USDA to adequately regulate the biological industry, particularly the growing segment of the industry which was specifically structured to escape USDA registration.

In our opinion, the currently existing split of jurisdiction over animal biologics between USDA and FDA is impractical, inefficient and largely ineffective to this point. And certainly it does not engender confidence in the quality of the nation's veterinary biologic supply. We would encourage legislative action to consolidate the jurisdiction within one Agency, whether it be USDA or FDA. In fact, such an attempt has been made. Several years ago a bill introduced by Congressman Wampler, would not only have given USDA more enforcement authority, but would also give USDA the jurisdiction to regulate all veterinary biologics produced and marketed in the United States. Insufficient support and backing caused the bill to die, and the bill has not been reintroduced.

At the time that USDA was pushing for legislative action, the FDA was advised that establishments preparing veterinary biologics solely for “intrastate” commerce are not subject to the Virus-Serum-Toxin Act. The USDA encouraged FDA attention to this heretofore unregulated industry. Subsequently, we issued an assignment to our field offices requesting inspection of twelve (12) unlicensed veterinary biologic manufacturers for the purpose of obtaining information and assessing the character of this industry. We attempted to learn about the types of products produced, and to evaluate the conditions under which the products were produced. One of the first inspections attempted met with refusal and a temporary restraining order preventing inspection by the government. The plaintiff argued before the Court that federal regulation of veterinary biologics rests solely with the USDA. The basis for this argument was Section 902k) of the Federal Food, Drug and Cosmetic Act which states:

"Nothing contained in this Act shall be construed as in any way affecting, modifying, repealing or superceding ... the virus, serum, toxin, and analogous provisions, applicable to domestic animals, of the Act of Congress approved March 4, 1913 ..."

The plaintiff argued that Congress specifically excluded animal biologics from the FD&C Act under this provision, and further that since the VST Act does not provide jurisdictional authority over “intrastate” producers, no federal agency had authority. The government argued that the animal biologic in question was a drug within the meaning of the Food, Drug and Cosmetic Act, and jurisdiction is established since a
component of the biologic moved in interstate commerce. Additionally, since the VST Act did not provide jurisdiction to USDA over the "intrastate" animal biologics, the restricting clause in Section 902(k) did not apply.

The District Court for the District of South Dakota, Southern Division, initially held for the plaintiff. The government appealed the decision. In an *en banc* decision by the Eighth Circuit Appellate Court, it was ruled that indeed FDA does have jurisdiction over animal biologics. Animal biologics are drugs under the meaning of the drug definition in the Federal Food, Drug and Cosmetic Act since they are "intended for use in the... cure, mitigation, treatment, or prevention of disease in... animals." On April 19, 1982, the Supreme Court denied *Certiorari* on an appeal for review of the lower court decision, allowing the Appeals Court decision to stand as ruling in this case. This is a significant decision and quite clear cut.

Now that the Agency's inspectional and regulating authority over veterinary biologics has been clearly articulated in the Courts, the Agency must assess the scope of its planned program activity in the regulation of unlicensed veterinary biologics. This is underway at this very time. As you undoubtedly already know, under a recent Memorandum of Understanding between USDA and FDA, it was agreed among other things, that the two agencies would provide each other with information regarding veterinary biologic products under the jurisdiction of the VST Act or FD&C Act so as to coordinate investigation and enforcement activities and avoid duplication of effort. Additionally we have agreed to provide information to each other on pending investigations and enforcement actions.

To date, the FDA has not adopted a compliance program to cover the regulation of unlicensed animal biologics. Let us step back for a moment to review the current program on the regulation of human biologic products, to lend perspective to how the FDA approaches its regulatory responsibilities. These products, as I stated earlier, are subject to the provisions of both the Public Health Service Act and the Federal Food, Drug and Cosmetic Act. The Public Health Service Act is similar to the Virus-Serum-Toxin Act in that both authorize inspection and licensing of biologics producers. Individual lots or serials of licensed human and veterinary biologic products are tested prior to release in accordance with the appropriate regulations. Human biologics must also be in compliance with the FD&C Act. The firms must be registered with FDA under Section 510 as drug manufacturers. Finally, human biologics producers must comply with the requirements of the current Good Manufacturing Practices regulations for finished pharmaceuticals.

Unlicensed veterinary biologic products are subject to all of the drug provisions of the Federal Food, Drug and Cosmetic Act. USDA licensed veterinary biologic products historically have been exempted from Section 512, the new animal drug provisions of our Act, and from Section
510, drug registration with the FDA. Additionally, animal biologics produced in accordance with the USDA license and outline of production are considered to be in compliance with current good manufacturing practice requirements. With unlicensed veterinary animal biologic products, these exemptions would not apply. These products are subject to all appropriate provisions of the Federal Food, Drug and Cosmetic Act.

With regard to the new animal drug issue, the Agency to date has not reviewed individual veterinary biologic products to determine whether they are or are not generally recognized as safe and effective by qualified experts. This issue is not our highest priority for assessment at this time. Nevertheless, should it be concluded at any time that a veterinary biologic product is a new animal drug, continued marketing without approval would cause the product to be adulterated under the FD&C Act and subject to enforcement activity such as seizure or injunction.

Historically, it has been the agency’s position that the law requires adequate assurance that production quality control in the manufacture of a drug product provides appropriate assurance of safety, identity, strength, quality and purity for the protection of the animal and public health. As a result, the Agency established through rulemaking minimal standards for the manufacture of drug products. These current Good Manufacturing Practice regulations, called GMP’s, are required to be followed by all human and veterinary drug manufacturers, and of course biologic manufacturers as well. Under the Federal Food, Drug and Cosmetic Act, a drug is deemed to be adulterated unless the methods facilities and controls used in the manufacture, processing, packing and holding and the facilities and controls used therefor conform to current good manufacturing practice. As stated earlier, licensed human biologic producers are required not only to adhere to the biological products standards, but also the drug GMP’s. In my opinion, these standards of quality control are an appropriate basis for providing a degree of assurance for the consistency of product quality. We must remember that under the Food, Drug and Cosmetic Act, the Agency is not specifically authorized to control the release of individual lots based upon their satisfaction of tests performed either by the firm or by the Agency. Since FDA is not in the position of requiring lot by lot testing and Agency approval prior to release of veterinary biologies, GMP requirements offer the only assurances of consistency among production batches. As I stated earlier, the Agency has not yet adopted a program for the comprehensive regulation of unlicensed veterinary biologics. Although we clearly have jurisdiction over the regulation of veterinary biologics, I am not prepared to address a planned enforcement strategy. We are assessing our strategy with deliberate and thoughtful caution. Any planned program must be generally consistent with product standards for similar products regulated by USDA, although the FD&C Act is in some respects more demanding than the VST Act. Our goal is to provide reasonable assurance to the public that all veterinary biologics meet appropriate quality standards and are safe and effective for their intended purposes.
And of course the FDA has competing priorities. So a carefully struc-
tured program must be devised to maximize efficiency and effectiveness.
This should not be taken to mean that until such time as a definitive
program is developed we will not implement regulatory actions when
situations occur which pose threats to human or animal health. We will
not hesitate to carry out our mandate under the law. I anticipate that the
Agency will issue a formal statement in the near future notifying all
unlicensed biologic manufacturers of the Agency's regulatory program
and outlining the specific requirements necessary for compliance with
the Federal Food, Drug and Cosmetic Act. This, of course, implies that an
alternative exists for this industry. These facilities may become licensed
by the USDA and comply with their regulatory requirements. I suggest
that this industry has an important stake in establishing its integrity in
today's highly visible and vital American agribusiness industry. FDA's
role is to assure that this occurs.

Presented on November 12, 1982, at the U.S. Animal Health Association Annual Meeting,
Nashville, Tennessee, by Lester M. Crawford, D.V.M., Ph.D., Director, Bureau of Veterinary
Medicine, Food and Drug Administration.

1'Grand Laboratories v. Harris, Court of Appeals Eight Circuit, 1981.
PROGRESS IN MULTIVALENT MODIFIED LIVE VIRUS BLUETONGUE VACCINES: COMMENTS ON SAFETY AND EFFICACY.

S. McConnell and C.W. Livingston Jr.

INTRODUCTION

In response to a request by Texas sheep producers, the Texas Agricultural Experiment Station was asked to assist in obtaining or producing a vaccine to prevent bluetongue disease (BT) in Texas sheep. The task was assigned to the authors and in September 1976 a program was initiated to produce an acceptable product. The presence of multiple serotypes of bluetongue virus in Texas and in the United States suggested the need for a multivalent vaccine.

CONSIDERATIONS

Because of the wide host range and multiple modes of transmission of bluetongue virus (BTV), a number of questions were entertained prior to initiating the program. These included (but were not limited to):

1. Should the vaccine be developed for use in sheep only or for all susceptible ruminant species?
2. Should the product be designed for in state use only or for commercial production—thus interstate use?
3. Should the product be a modified live virus vaccine or a killed product?

Live and killed vaccines have different characteristics, advantages and disadvantages. In our opinion, a safe, immunogenic attenuated polyvalent vaccine would possess a number of advantages. The vaccine would be simpler to produce in large volumes, easier to administer and should mimic more closely the natural infection. The principle problems would be a concern by some about reversion to virulence and the development of new serotypes via reassortment of genome segments.

A killed vaccine would avoid the problem of reversion to virulence and perhaps the potential problem associated with reassortment. However, a killed product would be more expensive to produce, require multiple injections (thus less acceptable to livestock producers) and would stimulate the immune system only marginally.

We answered these questions based on available data and concepts as follows:

1. BTV infects multiple species of ruminants, and is economically important in cattle as well as sheep. Our decision was to produce a product for multiple species use.

---

*Department of Veterinary Microbiology and Parasitology, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843 and TAES, San Angelo, Texas.*
2. A good product for multiple species use should also meet the requirements for federal licensing and interstate use. Therefore we based our development program on the requirements for vaccine products as specified in: Code of Federal Regulations, Title 9—Animal and Animal Products.

3. We choose to produce an attenuated product—BTV Modified live virus vaccine. This decision was based partially on personal preference and experience and on the basis that the vast majority of effective viral vaccines used either in veterinary medicine or in human medicine are of an attenuated nature.

Additionally, we choose to do the majority of our testing under monitored field test conditions. This type testing has a number of disadvantages, the prime consideration being an inability to control field exposure to natural disease. Budgetary constraints dictated on approach which would maximize our efforts, thus the field test program. Where funds permitted, critical control testing in confined sheep was conducted.

APPROACH

Standard vaccine production practices were followed. Seed viruses of each of the four accepted international serotypes i.e. 10, 11, 13 and 17b were obtained from Agricultural Research Services, USDA, Denver, Colorado. Each serotype was obtained as infected sheep blood in standard preservative.

The viruses were adapted to growth in cell culture, triple plaque purified and subjected to attenuation by laboratory manipulation. Safety testing, sterility testing, freedom from adventitious agents and virulence/reversion to virulence testing were conducted throughout the study. Periodically, efficacy of each individual serotype was evaluated by challenge with homologous strains of virulent virus. Appropriate unvaccinated controls were used throughout the program.

In the developmental phase of production, each individual serotype was tested independently. Subsequently, the vaccines were tested as dual vaccines in various combinations and finally as a quadrivalent vaccine. Vaccinated sheep and controls were challenged to assay for efficacy. In some instances, challenge of immunity was serendipitously occasioned by natural exposure to BTV. In each of the BT outbreaks, vaccinated sheep were unaffected whereas disease was observed in unvaccinated companions.

PRESENT STATUS

The vaccine(s) was tested using several different parameters:

bTAMU—It 17 original isolate sent to ARS-USDA Denver, Colorado, revived by sheep passage and returned to TAMU.
LIVE VIRUS BLUETONGUE VACCINES

A. Safety:

1. Each individual serotype was tested during attenuation by challenge of susceptible yearling ewes with 10cc of undiluted cell culture product (titer ca. 10^{6.9}) via the intravenous route of inoculation. These studies were later extended to goats and calves. The early data is presented in Table 1. In 1979, we vaccinated goats and cattle with the dual vaccines and over 1100 sheep with the 4-way product (Table 2). To date over 7000 ruminants have been vaccinated with the experimental vaccines with no untoward reactions observed.

2. The safety protocol for wildlife species is shown in Table 3. In this study 42 whitetail fawn and 200 Mouflon and Mouflon-crosses of varying ages, sexes and stages of pregnancy were vaccinated with the 4-way product. No untoward reactions were observed. The agar-gel precipitin status of Mouflon are presented in Table 4 and Table 5. Twelve whitetail fawn maintained under close supervision were vaccinated with the 4-way product. Two of the vaccinates were challenged with virulent virus. We were not permitted to include susceptible control fawn. No untoward reactions were observed following either vaccination or challenge.

3. Four groups of registered cattle, each herd exhibiting breeding difficulties and/or abortion problems were vaccinated with the 4-way vaccine during 1981-1982. A total of 416 cows in varying stages of gestation (range 10 days to 9 months) were included in this study. The data is summarized in Tables 6, 7 and 8. The four herds (Table 6) were located in three areas of the State i.e. Northeast, Central and South. Each herd was tested by the Texas Veterinary Medical Diagnostic Laboratory (TVMDL) for the standard abortion screen, the cows well cared for and under an exceptional herd health management scheme. On the day of vaccination, each cow was palpated for pregnancy, swabs taken for ureaplasma assay and serum collected for the AGP test. Repalpation and rebleed for serology was done on day 60 post vaccination. This information is shown in Tables 7 and 8. There was little or no change in pregnancy status in two herds, a third showed an increase of 15% in the number of cows pregnant. The cows in the latter herd (Alexander herd) have all calved and cows bred back. No abortions occurred and all calves were normal at birth. The fourth herd (Schindle) has had 5 abortions to date: two (twins) occurred following high level azium therapy; two a 4 month and an 8 month fetus were undiagnosed and the last is under study. Agar-gel precipitin testing for BTV shows no relationship between BTV and abortion.

4. Fetal Safety. Eight sheep fetuses of varying ages were injected in utero with undiluted 4-way vaccine. The fetal age and number injected is shown in Table 9. Pregnant ewes were anesthetized, the uterus exteriorized and the fetus located by manual manipulation.
Each fetus was injected (thru the uterine wall) with 2 ml of undiluted product, the uterus replaced, the incision closed with sutures and the ewes returned to the recovery pens. Each ewe was observed daily for evidence of disease or abortion. Six lambs were collected by cesarean section from the three ewes inoculated at 30 days of gestation. Two of the six died shortly after delivery. Four lambs were collected from the 60 day group and died shortly after delivery. Four lambs were born naturally (2 days before scheduled surgery) to the 90 day ewes. One died shortly after birth. This lamb had a hole in the diaphragm and was the only lamb found with an identifiable defect. Gross and microscopic observations on the other dead lambs showed no evidence of BTV infection. This study is presently being repeated.

B. EFFICACY AND DURATION OF IMMUNITY

1. During 1982, groups of BTV precipitin negative sheep, goats and cattle were inoculated with a standardized 2 ml dose of 4-way BTV vaccine. Each animal was monitored daily for evidence of disease. Blood was collected in OCG on day 8, 10 and 12 post vaccination and tested for presence of vaccine virus. On day 30 post vaccination, each animal was bled for serology and challenged with a whole blood containing 500-1000 sheep infectious doses of each of the four IT BTV's. Each animal was observed daily for evidence of infection or other responses to the virulent virus challenge. Blood samples were collected post challenge and assayed for virus content. Appropriate controls were included throughout the study. We observed clinical disease in the unvaccinated control sheep but not in the vaccinates. To date we have not been able to reisolate either vaccine virus or wild-type virus from the challenged vaccinates.

2. White-tail deer fawn study. Twelve white-tail fawn were vaccinated with the standard 2 ml dose of 4-way vaccine. Two of the 12 were challenged with a pool of all four wild-type viruses. No clinical disease was observed. The fawn continued to eat, gain weight and maintain normal activity. Both challenged fawn were fully protected by the vaccine however we were not permitted to include unvaccinated control fawn in this study. Vaccine virus (IT BTV's) was reisolated from 3/12 fawn on day 9 post vaccination. Virulent virus was isolated from the two challenged fawn—one IT BTV 10, the other ITBTV 17.

3. Two groups of sheep vaccinated previously, were tested for duration of immunity. The data is presented in Table 10. An acceptable level of protection to virulent virus challenge was found in sheep vaccinated 1 year previously but only marginal protection was observed in the ewes vaccinated 2 years previously.

In summary, (Table 11 and Table 12) the studies conducted to date suggest the 4-way modified live virus BTV vaccine has a good potential,
and is safe and effective in a broad range of ruminant species. All the data has not yet been generated from the studies reported.

Table 1

<table>
<thead>
<tr>
<th>Test for Attenuation and Safety</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Monovalent Vaccines</strong></td>
</tr>
<tr>
<td>Sheep</td>
</tr>
<tr>
<td>Spanish goats</td>
</tr>
<tr>
<td>Calves</td>
</tr>
</tbody>
</table>

| **Dual Vaccines** |
| Sheep | 6 |

Table 2

<table>
<thead>
<tr>
<th>Experimental Vaccine Field Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Animal Species</strong></td>
</tr>
<tr>
<td>Sheep</td>
</tr>
<tr>
<td>698</td>
</tr>
<tr>
<td>Goats</td>
</tr>
<tr>
<td>Cattle</td>
</tr>
</tbody>
</table>

TOTAL (3402)
Table 3

Wildlife Species

DEER
1. 12 white-tail fawn at TAMU
2. 31 white-tail fawn at Y-O Ranch

SHEEP
1. 200 Mouflon and Mouflon crosses

Table 4

Pre-vaccination Agar Gel Precipitin Results of Mouflon and Mouflon-cross Sheep

<table>
<thead>
<tr>
<th></th>
<th>BTV</th>
<th>EHDV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>2/6 (33.3%)a</td>
<td>0/6 (0%)</td>
</tr>
<tr>
<td>Female</td>
<td>50/109 (45%)</td>
<td>21/109 (19.3%)</td>
</tr>
<tr>
<td>Male</td>
<td>4/40 (10%)</td>
<td>2/40 (5%)</td>
</tr>
<tr>
<td>Female</td>
<td>12/48 (25%)</td>
<td>4/48 (8.3%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>68/203 (33.5%)</td>
<td>27/203 (13.3%)</td>
</tr>
</tbody>
</table>

aNNumber positive/number tested (percent positive).
Table 5

Post-vaccination Agar Gel Precipitin Results of 35 Mouflon and Mouflon-cross Sheep

<table>
<thead>
<tr>
<th></th>
<th>BTV</th>
<th>EHDV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>0/0 (0%)</td>
<td>0/0 (0%)</td>
</tr>
<tr>
<td>Adult</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>13/15 (86.7%)</td>
<td>5/15 (33.3%)</td>
</tr>
<tr>
<td>Male</td>
<td>1/1 (100%)</td>
<td>1/1 (100%)</td>
</tr>
<tr>
<td>Juvenile</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>7/19 (36.8%)</td>
<td>4/19 (21.05%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>21/35 (60%)</td>
<td>10/35 (28.6%)</td>
</tr>
</tbody>
</table>

*aNumber positive/number tested (percent).*

Table 6

Modified Live BTV Cattle Vaccination

<table>
<thead>
<tr>
<th>Herd</th>
<th>Breed</th>
<th>Number of Cows*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alexander</td>
<td>Simmental</td>
<td>180</td>
</tr>
<tr>
<td>Bennett</td>
<td>Simmental</td>
<td>59</td>
</tr>
<tr>
<td>Salyer</td>
<td>Angus</td>
<td>54</td>
</tr>
<tr>
<td>Schindel</td>
<td>Jersey</td>
<td>123</td>
</tr>
</tbody>
</table>

*All bred --- All registered.*
Table 7

Modified Live BTV Herd History

<table>
<thead>
<tr>
<th>Herd</th>
<th>Percent Pregnant* Day 0</th>
<th>Percent Pregnant* Day 60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alexander</td>
<td>57</td>
<td>72</td>
</tr>
<tr>
<td>Bennett</td>
<td>39</td>
<td>39</td>
</tr>
<tr>
<td>Salyer</td>
<td>36</td>
<td>36</td>
</tr>
<tr>
<td>Schindile</td>
<td>93</td>
<td>92</td>
</tr>
</tbody>
</table>

*All 3 trimesters represented

Table 8

Modified Live BTV Cattle Study

<table>
<thead>
<tr>
<th>Herd</th>
<th>No. Cattle</th>
<th>% AGP Positive Day 0</th>
<th>% AGP Positive Day 60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alexander</td>
<td>184</td>
<td>46</td>
<td>44</td>
</tr>
<tr>
<td>Bennett</td>
<td>60</td>
<td>40</td>
<td>65</td>
</tr>
<tr>
<td>Salyer</td>
<td>60</td>
<td>33</td>
<td>ND</td>
</tr>
<tr>
<td>Schindile</td>
<td>211</td>
<td>12.7</td>
<td>ND</td>
</tr>
</tbody>
</table>
Table 9

Fetal Safety

<table>
<thead>
<tr>
<th>SHEEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 3 - 30 day fetuses</td>
</tr>
<tr>
<td>2. 3 - 60 day fetuses</td>
</tr>
<tr>
<td>3. 2 - 90 day fetuses</td>
</tr>
</tbody>
</table>

Table 10

Duration of Immunity

<table>
<thead>
<tr>
<th>Number of Sheep</th>
<th>Vaccinated</th>
<th>Fever</th>
<th>Challenge Response</th>
<th>Weight Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>Yes (1 yr.)</td>
<td>2/8</td>
<td>2/8</td>
<td>0/8</td>
</tr>
<tr>
<td>2</td>
<td>No</td>
<td>2/2</td>
<td>2/2</td>
<td>2/2</td>
</tr>
<tr>
<td>10</td>
<td>Yes (2 yr.)</td>
<td>8/10</td>
<td>0/10</td>
<td>ND</td>
</tr>
<tr>
<td>2</td>
<td>No</td>
<td>2/2</td>
<td>2/2</td>
<td>ND</td>
</tr>
</tbody>
</table>
Table 11

Vaccine Efficacy Test

A. Humoral Immunity

* Serum Neutralization
* Agar Gel Precipitation

B. Challenge of Immunity

* Homologous Virulent Virus
  1) Temperature
  2) Reisolation

* Immunology

Table 12

BTV - Modified Live Virus Vaccines

1. All 4 serotypes plaque-purified
2. All 4 serotypes attenuated
3. Safety test satisfactory
4. Protection from disease satisfactory

REFERENCES

1. Livingston, C.W. and Hardy, W.T.: Isolation of an Antigenic Variant of Bluetongue Virus. AJVR 25 (1964), 1598-1600.
ACKNOWLEDGEMENTS

The authors wish to thank Dr. Leo B. Merrill, TAES, Sonora, Texas; Dr. Carl S. Menzies, Dr. Maurice J. Shelton, Dr. M.C. Calhoun and J.E. Huston, TAES, San Angelo, Texas; Dr. Percy R. Turner, Water Valley, Texas; and Mr. Tom Carter, Mertzon, Texas for providing us with support and livestock for these studies. We are indebted to Mrs. Georgia Cummings and Mrs. Betty Gauer for their technical assistance.
DEVELOPMENT OF CHEMICALLY SYNTHESIZED ANTIGENS
FOR VACCINES

James L. Bittle, D.V.M.
LaJolla, California

INTRODUCTION

Beginning with Jenner's basic discovery in 1797 that Cowpox Virus would immunize man against smallpox infection, there has been a constant determined effort by man to find a way to protect himself and the animal population against the major infectious diseases that threaten their well-being.

The early attempts at immunization were primarily concerned with producing protection with safety being less of a concern. As diseases such as Smallpox, Rabies, Diptheria, Tetanus and Avian Cholera were gradually controlled by the use of vaccines, the safety of various products became more important. Thus, the proposition that the benefit gained from using a vaccine should outweigh the risk from adverse reactions that may occur became accepted. So, as disease is controlled the emphasis has shifted to developing safer immunogens and yet to maintain their high degree of efficacy. This has been brought about by a continued improvement in methods of antigen production. The various methods used to produce vaccines include living substrates such as tissue suspension, the embryonated egg, cell culture for viral vaccines and artificial substrates for bacterial vaccines. The object is to cultivate the whole organism in as high a concentration, yet in as pure a state as possible.

PRESENT TECHNOLOGY

Most of the vaccines produced today are produced under conditions that minimize contamination with substrates. One of the most sophisticated methods is tissue cultures which is used on a mass scale to produce the common live attenuated viral vaccines. The difficulty in removing the unwanted components has made it necessary to depend on living attenuated organisms that multiply in the host. These can be produced by keeping the antigenic mass at a minimum and yet produce the desired effect in terms of immune response. The problem, of course, with living products is their potential danger in causing disease either by the immunizing antigen or by the contaminating substrates. Thus, vaccines containing living attenuated agents such as Rabies, Bovine Viral Diarrhea and Infectious Bovine Rhinotracheitis have been shown to produce undesirable side effects including the actual disease.

In contrast to these types of vaccines, inactivated products offer the advantage of improved safety, but are often weak in terms of stimulating an immune response, and therefore require multiple inoculations at regular intervals. This may result in undesirable side effects due to an immune response to unnecessary antigens in the vaccines. Attempts to
CHEMICALLY SYNTHESIZED ANTIGENS

concentrate the antigenic determinants of microorganisms by separating various fractions either by chemical or physical means have been tried. These so-called subunit vaccines have met with only limited success because of the expense involved in the methodology to attain greater efficacy over the products made from whole organisms.

The point is, however, that although an organism may be produced in relatively pure form, the antigenic determinants comprise a small part of the total antigenic structure. Thus, a high percentage of the antigen is not necessary for immunization and undoubtedly contributes to the undesirable side effects often seen.

It should be pointed out that the recombinant technology has the same objective; that is to produce a more purified antigen, but as the substrate used to cultivate the antigen is usually a bacterial or other microorganisms, the resulting product still contains the contaminants of that biological system.

SYNTHETIC ANTIGENS

The aim of our studies was, therefore, to produce the antigenic determinants in the highest purity possible, so that the antibody response is directed in the most specific way, and not to undesirable antigens.

Lerner, et al., at the Research Institute of Scripps Clinic showed that a synthetic antigen derived from a gene that codes for an envelope antigen would produce antibody that recognized the native protein. With this technology, we have made peptides by chemical solid state synthesis to a major portion of Foot and Mouth Disease (FMD) viral protein (VP1) in cooperation with Dr. Fred Brown of The Animal Virus Research Institute at Pirbright, England, and when inoculated into guinea pigs produced neutralizing antibody that protected on challenge. We have also vaccinated animals and maintained these for six months with antibody levels that would be considered protective. Synthetic peptides have also been made for the HA1 and HA2 of Influenza A and for the surface antigen of Hepatitis B. Those synthetic peptides will induce antibody for both of these agents that recognizes the native structure.

The method of producing the peptides is by solid state Merrifield synthesis. In this scheme, the amino acids are attached sequentially to an insoluble resin through its acid end with its amino end protected with t-butyloxycarbonyl (BOC). The BOC is removed by adding acid forming an amine salt. A base is added to neutralize the salt to free amine. A second amino acid is added, etc. The peptide is cleaved from the resin by hydrogen fluoride. Thus, peptides of various lengths may be synthesized in relatively pure form. These peptides by themselves may be antigenic, but their antigenicity is increased greatly by coupling them to a carrier protein and by adding an adjuvant to delay their absorption.

The host will recognize the specific peptide sequence as being foreign
in the same manner that it recognizes the same peptide structure (or the antigenic determinant) on the native antigen. Thus, antibody is made to the specific sequence, and if the peptide sequence is correct, the antibody may have strong neutralizing activity. The antibody response is not necessarily dependent on the antigen's conformation but probably more dependent on the amino acid sequence and the exposure of this sequence to immune recognition.

The advantages of chemically synthesized peptide antigens are obvious and these are shown below:

1) High degree of purity will produce a more specific antibody.

2) Products will be safer because they are purer and do not contain unwanted antigens.

3) The increased concentration of antigenic determinants will increase potency.

4) Chemically synthesized peptides in general are quite stable and should have a much better shelf life than antigens made by present means.

5) Peptide chemical synthesis is much more controllable and predictable than the biological systems used to make antigens today.

6) The cost of antigens made by this method should be considerably reduced over biological production when all factors are considered.

Thus, using chemically synthesized peptides, highly specific, safe and stable vaccines will be developed in the future that will add greatly to our ability to control diseases in both man and animals.
REPORT OF THE 1982 COMMITTEE ON BIOLOGICS

Chairman: R.F. Kahrs, Gainesville, FL
Vice Chairman: R.W. Loan, College Station, TX

D.C. Alexander, Ont., Canada; M.H. Bairey, IA; D.E. Baldwin, NB; W.H. Beckenhauer, NB; D.E. Bordt, IL; A.C. Braemer, CA; E.L. Drake, NV; John Finnell, IL; D.A. Fuller, IA; J.S. Gloyd, IL; Richard Hall, GA; B.B. Hancock, IA; L.E. Hanson, IL; W.P. Heuschele, CA; R.E. Horton, NJ; Majon Huff, CO; D.W. Johnson, MN; G.L. Johnson, KS; D.E. Kahn, NJ; L.L. Lauerman, Jr., AL; Vincent Marshall, NB; Duane Pankratz, SD; G.V. Peacock, FL; R.J. Price, MD; D.C. Randall, CO; R.C. Stewart, IA; K.E. Thayer, OR; Hsi-tang Tung, IL; R.R. Turner, TX; J.D. Todd, KS; C.D. Van Houweling, VA; G.B.E. West, CA; Robert Williams, IN

On invitation of the Committee, Dr. Lester Crawford, Director, Bureau of Veterinary Medicine of the FDA addressed the role of the FDA in regulation of veterinary biologics stating FDA authority encompassed intrastate producers and that the FDA will work in partnership with USDA, that the two agencies are preparing a revision of the Virus Serum Toxin Act of 1913 and that he would keep the Committee apprised of progress. A full text of his remarks presented at the General Session will appear in the Proceedings.

The following invited papers were presented on the effects of emerging technologies on veterinary biologics.

1. Monoclonal Antibodies — A New Tool for the Biologics Industry Dr. Richard Van Deusen, Ames, Iowa

2. Development of Chemically Synthesized Antigens for Vaccines Dr. James L. Bittle, La Jolla, California

3. Restriction Enzyme Analysis (Virus Finger Printing) for Distinguishing Vaccine Viruses from Field Strains. Dr. David Reed, Ames, Iowa.


The speakers indicated technology is now available and in use for developing highly purified diagnostic reagents, for developing a series of safe and effective vaccines by chemical synthesis of antigens, and for distinguishing vaccine viruses from field viruses. Dr. Al Strating, director of Veterinary Services Laboratories of the USDA, indicated the Veterinary Biologics group is prepared to understand and regulate products developed using these technologies and that equipment and staff is being continually updated to meet this challenge. He announced Dr. David Esperseth was assuming leadership of the Veterinary Biologies Staff of USDA. The Committee instructed the chairman to congratulate him on his appointment and invite him to participate in Committee activities.
The following papers were presented on experimental bluetongue vaccine.

1. Progress in Multivalent Modified Live Virus Bluetongue Vaccines: Comments on Safety and Efficacy. Dr. Stewart McConnell, College Station, Texas.

2. Progress in Inactivated Vaccines for Bluetongue: Comments on Safety and Efficacy. Dr. Bennie Osburn, Davis, California.

These speakers indicated preliminary data suggest progress toward safe effective bluetongue vaccines.

At the request of the Chairman, Dr. David Esperseth summarized the Memorandum of Understanding on Regulation of Veterinary Biologics published in the Federal Register as follows:

The USDA regulates products sold in interstate commerce and the FDA regulates products sold in intrastate commerce. A six man committee representing both agencies will cooperatively adjudicate questions of jurisdiction. People with questions may contact officials of either agency.

Dr. Miles Bairey, of Veterinary Services Laboratory reported changes published in the Federal Register regarding autogenous bacterins as:

1. Removal of the limits on serial size
2. Restriction of use of autogenous bacterins to premises from which the organism originated has been expanded to include "all adjacent premises at risk"
3. The time limit on preparation of bacterins from an isolate has been extended from 30 days to one year.
4. Six month expiration date on autogenous bacterins have been extended to 18 months.

The Committee agreed to keep posted on any forthcoming biologics legislation for next years meeting.
A REVIEW OF METHODS TO CONTROL BOVINE LEUKOSIS

J. M. Miller and M. J. Van Der Maaten
U.S.D.A., Agricultural Research
National Animal Disease Center
P.O. Box 70, Ames, Iowa 50010

Early in this century it was recognized that lymphoid tumors of cattle were not randomly distributed in the bovine population.1,2 Cases were frequently concentrated in a few herds, and the disease was observed to spread from herd to herd and from one geographic region to another. In the 1950's, Bendixen3 conducted epidemiologic studies which showed that there were different clinicopathologic forms of bovine leukosis, but only one of them had the characteristics of an infectious disease. This form, also known as adult type lymphosarcoma, was designated Enzootic Bovine Leukosis (EBL), whereas all other forms were grouped together and called Sporadic Bovine Leukosis. Bendixen also confirmed an earlier finding of German scientists that in herds with EBL there were frequently many apparently healthy cattle with abnormal hemograms, specifically a persistent elevation of blood lymphocytes. This condition of persistent lymphocytosis (PL) was thought to be a subclinical form of EBL and hematologic examinations were routinely used in epidemiologic and experimental studies of the disease. Standards were developed for the determination of PL and these became known as hematologic "keys." The Bendixen key was the most widely known because it was used for the first EBL control effort, initiated in Denmark in 1959.

During the first phase of the Danish program, the detection of affected herds was accomplished by making clinical leukosis (tumor) a notifiable disease.4 Each herd so identified was examined with the hematologic test and if animals with PL were found, the herd was either permanently quarantined or slaughtered. Herd owners were compensated monetarily when they chose total depopulation. In 1969, the program was intensified and hematologic examinations were routinely applied to all herds in the country. Each year one-third of the cattle population was tested and the policy of herd quarantine or slaughter was continued when an EBL diagnosis was made. This protocol was followed throughout the 1970's. During those years, researchers were gradually accumulating evidence that a virus was the primary etiologic agent involved in the disease.5,6 Several serologic tests were developed for detection of cattle that were infected by the bovine leukosis virus (BLV) and it was shown that these tests were more sensitive and specific than the hematologic keys.

In 1979, Denmark adopted an agar gel immunodiffusion test for antibody to the BLV glycoprotein antigen (gpAGID) to replace the Bendixen key in its leukosis control program.7 The country-wide screening tests were discontinued except in certain areas that had been previously identified as having a high incidence of EBL. Other herds were selected for serologic examination if any of the following conditions applied: (1)
known contact with herds that had experienced clinical disease or that had BLV-infected cattle; (2) a reported case of lymphosarcoma; (3) previously regarded as a suspect herd when examined by hematology. In all instances, herd quarantine or slaughter was required when the BLV infection was found in 1 or more animals.

This year (1982) the Danish program was changed and now instead of herd slaughter, it is only necessary to remove the individual BLV reactors. The herd is then quarantined, however, until it has completed 5 consecutive yearly tests with completely negative results. A new procedure for serologic surveillance has also been instituted. Instead of herd screening, tests are conducted on serums that are collected from every sixth animal passing through slaughter plants. When reactors are found they are traced back to the herd of origin and the entire herd is then examined. A similar procedure is followed when tumor cases are reported.

Besides Denmark, the country with the longest history of bovine leukosis control is the Federal Republic of Germany, where several states established policies in the early 1960's to deal with the problem. The programs were conducted on a voluntary basis but modest indemnity payments were provided to compensate for the animals slaughtered. In most cases this involved cows with PL and their offspring. Herd slaughter was conducted only if the hematologic test detected a very high proportion of affected animals. In the mid 1970's, when BLV serologic tests replaced hematology, the slaughter requirement was limited to individual reactors only.

The Danish and West German control programs have been successful in reducing the incidence of lymphoid tumors and the prevalence of BLV infection in cattle. In Denmark, the tumor rate in certain high incidence areas dropped from 29/100,000 cattle in 1960 to about 3/100,000 in 1975. There are currently only 30 known BLV-infected herds in the country and just 2 reactors were found in tests of the first 10,000 serum samples collected at slaughter plants. In the German state of Lower Saxony, where 1120 lymphosarcoma cases were reported in 1963, only 27 were diagnosed in 1981. Application of the serologic testing program (gpAGID test) in selected EBL herds of that area reduced the BLV prevalence from 14% to 0.3% in 2 years. Only 7% of the herds that were infected at the beginning of the program still had reactors after the 2 years of testing. It was observed, however, that reactors were found in some herds which had been completely seronegative in as many as 3 or 4 consecutive tests, conducted at 6-month intervals.

Several possible reasons have been suggested for the failure to eradicate BLV in some herds. For example, in a similar program in Belgium, investigators showed that the most common problems were a lack of cooperation or fraud by owners, and laboratory or clerical errors in the testing procedures. Other workers have suggested that the gpAGID test lacks sufficient sensitivity and that a radioimmunoprecipi-
METHODS TO CONTROL BOVINE LEUKOSIS

tation assay, an enzyme-linked immunoassay, or a serum neutralization test would lead to a faster and more complete removal of BLV-infected animals. Another factor which could cause this type of problem in a control program is an unusually long virus latency in some cattle. After experimental infection, cattle usually become seropositive to BLV within 3 months. Recently, however, Straub observed seroconversion in a few cattle 3 years after they had been taken from their BLV-infected dams and placed in individual isolation units.

Although the total eradication of BLV from a large geographic area may require a prolonged effort, the initial results of several trials support an opinion that the prevalence of infection can be rapidly reduced in large cattle populations, and that total eradication of the virus can be easily achieved in most herds. All of these programs are based on the serologic tests, primarily gpAGID, but different protocols are followed for regulating the investigation methods and the disposition of infected animals (or herds). The choice for a particular protocol option appears to be primarily determined on the basis of relative cost. Naturally the most aggressive programs are the most successful, but there seems to be general agreement that control of BLV infection, and thereby EBL, is possible with the technology currently available.

In some herds, the elimination of all BLV-infected cattle is not feasible because of monetary or genetic considerations. For these situations, the segregation of noninfected cattle from virus carriers may be a preferable method of control. Experience with this type of program is limited, but preliminary results indicate that rather minimal separation efforts can effectively prevent significant levels of virus transmission. Recently the segregation approach has been initiated in large cattle populations of state-owned herds in Yugoslavia. All cattle that are seropositive in the gpAGID test are moved onto isolated farms and maintained there until culled for slaughter. Progeny from infected herds are raised in isolation and after they have reached breeding age only the seronegative animals are allowed to return to the “clean” herds. Based on the criterion of 2 consecutive negative serologic tests, this program has resulted in a decrease in the percentage of infected farms from 21.4% in 1981 to 2.901 in 1982.

There have been very few attempts to develop a vaccine for the prevention of BLV infection. Thus far the only immunogen which appears to be effective is the surface glycoprotein of the virus. Unfortunately, this is the same antigen that is used in most of the serologic tests for identification of infected animals. Because these tests could not differentiate vaccinated noninfected cattle from virus carriers, such a vaccine would probably not be acceptable to countries or individuals that have BLV control programs. It would only be possible to add vaccinated animals to such herds if some method other than a serologic test for glycoprotein antigen was used to detect infected cattle. At present, however, no good alternative diagnostic method is available except in a very few research laboratories.
Recently, another type of vaccine has been investigated which does not cause BLV seroconversion in recipient cattle because the immunogen is a tumor cell line derived from non-BLV-infected (sporadic type) bovine lymphosarcoma.\textsuperscript{27,28} The trials to date are limited and nonconclusive, however, and there is no clear biologic explanation that would support expectations for such a vaccine to be protective against BLV infection.

It has also been suggested that embryo transfer may be a useful tool for preserving valuable genetic stock from BLV-infected cows. Some recent reports suggest that this approach would be scientifically valid.\textsuperscript{29,31} By combining the results of these studies, it appears that embryos from 66 BLV-infected donor cows have been transferred into a total of 116 BLV-negative recipients and 60 live calves have been produced. Results of serologic tests on the recipient cows and the calves indicate that there was no transmission of infective BLV during the transfer procedure. It should be noted, however, that the fluids of uterine flushings from some of these donors contained BLV-infected lymphocytes.\textsuperscript{32} This finding suggests that virus transmission would be possible if embryos were not carefully rinsed prior to transfer.

In spite of the apparent suitability of embryo transfer as a method for BLV control, it has been questioned whether the same goal might not be accomplished more economically by other methods.\textsuperscript{30} BLV-free calves from infected dams have been successfully reared in isolation, using a serologic test to detect any infected animals and then immediately removing them.\textsuperscript{12,22} In another study,\textsuperscript{33} calves from BLV-positive dams were fed colostrum from negative cows and reared in isolation; they remained virus-free at the end of 1 year. However, the recent evidence that suggests BLV can sometimes have a prolonged latency\textsuperscript{15} perhaps indicates there will always be a certain amount of risk in assuming an absolute BLV-negative status, especially in cattle that are known to have been exposed to the virus.

There has never been any government-sponsored program for bovine leukemia control in the United States. Major interest in the disease, at the national level, is the alleviation of difficulties created by the increasingly stringent BLV requirements that many countries are placing on the importation of our cattle. The most serious problem in this regard is to satisfy regulations which demand that cattle must come from BLV-negative herds. The Bluetongue-Bovine Leukosis Committee of the U.S. Animal Health Association has adopted a resolution recommending that the Animal and Plant Health Inspection Service of U.S.D.A. institute a voluntary program for the establishment and maintenance of BLV-free herds.\textsuperscript{34} The object of this proposal is to provide an official certification system that would not be an economic hardship for herd owners who wish to participate, but that would be acceptable to countries or individuals that wish to purchase BLV-free cattle. The committee's proposal is outlined below:
METHODS TO CONTROL BOVINE LEUKOSIS

Test — To be designated by the Deputy Administrator of Veterinary Services, APHIS.

Herd — One or more animals on common ground, separated from all other cattle by a minimum of 60 feet, with separate feed, water, and drainage system.

I. Qualifying the herd
   A. All animals in the herd shall be tested
   B. All reactors shall be removed and:
      1. Slaughtered, or
      2. Identified and segregated, or
      3. Identified and sold with a health certificate which indicates that it is BLV-infected
   C. The herd shall have 3 negative tests at 60- to 90-day intervals

II. Maintaining BLV-free status
   A. Additions to the herd
      1. If from a BLV-free herd, the animal must be negative to a test within 30 days of entry
      2. From other herds, animal must be negative in 2 tests, not less than 90 days apart, while in quarantine.
   B. Herd must be tested annually and remain completely negative

It is anticipated that the resolution recommending this proposal will be presented to the Executive Committee of the U.S.A.H.A. for its approval in 1982.

References


9. Schmidt F-W. Personal communication.


26. Portetelle D, Bruck C, Burny A, Dekegel D, Mammerickx M and Urbain J. Detection of
METHODS TO CONTROL BOVINE LEUKOSIS


INTRODUCTION

Bluetongue virus (BTV) infection of cattle is relatively common in the majority of western, central and southeastern regions of the United States. Serologic evidence of infection ranges from 6 to 60% of the cattle tested in these areas (1-3). Virus isolation techniques, which were employed on cattle blood samples obtained over a 4 year period in California, indicated that at certain times of the year (predominantly August through October) 4 to 8% of the cattle tested were viremic with BTV (3). The animals from which samples were collected throughout the survey did not show clinical signs of bluetongue (BT) disease (2,3). The prevalence of infection in the absence of clinical disease is not surprising in that numerous reports have documented an absence of overt clinical disease in field cases of BTV-infected cattle (2-9).

Bekker et al (10), in 1934, reported a clinical syndrome in cattle which was very similar to that seen in foot-and-mouth disease. This early report suggested that the disease was caused by BTV, based on similarities to BT in sheep and cross-protection studies in sheep using blood from diseased cattle and known BTV isolates. The clinical signs described included a transient febrile response, excessive salivation, dermatitis, localized inflammation with necrosis of the buccal mucosa, skin lesions of the udder, excoriation of the epidermis in the interdigital space, stiffness of gait, coronitis, lameness and loss of condition. Subsequent reports indicated that clinical signs of disease, similar to those described by Bekker et al, have been observed in cattle from which BTV was recovered (4,5,11-15). Metcalf et al suggested that the lesions associated with clinical BT disease in cattle were the result of a hypersensitive reaction induced by prior exposure to other serotypes of BTV or related viruses (13). Experimental reproduction of clinical BT in cattle has never been adequately demonstrated, and such infection typically results in only a mild febrile response, transient leukopenia and protracted period of viremia (4,5,10,16-18). The only suggestion of clinical BT experimentally reproduced in cattle, other than Bekker's early work, was that of Luedke et al in which a dermatitis was observed in 2 of 3 cattle
infected with BTV by the bites of infected *Culicoides variipennis* (15). The specific mechanism(s) by which the lesions and disease associated with BTV infection occur have not been defined. This report describes an experimental disease in cattle which closely resembles that described as clinical BT disease.

**MATERIALS AND METHODS**

**Virus Propagation and Inactivation**

BTV serotype 17 (62-45-S strain) was propagated in baby hamster kidney (BHK-21) cells and titrated in microtiter on fetal bovine bone marrow (FB-4-BM) cells. The virus (10⁶ tissue culture infectious doses/ml) was inactivated with chlorine dioxide (Alcide, Alcide Corp., Plainview, N.Y.). The final concentration of Alcide in the virus suspension was 2%. The virus-Alcide solution was held at 25°C for 1 hour and the reaction was stopped with sodium thiosulfate. All preparations of inactivated BTV were assayed for residual live virus by intravascular (IV) inoculation of embryonating chicken eggs (ECE), cell culture (BHK-21) and BTV susceptible sheep.

**Immunization**

The immunization (sensitization) schedule is summarized (Table I). Six Holstein calves (6 months of age) were used; 4 were sensitized principals and 2 were unsensitized controls. The 4 principals were sensitized by 2 subcutaneous (SC) injections (6 week interval) of 4 ml inactivated BTV in 4 ml aluminum hydroxide (AlOH) adjuvant. Immunopotentiators were administered simultaneously with the BTV inoculations; 2 calves received 2 doses of levamisol (levamisol phosphate, Pitman-Moore Inc., Washington Crossing, N.J.) (6 ml SC/dose) and 2 calves received 2 doses of cimetidine hydrochloride (Tagamet, Smith, Kline and French, Philadelphia, Pennsylvania) (300 mg SC and 300 mg IV/dose). The 4 principals and 2 controls were inoculated with virulent BTV serotype 17 (2.5 x 10⁵ embryo lethal doses) 76 days after the first inoculation of inactivated virus.

**Virus Isolation**

Virus isolation and titration, after virulent virus challenge of immunity, was by IV inoculation of 0.1 ml washed heparinized blood into each of 6 ECE. Positive viral isolates were identified as BTV by a plaque inhibition assay (19).

**Serology**

Group-specific antibodies to BTV were detected by agar gel immunodiffusion (AGID) (20). Serotype-specific antibodies were determined by a virus neutralization (VN) test in microtiter. Serial 5-fold dilutions of serum were used in the VN test. Results were recorded as the inverse of the highest serum dilution which neutralized ≥90% of 100 tissue culture infectious doses of BTV serotype 17.

**Cell Mediated Immunity**

A lymphocyte stimulation (LS) assay was utilized as an *in vitro*
correlate of cell mediated immunity (CMI). The presence of a CMI response, induced by sensitization and challenge with BTV, was determined by using BTV serotype 17 soluble antigen to stimulate the lymphocytes. A positive CMI response was considered to occur when the stimulation index (SI) \( [SI = \text{counts per minute (CPM) antigen stimulated} + \text{CPM negative control antigen stimulated}] \) and the \( \Delta \text{CPM (CPM antigen stimulated} - \text{CPM negative control antigen stimulated)} \) exceeded 2 and 1000 CPM, respectively.

**RESULTS**

Group-specific antibodies to BTV, as determined by AGID, were first detected 7 days after the second inoculation of inactivated virus. Neutralizing antibodies were not detected after either the first or second inoculation. A CMI response was observed in the 4 inoculated principals but the temporal appearance of this response was different between the 2 groups. The 2 calves inoculated with BTV plus cimetidine responded with a positive LS assay 36 days after the first injection; the 2 calves inoculated with BTV plus levamisole did not have a positive response until after the second injection.

After virulent virus challenge, the 4 principals developed VN antibodies 14 days post challenge (PC). A positive CMI response was detected, but quantitatively reduced, as compared to post-inoculation (PI) values. The 2 challenge controls developed VN antibodies 14 days PC, but a CMI response was not detected.

All 6 calves became viremic 4 to 7 days after challenge with virulent BTV serotype 17. Viremia persisted in the principals until 28 to 39 days and in the controls until 21 to 25 days PC. Immunologic and virologic results are summarized (Table II).

At 11 days PC the 4 principal calves developed clinical disease that was characterized by profuse salivation, ulceration of the oral mucosa, generalized hyperesthesia (most prominent over the dorsum and coronary bands), severe dermatitis (confined to thoracic and cervical areas) and inflammation of the coronary band. Clinical disease was not observed in the 2 challenge control calves.

Microscopic examination of the affected skin of the principals revealed a severe dermatitis characterized by multifocal intraepidermal spongiosis and neutrophilic exocytosis that often led to erosions, ulcerations and a surface exudate. The upper portions of the dermis exhibited a diffuse inflammatory reaction; there was a marked tendency toward perivascular cuffing of small caliber vessels. Cuffs were composed of lymphocytes, eosinophils, histiocytes, and a few mast cells. In addition to cellular components, there was perivascular edema and edema within dermal papillae.

**DISCUSSION**

The clinical disease experimentally induced by sensitization with
inactivated BTV, in association with immunomodulatory drugs, closely parallels that described as clinical BT in cattle. Prior experiments (unpublished data) that utilized only 1 inoculation of inactivated BTV, without immunomodulatory drugs, appeared to induce some protection to a subsequent virulent challenge of immunity, rather than the establishment of a sensitized condition that led to clinical disease.

Although these experiments need to be repeated, the results suggest that clinical BT disease in cattle may be immunologically mediated through sensitization to BTV, or BTV-related, antigen(s). The mechanism for this phenomenon is currently being studied. Preliminary studies (unpublished data) suggest that an immediate-type hypersensitivity occurs and involves a heat-labile serum factor, possibly IgE. This possibility is based on passive cutaneous anaphylaxis (PCA) studies using sera taken from the clinically sick principals in the present study. The PCA studies consisted of intradermal inoculation of the above mentioned hyperimmune serum and cell culture adapted BTV into the same site; this resulted in a wheal-and-flare reaction in the recipient calf.

### INOCULATION (SENSITIZATION) PROTOCOL

<table>
<thead>
<tr>
<th>Days Post Inoculation</th>
<th>2 calves</th>
<th>2 calves</th>
<th>2 calves</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 and 42</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*inactivated BTV-17</td>
<td>4 ml (SQ)(^a)</td>
<td>4 ml (SQ)</td>
<td>none</td>
</tr>
<tr>
<td>*adjuvant</td>
<td>4 ml A10H(^b)</td>
<td>4 ml A10H</td>
<td>none</td>
</tr>
<tr>
<td>*immunopotentiator</td>
<td>cimetidine (300 mg IV(^c)) &amp; 300 mg SC</td>
<td>levamisol (6 ml IV)</td>
<td>none</td>
</tr>
</tbody>
</table>

| 76                    |          |          |          |
| *virulent BTV-17      | 2.5 \(\times\) 10\(^5\) ELD\(_{50}\) SC | 2.5 \(\times\) 10\(^5\) ELD\(_{50}\) SC | 2.5 \(\times\) 10\(^5\) ELD\(_{50}\) SC |

a. SC = subcutaneous inoculation  
b. A10H = aluminum hydroxide adjuvant  
c. IV = intravenous inoculation  
d. ELD\(_{50}\) = embryo lethal dose, 50% endpoint
**Immunological and virological response of cattle to inoculation (inactivated bluetongue virus type 17) and challenge (live virulent bluetongue virus type 17) with bluetongue virus: A summary.**

<table>
<thead>
<tr>
<th>Animal number</th>
<th>Immunization</th>
<th>1st inoculation</th>
<th>2nd inoculation</th>
<th>Post immunization</th>
<th>Post challenge</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>AGID&lt;sup&gt;a&lt;/sup&gt;</td>
<td>VN&lt;sup&gt;b&lt;/sup&gt;</td>
<td>CMIC&lt;sup&gt;c&lt;/sup&gt;</td>
<td>AGID VN CMI</td>
</tr>
<tr>
<td>1</td>
<td>BTV-17/cimetidine</td>
<td>Neg</td>
<td>Neg</td>
<td>Pos</td>
<td>Pos Pos Pos</td>
</tr>
<tr>
<td>2</td>
<td>BTV-17/cimetidine</td>
<td>Neg</td>
<td>Neg</td>
<td>Pos</td>
<td>Pos Pos Pos</td>
</tr>
<tr>
<td>3</td>
<td>BTV-17/levamisol</td>
<td>Neg</td>
<td>Neg</td>
<td>Neg</td>
<td>Pos Pos Pos</td>
</tr>
<tr>
<td>4</td>
<td>BTV-17/levamisol</td>
<td>Neg</td>
<td>Neg</td>
<td>Neg</td>
<td>Pos Pos Pos</td>
</tr>
<tr>
<td>5</td>
<td>none (control)</td>
<td>Neg</td>
<td>Neg</td>
<td>Neg</td>
<td>Pos Pos Neg</td>
</tr>
<tr>
<td>6</td>
<td>none (control)</td>
<td>Neg</td>
<td>Neg</td>
<td>Neg</td>
<td>Pos Pos Neg</td>
</tr>
</tbody>
</table>

<sup>a</sup> AGID = agar gel immunodiffusion (detects group specific antibodies to BTV).

<sup>b</sup> VN = virus neutralization (detects serotype specific antibodies to BTV).

<sup>c</sup> CMI = cell mediated immunity (a lymphocyte stimulation assay utilizing BTV soluble antigen was used as a correlate of CMI).


The meeting was called to order at the scheduled time in the Jefferson Room. Twenty-two of the committee members were present; in addition there were over 30 interested USAHA members in attendance.

The committee considered the two subject areas separately with the initial discussion being led by Dr. Miller on bovine leukosis.

**BOVINE LEUKOSIS**

In a discussion of bovine leukosis virus control, 2 examples were presented. Dr. Janice Miller reported the eradication of virus from a commercial Jersey herd using the agar gel immunodiffusion test. At the first test in 1976, 15% of the adult herd was seropositive. During the next 3 years reactor cows were selectively culled. In 1979, the herd was retested and the virus prevalence was only 6%. All reactors were immediately removed and since that time the entire herd has been virus-free, as determined by yearly serologic tests. Dr. Don Abt described 2 calf-rearing trials in an experimental dairy herd with a 100% infection rate. One group of calves was raised in strict isolation and another group with less stringent separation requirements. Beginning at 6 months of age, the calves were tested at 2-3 month intervals by RIA and reactors were immediately removed. Of the animals that have reached 3 years of age, 35 of 41 remain free of infection. In contrast, a group of 18 control calves reared by conventional methods are all infected.

Dr. Janice Miller outlined some of the most important findings presented at the 5th International Symposium on Bovine Leukosis held in October, 1982, in Germany. Researchers in Belgium have produced a monoclonal antibody to a 15,000 dalton fragment of the virus glycoprotein which neutralizes infectivity. It is proposed to use this antibody to "catch" antigen on plates for use in ELISA testing, thus improving specificity. A report from Germany described calves which were held in isolation for 3 years after BLV exposure with no evidence of infection but during the 3rd year became serologically and virologically positive. This evidence of long virus latency in some animals was suggested as a deterrent to eradication of BLV in a few herds.
In Denmark, the BLV eradication program has been changed from one of herd-depopulation to removal of individual reactors. Also, the nationwide herd screening has been replaced by serologic surveillance of slaughter house specimens. Yugoslavia has instituted a control program in state-owned herds that is based on the segregation of BLV-positive cattle on isolated farms. Control programs in other countries are limited and conducted on a voluntary basis, but all report that the currently available technology is adequate for satisfactory control of virus spread.

The committee discussed a resolution to recommend that APHIS implement a voluntary certification program for BLV-free herds. After minor changes the resolution was unanimously adopted.

BLUETONGUE

Dr. James Pearson, Ames, Iowa, summarized activities at NVSL. The Immunodiffusion (ID) test is the official test for export to Canada. Forty-seven laboratories are approved by the USDA to conduct the bluetongue ID test for export of animals. NVSL supplied enough ID antigen to test over 300,000 animals last fiscal year. Approximately 28% of the serum samples submitted to the laboratory were positive in January, while 37% were positive in August, indicating a seasonal difference in antibody activity. Three hundred and ninety-five samples were tested for bluetongue (BT) and epizootic hemorrhagic disease (EHD); 15% were positive for EHD; however, the same number of samples were positive for both BTV and EHDV. There were 3 BTV isolates from outbreaks in sheep in Idaho, Utah and Nevada during 1982. Most viral isolates are typing out as 11. Virus isolation attempts on serologically negative bulls' semen were conducted on 4,306 collections and all were negative.

Dr. Livingston, Texas, reported on the seroconversion to four different serotypes of BT virus during the late winter and early spring months. There was no evidence of clinical disease in any of the sheep. Dr. Vic Nettles reported that the hemorrhagic syndrome continues in whitetailed deer in the Southeast. Epizootic hemorrhagic disease or bluetongue viruses were recovered from 3 different deer in 1982. Forty percent of the deer carry serological BTID titers for one or both viruses.

Dr. Lyn Barber described studies on Integrated Disease Management, inactivated virus vaccine, embryo transfer, immunotolerant cattle, improved diagnostic methods and the cooperative studies in Florida, Colorado, and California. Integrated Disease Management programs are underway in Colorado and Nebraska. Two 36-square mile areas in each state are being monitored for insects and virological and serological studies on ruminants. One area in each state has vector control programs. In one area 32 virus serotype 10 isolates were made from clinically normal cattle and sheep. Of twenty-seven cattle from which virus was isolated, 13 were BTID positive and 14 negative at the time of virus recovery. Four of six seronegative animals failed to seroconvert to the BTID test when tested up to three months later. Three of five sheep from which virus was recovered failed to seroconvert on the BTID test.
Inactivated vaccine virus studies are continuing with a new program being initiated in Oklahoma. Other means of inactivating the virus are being investigated.

A cooperative study between Colorado State University, Agriculture Canada, and ABADRL on the effects of bluetongue virus infection on embryo transfer is underway. Improved diagnostic testing procedures being developed include adaptation of the HIG test for EHDV, radioimmunoassay for quantitating antibody to BTV, ELISA assays on monoclonal antibody—hybridoma technology. Monoclonals have been developed to serotype 10. These antibodies are being adapted for fluorescent antibody assays and isolation of viral polypeptides. Cell mediated studies are underway on the detection of viral antigen in host tissues.

Cooperative studies were initiated with the Universities at Florida, Colorado State, and California following termination of the APHIS contracts in January, 1982. Funds have not yet been allocated for the 1983 fiscal year.

California studies have been directed to longitudinal epidemiological studies, fetal bovine infections, and new diagnostic technology. Epidemiologic studies include testing over 30,000 samples in 5 years. Over 450 BTV isolates have been made. Serotype 11 accounts for 60% of the virus isolates, followed by 17 at 21%, serotype 13 at 10% and serotype 10 at 9%. Approximately 40% of the cattle and 23% of the sheep from which virus was isolated did not have BTID antibodies. Virus isolations are seasonal, occurring between July and December. No persistent carrier animals have been identified. Bluetongue infection occurs in sheep which do not show evidence of disease. Studies on fetal bovine infection indicate that fetuses infected between 80 and 125 days gestation are killed by serotype 11, and EHDV whereas, serotypes 10, 13 and 17 cause hydranencephaly. BTID antibodies are present by 145 days gestation, neutralizing antibodies after 200 days gestation and virus is not recovered after 200 days gestation. Calves at birth were not immunotolerant, nor was virus present.

The diagnostic procedures now underway include monoclonal-hybridomas to serotype 17; ELISA system for antigen capture; identification of sub-populations of cells infected with virus; development of assay for viral RNA segments; and gene cloning.

Dr. Jack Pitcher, Maryland, presented information on changes in Federal policy and regulations. The policy on interstate movement of animals has been changed to include BT as a “reportable disease of all susceptible species” rather than sheep. Those animals showing clinical signs of disease are identified on health certificates. The new import policy statement was presented and discussed. The policy is being distributed to committee for review and comment. These comments will be submitted to Dr. Pitcher for review and consideration by APHIS.

Dr. Hugh Metcalf reported on responses to the proposal for “Iden-
An announcement was made that an International Workshop and Symposium on Bluetongue and Related Orbiviral Infections is being planned for Asilomar, California, January 17-21, 1984.

One resolution was proposed for bluetongue. This is being forwarded to the Resolution Committee for consideration.

RECOMMENDATION

(1) That policies on regulations proposed for change by APHIS on bluetongue and bovine leukemia be forwarded to the Committee Chair for distribution and discussion prior to the scheduled USAHA committee meetings.
AN EPIDEMIOLOGICAL STUDY OF AN ADULT VACCINATED JERSEY HERD INFECTED WITH BRUCELLA ABORTUS

Scott L. Reynolds, DVM, MSPH
James L. Alexander, DVM, MPVM
TEXAS ANIMAL HEALTH COMMISSION
Austin, Texas

INTRODUCTION

This report is an account of one approach to the management of brucellosis in a herd of dairy cows chronically infected since 1974. The herd had suffered losses of 124 cows involving three episodes of infection with *Brucella abortus*. This number included 12 reactors removed within 3 months prior to adult vaccination. No previous management procedures had been successful in controlling brucellosis.

MATERIALS AND METHODS

*Animals*

The registered Jersey herd consisted of both raised and replacement animals. Bulls were utilized in a controlled breeding program. The herd was positive to the Brucellosis Ring Test (BRT) at time of vaccination. All animals were tested ten days prior to inoculation and reactors removed from the herd. One hundred and nineteen cows were inoculated subcutaneously with $0.5 \times 10^9$ Strain 19 *Brucella abortus* organisms. All animals were bled and tested 120, 150, 180, 201, 222, 243, 303, 353, 366, 413, 443 and 540 days post vaccination. Cows showing a serological response were separated from the herd and those with calf were further isolated. Suspect cows showing titers after 150 days post vaccination were milked for culture assays. Animals showing negative cultures were remilked and cultured for a total of three milkings at 30 day intervals. One animal negative on initial culture assay was not available for further culture work. Animals showing positive cultures were slaughtered. Cattle showing a serological response after 8 months post vaccination were defined as a persistent titered (PT) animal.

CULTURE TECHNIQUES

*Milk*

The udders of suspect animals were washed and dried prior to milking. All quarters were milked and milk pooled as a composite sample in a whirl pack. Each specimen was immediately stored under dry ice until received at laboratory. Specimens were assayed by the State and Federal Cooperative Laboratory, Texas Animal Health Commission, Austin, Texas, according to procedures utilized by National Diagnostic Laboratory in Ames, Iowa, except Farrels media was substituted for W media.
**BRUCELLA Abortus**

**Lymph Nodes**

The suprarah Alpine, atlantal, parotid, mandibular, supra-mammary and internal ileac lymph nodes were removed from slaughtered suspect animals showing a negative milk culture. All tissues were packed in whirl packs and stored immediately in dry ice until received at the laboratory. Lymph nodes were removed from surrounding fat, dipped in alcohol, flamed, sliced, and placed in sterile physiological saline. Solid and liquid materials were separated by a Stonmacher. The liquid portion was assayed in the same manner as milk.

**CONVENTIONAL SEROLOGIC TESTS**

The Card, Rivanol and Tube Complement Fixation (CF) tests were performed by the State and Federal Cooperative Laboratory, Texas Animal Health Commission, Austin, Texas, and interpreted as prescribed in the Uniform Methods and Rules. The Rivanol and CF tests are routinely performed only on Card Positive animals.

**AUTOMATED SEROLOGICAL TEST**

The Technicon Auto Analyzer II, located at the State and Federal Laboratory, Texas Animal Health Commission, Austin, Texas, was used for automated Complement Fixation Tests (CFT). A detailed description of reagents may be found in the Technicon Instruction Manual on "Automated Complement Fixation Testing." Diagrams of equipment and the flow of serum and reagents have been pictured in detail.

**RESULTS**

Nineteen isolates of *B. abortus* were made from thirty-five animals. Fourteen of these isolates were identified as Bio Type I while five were Strain 19.

As shown in Table I, 12 animals at 120 days post vaccination showing CFT responses 20+ or greater were culture positive. Isolates of Strain 19 *B. abortus* were made from two of these cows. Cow No. 362 was culture positive for Field Strain but was negative on the card test and 80+ on the CFT. Cow No. V121 was culture positive for Strain 19 but was not tested on the CFT due to lack of serum. Cow No. T90 showed a CFT of 160+ and was negative at one milking. The lymph nodes were lost at slaughter, so the animal was not available for further culture assays. Cow No. T42 showed reactor level titers on all tests except a 10+ on the CFT. This animal was negative on milk culture and subsequently on all serological tests.

Table II summarizes serodiagnostic tests and culture results as compared to duration of titer on animals 120 days post vaccination. Five suspect animals showed a negative CFT and were negative on all tests at 150 days. Three suspect animals showed a negative CFT, were culture negative and subsequently registered negative on all serological tests on an average of 187 days post vaccination. One animal, No. W10 showed...
20+ on CFT but was negative on milk cultures. The cow became negative on CFT at 150 days and subsequently went negative to all tests in 201 days. Three suspects showed a negative CFT, were negative on milk and lymph nodes and showed persistent titers at time of slaughter. Three suspects showing a negative CFT were negative on milk cultures, subsequently were persistent titered and remain in the herd.

Table III summarizes suspects showing serological responses who were previously negative at 120 days post vaccination. Six animals had CFT titers of 20+ or greater at 150, 180, or 201 days respectively. Cow No. T37 showing a CFT of 20+, increased to a CFT of 80+ and was slaughtered at 180 days. W78 was slaughtered at 303 days. Her history will be summarized in Table IV. All six animals demonstrated positive cultures. Four animals were positive for field strain Brucella abortus and two for Strain 19. The last positive cultured animals in the herd were tested at 201 days post vaccination.

Table IV compares CFT titers with the card test. Cow No. 362 was 80+ on CFT test thirty days prior to a positive card test and was positive for field strain Brucella abortus. Cow No. 78 was 40+ on the CFT at 150 days and was slaughtered at 303 days without a positive card test at any time interval. After removal of cow No. 78, the BRT for the herd went negative. Culture assays of cow No. 78 revealed both milk and lymph nodes to be positive for Strain 19.

Why some infected cattle failed to show a positive card test was not determined. It is hypothesized that the negative test may be due to excess antibody; however, no tests were made to verify this assumption.

Table V compares the incidence of persistent titers with trimester of gestation at time of vaccination. Animals showed the highest incidence of titer persistence when vaccinated during the second trimester. No animal vaccinated when open showed a persistent titer.

Table VI summarizes a comparison between animals showing positive cultures for Strain 19 and trimester of gestation at time of vaccination. Animals vaccinated during the first two trimesters of gestation were negative for Strain 19. Three animals (11.54%) showed positive cultures for Strain 19 after vaccination during the third stage of gestation. One cow, No. V39, vaccinated when open showed a positive culture of Strain 19. Cow No. 78, was positive for Strain 19, but breeding history was unknown.

DISCUSSION

The results of this study indicate that the CFT at 21 day intervals was the most reliable test to predict reactors but was unable to discriminate between Field Strain or Strain 19 infection. Furthermore, no CFT negative animals were positive for Brucella abortus at any time interval studied. The last culture positive animal appeared in the herd 201 days post vaccination.
Other observations showed animals vaccinated at the second trimester of gestation were more prone to show persistent titers than animals vaccinated at other stages of gestation. Conversely, animals vaccinated in the 3rd trimester were more prone to have Strain 19 infected udders. One animal, open at time of vaccination, had a Strain 19 infected udder. This animal had calved three weeks prior to vaccination. A total of five animals (4.2%) experienced Strain 19 infected udders.

Test intervals were decreased from 30 to 21 days to enhance the probability of detecting infection prior to calving.

These data show that the addition of adult vaccination contributed greatly to the control of brucellosis in this herd. Other herds may respond differently especially when abortion after vaccination contributes to further exposures. The CFT was a reliable predictor of infection. How effective it will be under more severe conditions of exposure remains to be determined.

Whether the herd is truly free from brucellosis remains to be seen. Long term follow-up studies will be made to determine the efficacy of this management technique.

**TABLE I**

<table>
<thead>
<tr>
<th>ANIMAL</th>
<th>CARD</th>
<th>RIV.</th>
<th>CF</th>
<th>CFT</th>
<th>MILK LYMPH NODES</th>
<th>DURATION OF TITER</th>
</tr>
</thead>
<tbody>
<tr>
<td>*</td>
<td>P</td>
<td>200</td>
<td>164</td>
<td>160+</td>
<td>FS</td>
<td>SL</td>
</tr>
<tr>
<td>H266</td>
<td>P</td>
<td>200</td>
<td>164</td>
<td>80+</td>
<td>FS</td>
<td>SL</td>
</tr>
<tr>
<td>F99</td>
<td>P</td>
<td>50</td>
<td>164</td>
<td>80+</td>
<td>FS</td>
<td>SL</td>
</tr>
<tr>
<td>362</td>
<td>N</td>
<td>NT</td>
<td>NT</td>
<td>80+</td>
<td>FS</td>
<td>SL</td>
</tr>
<tr>
<td>V121</td>
<td>P</td>
<td>200</td>
<td>164</td>
<td>NA</td>
<td>S19</td>
<td>S19</td>
</tr>
<tr>
<td>T76</td>
<td>P</td>
<td>200</td>
<td>164</td>
<td>80+</td>
<td>S19</td>
<td>SL</td>
</tr>
<tr>
<td>V23</td>
<td>P</td>
<td>50</td>
<td>84</td>
<td>20+</td>
<td>S19</td>
<td>SL</td>
</tr>
<tr>
<td>T90</td>
<td>P</td>
<td>200</td>
<td>164</td>
<td>160+</td>
<td>NI</td>
<td>NA</td>
</tr>
<tr>
<td>T42</td>
<td>P</td>
<td>100</td>
<td>164</td>
<td>10+</td>
<td>NI</td>
<td>Neg. - 180 days</td>
</tr>
</tbody>
</table>

* 7 animals in group; NA - Not available; FS - Field Strain; S19 - Strain 19; NI - No Isolate; NT - No Test; SL - slaughtered
TABLE II

COMPARISON OF RESULTS OF SERODIAGNOSTIC TEST AND CULTURE RESULTS TO DURATION OF TITERS, ON ANIMALS 120 DAYS POST VACCINATION.

<table>
<thead>
<tr>
<th>ANIMAL</th>
<th>CARD</th>
<th>RIVANOL</th>
<th>CF</th>
<th>CFT</th>
<th>MILK LYMPH NODES</th>
<th>DURATION OF TITER</th>
</tr>
</thead>
<tbody>
<tr>
<td>W38</td>
<td>P</td>
<td>100</td>
<td>43</td>
<td>N</td>
<td></td>
<td>N 150 days</td>
</tr>
<tr>
<td>X19</td>
<td>P</td>
<td>25</td>
<td>41</td>
<td>N</td>
<td>NI</td>
<td>N 180 days</td>
</tr>
<tr>
<td>V82</td>
<td>P</td>
<td>25</td>
<td>82</td>
<td>N</td>
<td></td>
<td>N 150 days</td>
</tr>
<tr>
<td>*</td>
<td>P</td>
<td>25</td>
<td>N</td>
<td>N</td>
<td>NI(1)</td>
<td>N 150-180 days</td>
</tr>
<tr>
<td>W10</td>
<td>P</td>
<td>N</td>
<td>NT</td>
<td>20+</td>
<td>NI</td>
<td>N 201 days</td>
</tr>
<tr>
<td>786</td>
<td>P</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td></td>
<td>N 150 days</td>
</tr>
<tr>
<td>T80</td>
<td>P</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>NI</td>
<td>N 201 days</td>
</tr>
<tr>
<td>V117</td>
<td>P</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td></td>
<td>N 150 days</td>
</tr>
<tr>
<td>W65</td>
<td>P</td>
<td>50</td>
<td>N</td>
<td>N</td>
<td>NI</td>
<td>PT SL</td>
</tr>
<tr>
<td>053</td>
<td>P</td>
<td>50</td>
<td>41</td>
<td>N</td>
<td>NI</td>
<td>PT</td>
</tr>
<tr>
<td>W92</td>
<td>P</td>
<td>N</td>
<td>23</td>
<td>N</td>
<td>NI</td>
<td>PT SL</td>
</tr>
<tr>
<td>377</td>
<td>P</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>PT</td>
<td></td>
</tr>
<tr>
<td>X30</td>
<td>P</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>PT</td>
<td></td>
</tr>
<tr>
<td>V110</td>
<td>P</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>PT</td>
<td></td>
</tr>
</tbody>
</table>

* 2 animals in group; N - Negative; NI - No Isolate; PT - Persistent Titer; CF - Manual Complement Fixation Test; CFT - Automated Complement Fixation Test; NT - No Test; (1) - One Animal; SL - Slaughtered

TABLE III

COMPARISON OF RESULTS OF SERODIAGNOSTIC TESTS TO CULTURE RESULTS NOT PREVIOUSLY SHOWING A SEROLOGICAL RESPONSE.

150 Days Post Vaccination

<table>
<thead>
<tr>
<th>ANIMAL</th>
<th>CARD</th>
<th>RIVANOL</th>
<th>CF</th>
<th>CFT</th>
<th>MILK LYMPH NODES</th>
<th>CULTURE RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>T37</td>
<td>P</td>
<td>50</td>
<td>44</td>
<td>20+</td>
<td>FS</td>
<td>SL 180 days</td>
</tr>
<tr>
<td>W78</td>
<td>N</td>
<td>NT</td>
<td>NT</td>
<td>40+</td>
<td>S19 S19</td>
<td>SL 303 days</td>
</tr>
<tr>
<td>V39</td>
<td>P</td>
<td>N</td>
<td>22</td>
<td>20+</td>
<td>S19</td>
<td>SL</td>
</tr>
<tr>
<td>W107</td>
<td>P</td>
<td>50</td>
<td>44</td>
<td>40+</td>
<td>FS</td>
<td>SL</td>
</tr>
</tbody>
</table>

180 Days Post Vaccination

<table>
<thead>
<tr>
<th>ANIMAL</th>
<th>CARD</th>
<th>RIVANOL</th>
<th>CF</th>
<th>CFT</th>
<th>MILK LYMPH NODES</th>
<th>CULTURE RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>W74</td>
<td>P</td>
<td>25</td>
<td>24</td>
<td>20+</td>
<td>FS</td>
<td>SL</td>
</tr>
<tr>
<td>V54</td>
<td>P</td>
<td>50</td>
<td>164</td>
<td>160+</td>
<td>FS</td>
<td>SL</td>
</tr>
</tbody>
</table>

SL - Slaughtered.
BRUCELLA ABORTUS

TABLE IV

<table>
<thead>
<tr>
<th>ANIMAL</th>
<th>POST VACC.</th>
<th>CARD</th>
<th>RIVANOL</th>
<th>CF</th>
<th>CFT</th>
<th>MILK</th>
<th>LYMPH NODES</th>
</tr>
</thead>
<tbody>
<tr>
<td>362</td>
<td>120 days</td>
<td>N</td>
<td>NT</td>
<td>NT</td>
<td>80+</td>
<td>FS</td>
<td></td>
</tr>
<tr>
<td>150 days</td>
<td>P</td>
<td>50</td>
<td>83</td>
<td>80+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>W78</td>
<td>120 days</td>
<td>N</td>
<td>NT</td>
<td>NT</td>
<td>N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>150 days</td>
<td>N</td>
<td>NT</td>
<td>NT</td>
<td>40+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>180 days</td>
<td>N</td>
<td>NT</td>
<td>NT</td>
<td>80+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>201 days</td>
<td>N</td>
<td>NT</td>
<td>NT</td>
<td>160+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>222 days</td>
<td>N</td>
<td>NT</td>
<td>NT</td>
<td>80+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>243 days</td>
<td>N</td>
<td>25</td>
<td>161</td>
<td>80+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>303 days</td>
<td>N</td>
<td>50</td>
<td>164</td>
<td>160+</td>
<td>S19</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NT - No test; FS - Field Strain; S19 - Strain 19

TABLE V

COMPARISON OF TITER PERSISTENCE WITH STAGE OF GESTATION AT TIME OF VACCINATION

(1st) 4/34 = 11.8%  (2nd) 4/18 = 22.2%  (3rd) 3/26 = 11.5%
(Open) 0/32 = 0%

(Trimester)

# animals exhibiting titers 8 months Post Vaccination
# animals vaccinated during studied trimester

TABLE VI

COMPARISON OF ANIMALS EXHIBITING POSITIVE ST19 MILK CULTURE WITH STAGE OF GESTATION AT TIME OF VACCINATION.

(1st) 0/34 = 0%  (2nd) 0/18 = 0%  (3rd) 3/26/ = 11.54%
(Open) 1/32 = 3.12%

(Trimester)

# animals exhibiting titers 8 months Post Vaccination
# animals vaccinated during studied trimester

REFERENCES


Implementation of the Brucellosis Information System (BIS) began in Tennessee in April 1981 with the installation of computer equipment contracted from the Harris Corporation. The equipment consisted of one central processing unit with CRT console, one magnetic tape and disc drive unit, one 300-line printer, and seven CRT key entry terminals. Three portable interactive terminals were added later.

The Tennessee cooperative brucellosis program had incorporated automated data processing in the late 1950's. Until 1978 the system centered around an IBM 408 accounting machine and used key punch cards for entering and storing data. Data programs within this system could be easily modified to fit changes in program activities. However, it required large amounts of storage space for data cards and was quite slow compared to the current generation of computers. It required several additional pieces of large equipment to support the IBM 408, and comparatively, was quite limited in the range of data that could be entered and retrieved.

In 1978 the system was linked to a centralized state owned computer located in downtown Nashville. It continued until 1981 to use key punch cards for data entry. Data storage was provided in the computer database, thus eliminating the need to store used data cards. While this change eliminated some storage problems and added speed to the system, it was still limited in scope by the dimensions of the key punch data entry card.

One might ask why program managers in Tennessee would opt to implement the Brucellosis Information System when there was a fairly good automated data system already in place. Generally, the answer lies in the vastly increased amounts of program data that can be entered, stored, and retrieved in a variety of formats to aid in more efficiently managing the brucellosis program. When utilized to the fullest, the system can provide current, accurate data to guide brucellosis workers at all levels of responsibility, from the veterinary medical officer managing the infected herd to those at the highest levels of brucellosis program planning.

Following installation of the Harris equipment, the period from April to July 1981 was spent training various personnel in the actual operation of the equipment, and in implementing the new BIS data programs. There are two classes of programs involved in the system. One has to do with the entry of data and the other involves the transmission, processing, storage and retrieval of data.

Basically, all personnel involved in the brucellosis program were
required to be retrained to some degree. The data entry programs were developed by the APHIS Automated Data Systems Staff. The data transmission, processing, storage and retrieval programs were developed by the Fort Collins Computer Center. Altogether, there were four phases of basic training necessary before implementing the system. These were:

1) Preliminary training in operation of the Harris Computer equipment;
2) Training of clerical and data processing personnel in data entry by the Automated Data Systems Staff;
3) Training of the same personnel in data processing and retrieval by the Fort Collins Computer Center Staff, and;
4) Training of field and laboratory personnel involved with the program.

As you know, data for automated systems is coded. Although an automated system had been utilized for years in the Tennessee program, BIS incorporated an entirely new set of codes. This necessitated the retraining of animal health technicians, veterinary medical officers, and accredited practicing veterinarians in the use of the new codes as well as new record forms. This phase of training was considered to be essential since most brucellosis data originates at the field level. Accuracy and legibility of data when initially recorded is absolutely essential if a computerized data system is to be successful. Computers will frequently detect and point out errors. However, they tend more frequently to perpetuate them and invariably will not correct unless ordered to do so.

The first attempt at actual program data entry began July 20, 1981. After 3-4 weeks, the program was temporarily halted in order to correct several 'bugs' in the software and data entry programs, and to allow time for further development of the data processing programs at Fort Collins. Data entry resumed on October 1, 1981, and has continued uninterrupted to the present time.

In terms of activity and funding, the brucellosis program is by far the largest conducted by the cooperative regulatory agencies in Tennessee. Needless to say, the changes wrought by the implementation of BIS were not accomplished without some problems. Retrospectively, these problems can be categorized into four basic areas:

1. The implementation of BIS required concurrent operation of dual record systems until all phases of a particular BIS program became completely operational. This resulted in markedly increased work loads with attendant frustrations and lowered morale.

2. In the beginning, communication between participants technically oriented in brucellosis and those in computer technology left a great deal to be desired because neither completely understood the language or technology of the other.

3. The distance between the Automated Data Systems Staff in Washington, D.C., the Fort Collins, Computer Center in Ft. Collins, Colorado, and implementation of BIS in Tennessee hindered communication further and delayed timely correction of problems. This was further compounded by the fact that the separate data entry and data
retrieval programs were not complete in the beginning, and had not been completely field tested.

4. With virtually the entire state-federal work force involved, resistance to change was not the least of problems encountered. This was especially true with field personnel because of the difficulty in demonstrating immediate tangible benefits.

For the most part, major difficulties encountered thus far have been overcome. Virtually all the problems experienced in Tennessee can be attributed to the newness of the program and lack of specific applied experience. The experience gained during the past year by all participants no doubt should make implementation of BIS in other states much easier.

The remainder of this discussion will deal with the actual operation of BIS at the state level with a look at some of the things accomplished with the system. To date, programs for herd tests, market cattle testing, and the pending brucellosis work file (tub file) have been implemented. Modify-structure and add-delete subprograms have been added so that previously entered data can be corrected, added to, or deleted from. These programs are in place and working smoothly at present. Programs for the brucellosis milk ring test, calfhood vaccination, epidemiology, indemnity, and health certificates are in various stages of development at this time and will be implemented when completed.

One significant benefit derived from the system is the ready access to individual animal test data. This adds a valuable dimension to tracing animal movements so long as permanent identification is maintained. The previous automated system in Tennessee used only backtag identification which at best was good for no more than 3-4 weeks. On-farm test data was not included except as a herd test summary. BIS provides for entering as many as three animal identifications in addition to the backtag, with the capability of tracing an individual animal by any of them. For the Tennessee program at least, this is a decided improvement. Figure 1 is a completed copy of the recently revised brucellosis test record, VS Form 4-34, showing various types of animal identification that can be entered, stored, and retrieved by BIS.

A valuable product provided by BIS in the management of infected and certified herds is the preprinted test record with matching worksheet. Figures 2 and 3 show these documents. Following the initial test, subsequent retest charts are preprinted for use by field personnel. These charts list in alpha-numeric sequence each animal that should be accounted for on the succeeding test. Once all identification is entered into the system, any permanent or semi-permanent identification can be used as the primary identifier to facilitate handling of the herd. The field person managing the herd chooses the primary identifier.

The worksheet, each recording up to 84 animals, is provided to facilitate and simplify paper work during the actual testing of the herd.
Information is transferred from the worksheet to the VS Form 4-34, which is then forwarded to the laboratory with the blood samples. Animals removed from the herd since the previous test are noted so they may be deleted from the subsequent test record. Animals missed for some reason are noted but remain on the preprinted chart for subsequent retests. Additions to the herd, as well as corrections, are added manually. Properly annotated, the preprinted test record reflects not only the exact number of animals remaining in the herd, but also their individual identification. This can be a valuable tool in the hands of field personnel managing infected herds, especially large ones. In the case of certified free herds, the preprinted record provides a new dimension of accountability in individual animal identification and management.

One of the most time consuming activities in brucellosis field work is the tracing of reactors and exposed cattle to their origin. Tracing efforts are often conducted months or years after movements occur. These efforts frequently involve one or more markets and dealers. With only one year of identification accumulated in the database thus far, the potential for impressive savings in time and effort is obvious. The key, of course, is permanent identification. In traces involving cattle properly identified, the system provides previous test dates that serve as landmarks to guide the effort. The result is a marked reduction in the time and effort as compared with the manual record search. Further, it will often identify the point at which a trace can be safely terminated, as well as the relative degree of individual risk in traces involving more than one animal. In states with moderate to high levels of brucellosis, this feature alone will add greatly to the cost effectiveness of the system.

A wide variety of reports may be generated from the BIS data-base. The reports may be routine and recurrent, or they may be special reports involving a wide variety of parameters. Figures 4 and 5 show the monthly Report of Brucellosis Activities (4-331) as generated from BIS. When all data programs are implemented. The completed 4-33D report can be produced within minutes from data accumulated in the computer. Special reports can be generated on command to provide organized information of any data in the system. This is a valuable new tool in the overall management of the brucellosis program in Tennessee, and will be more valuable as the system expands.

What is the impact of BIS on the Tennessee program? Since the system is not complete, it is difficult to assess what the ultimate impact will be. However, after one year the positive aspects far outweigh the negative. On the other hand, this is not meant to imply that the implementation of BIS was without problems and frustrations.

The greatest impact has been upon office and data processing personnel. The BIS involved new data entry equipment, new data entry programs, new data codes, and the entry of data not previously entered. The original data entry keyboards had the typewriter configuration rather than the previously used standard data entry keyboard. Initially,
the central processing unit lacked sufficient storage capacity to receive and process data at the rate entered by data entry personnel. For several months, both the old and new systems were operated concurrently until the BIS could be sufficiently developed, programmed, and fine tuned. Needless to say, these situations resulted in slower production, increased work load, frustration, and at times, low morale.

As experience was gained and problems resolved, the impact on office and data entry personnel changed from negative to positive. Such things as the marked reduction in files and filing, the reduction of time and errors in reports preparation, and the speed with which program records could be processed added new interest and challenge to the jobs.

The greatest impact on field personnel involved the learning of new program element codes and the additional time required in completing the preprinted test record. The alpha-numeric listing of primary animal identification required some changes in routines and work habits developed over many years. Field personnel are the group from which most brucellosis data originates. Therefore, continued training and monitoring to assure high quality field data are essential to the system. Experience has resulted in an appreciation for what the system can do as an additional tool in the management of infected herds, and in reduction of time involved in tracing infected and exposed animals.

From the administrative standpoint, the immediate impact was that of dealing with such things as housing and installation of equipment, training of personnel, delays in work activities as a result of time taken for training, and personnel frustrations. Fortunately, time and experience have solved most of these problems. Most of the cost in implementing BIS was defrayed by the national program. Local incidental costs have been surprisingly modest. One of the most heartening aspects of BIS has been the development of a high degree of teamwork and esprit de corps among all personnel who work with the system. Although as yet not completely implemented, the value in being able to store, organize, and retrieve large amounts of reliable data is readily apparent. This can contribute greatly to the routine program decision-making process. This capability also provides dependable, accessible information to program officials who may become involved in litigation.

After one year of experience in the Tennessee brucellosis program, it can be concluded that BIS will not reduce the total number of required program personnel. Neither will it reduce the immediate workload or cost of an effective brucellosis program. Properly applied however, it does have the potential to help brucellosis workers at various levels to better manage all aspects of such a program. As a result, some impacts which the Tennessee cattle industry might anticipate are: (1) A more effective brucellosis program, (2) Reduced economic losses to brucellosis, (3) Freer movement of cattle and, (4) More rapid progress toward eradication.

In Tennessee, BIS is viewed as a desirable refinement of an adequate
comprehensive brucellosis program. It is not viewed as a cure for basic shortcomings in surveillance, diagnosis, infected herd management, or epidemiology.
### BRUCELLOSIS ERADICATION PROGRAM

**BRUCELLOSIS TEST RECORD**

**STATE:** Tennessee

**OWNER:**
- **OWNER NUMBER:**
- **ROUTE:** 320X
- **STREET:**
- **ROAD:**

**TEST:**
- **TEST NO.:** 1
- **TEST DATE:** 10/1/83

**COMPLETE HERD TEST OF ALL ELIGIBLE ANIMALS:**

<table>
<thead>
<tr>
<th>SEX</th>
<th>REACTOR</th>
<th>HAD IN HERD</th>
<th>NATURE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>YES</td>
<td></td>
</tr>
</tbody>
</table>

**DIAGNOSIS:**
- **DIAGNOSIS CODE:**
- **DIAGNOSIS:**

**LABORATORY RESULTS:**

<table>
<thead>
<tr>
<th>REACTANT TAG NUMBER</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>X 156</td>
<td></td>
</tr>
<tr>
<td>X 509</td>
<td></td>
</tr>
<tr>
<td>X 427</td>
<td></td>
</tr>
<tr>
<td>X 269</td>
<td></td>
</tr>
<tr>
<td>X 158</td>
<td></td>
</tr>
<tr>
<td>X 411</td>
<td></td>
</tr>
<tr>
<td>X 129</td>
<td></td>
</tr>
<tr>
<td>X 613</td>
<td></td>
</tr>
<tr>
<td>X 639</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE:**
- All incomplete records will be returned for completion.

**CERTIFICATION FOR PAYMENT:**
- **GENERAL EMPLOYEE:**
- **SIGNATURE:**
- **DATE:**

**REMARKS:**

- The test results are valid as of the date signed.
- This record is confidential and should not be released to any other party.

**FIGURE 2**
<table>
<thead>
<tr>
<th>TUBE#</th>
<th>TAG NUM3</th>
<th>TUBE#</th>
<th>TAG NUM3</th>
<th>TUBE#</th>
<th>TAG NUM3</th>
<th>TUBE#</th>
<th>TAG NUM3</th>
</tr>
</thead>
<tbody>
<tr>
<td>61ASD4317</td>
<td></td>
<td>63ACD5304</td>
<td></td>
<td>63ACD6104</td>
<td></td>
<td>63ACD6124</td>
<td></td>
</tr>
<tr>
<td>63ART7904</td>
<td></td>
<td>63AVT2301</td>
<td></td>
<td>63AVT2302</td>
<td></td>
<td>63AVT2303</td>
<td></td>
</tr>
<tr>
<td>T 310L</td>
<td></td>
<td>X 63F69802</td>
<td></td>
<td>X 63G36153</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Figure 3**
### SECTION A - BLOOD TESTS OF CATTLE AND OFFICIAL CALF VACCINATION

**EXPENSE**

<table>
<thead>
<tr>
<th>VS EMPLOYEE</th>
<th>FEE-BASIS (INCL. AMS)</th>
<th>STATE-COUNTY-CITY</th>
<th>INDUSTRY OR OWNER</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>01.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>02.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>03.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>04.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>05.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CATTLE TESTED ON FARM OR RANCH</th>
<th>TOTAL TESTED</th>
<th>INFECTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>HERDS</td>
<td>271</td>
<td>7,119</td>
</tr>
<tr>
<td>CATTLE</td>
<td>55</td>
<td>174</td>
</tr>
<tr>
<td>REACTORS</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TESTS OF MARKET CATTLE REGARDLESS OF ORIGIN</th>
</tr>
</thead>
<tbody>
<tr>
<td>CATTLE SAMPLED</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TOTAL CATTLE SAMPLED</th>
</tr>
</thead>
<tbody>
<tr>
<td>7,615</td>
</tr>
</tbody>
</table>

### SECTION B - MARKET CATTLE TESTING SUMMARY

<table>
<thead>
<tr>
<th>SAMPLES COLLECTED</th>
<th>ORIGINATING THIS STATE</th>
<th>ORIGINATING OUT OF STATE</th>
<th>TOTAL CATTLE SAMPLED</th>
</tr>
</thead>
<tbody>
<tr>
<td>BACKTAGGED CATTLE</td>
<td>TESTED</td>
<td>REACTORS</td>
<td>TESTED</td>
</tr>
<tr>
<td>RANCH OR FARM</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>LIVESTOCK MARKETS</td>
<td>7,673</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>FED. SLAUGHT. EST.</td>
<td>1,970</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>STATE SLAUGHT. EST.</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>TOTAL</td>
<td>9,383</td>
<td>34</td>
<td>0</td>
</tr>
</tbody>
</table>

| TOTAL REACTORS | 0 | 0 | 0 | 0 | 0 | 0 |

**FIGURE 4**
## SECTION F - REASON FOR HERD TESTS ON FARM OR RANCH

<table>
<thead>
<tr>
<th>REASONS FOR TESTS</th>
<th>TOTAL TESTED</th>
<th>INFECTED HERDS</th>
<th>PERCENT</th>
<th>INF. RATE PER 10,000 ANIMALS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HERDS</td>
<td>CATTLE</td>
<td>HERDS</td>
<td>CATTLE</td>
</tr>
<tr>
<td>31. BRT FOLLOW-UP:</td>
<td>6</td>
<td>112</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>32. HCT FOLLOW-UP:</td>
<td>33</td>
<td>1,282</td>
<td>9</td>
<td>181</td>
</tr>
<tr>
<td>33. AREA TESTS (INCL PRIV):</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>34. BRT FOLLOW-UP:</td>
<td>22</td>
<td>1,465</td>
<td>14</td>
<td>1,112</td>
</tr>
<tr>
<td>35. HCT FOLLOW-UP:</td>
<td>32</td>
<td>976</td>
<td>7</td>
<td>278</td>
</tr>
<tr>
<td>36. ALL OTHER TESTS:</td>
<td>717</td>
<td>6,168</td>
<td>30</td>
<td>1,029</td>
</tr>
<tr>
<td>TOTAL:</td>
<td>810</td>
<td>12,003</td>
<td>60</td>
<td>2,600</td>
</tr>
</tbody>
</table>

## SECTION G - OTHER SPECIES OF ANIMALS BLOOD TESTED

<table>
<thead>
<tr>
<th>SPECIES</th>
<th>CODE</th>
<th>TOTAL TESTED</th>
<th>INFECTED</th>
<th>SUSPECTED</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>HERDS/LOTS</td>
<td>ANIMALS</td>
<td>ANIMALS</td>
</tr>
<tr>
<td>39. GOATS</td>
<td>01</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>40. SWINE ON FARM</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>41. SWINE AT LIVESTOCK MKT</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>42. SWINE AT SLAUGHTER</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>43. SWINE TOTAL</td>
<td>02</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>44. SHEEP</td>
<td>04</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>45. HORSES</td>
<td>05</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>46. BISON</td>
<td>06</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>47. DEER</td>
<td>07</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>48. OTHER</td>
<td></td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**FIGURE 5**

Margaret E. Meyer, Ph.D., Department of Epidemiology and Preventive Medicine, School of Veterinary Medicine, University of California, Davis, California

and

L. C. Vanderwagen, D.V.M., California Department of Food and Agriculture, Bureau of Animal Health, Sacramento, California

INTRODUCTION

Between the years 1947 and 1957, California initiated a program to control and then to eradicate bovine brucellosis. During the first 13 years the program was in operation, i.e. 1947-1960, the prevalence of bovine brucellosis was reduced from approximately 20%\textsuperscript{12} to 1.2%. During the next decade (1960-1970) the prevalence of this disease was reduced another 0.82%, i.e. from 1.2% to 0.38%. This decrease in prevalence enabled California to be classified as Certified Free of bovine brucellosis in 1969. At the time of certification, evidence of infection persisted in only 23 herds.

In the decade of 1970-1980, instead of having a further reduction in prevalence, California suffered the "rebound" phenomenon. Infection recurred in herds in which the disease had been eradicated and it also appeared in herds previously uninfected. In fact, during the 10 year period 1970-1980, 273 herds (168 dairy and 105 beef) became infected.

Even though additional diagnostic techniques were introduced during 1970-1980, and more stringent and wide ranging additional regulations were intercalated into the control and eradication program, it has not yet been possible to reduce the prevalence of bovine brucellosis to the 1969 level of 0.38%, much less to achieve true eradication.

The purpose of this investigation was to explore records maintained by the California Department of Food and Agriculture to determine intra and extra program factors that differ in 1970-1980 from 1960-1970 and that also would have an influence on the prevalence of bovine brucellosis.

MATERIALS AND METHODS

All the data included herein were obtained from records kept by the California Department of Food and Agriculture, Bureau of Animal Health and the California Crop and Livestock Reporting Service.

RESULTS

Table 1 shows the "rebound" phenomenon as it occurred in California during the early 1970's following certification in 1969, as free of bovine brucellosis. In the 10 year period following certification, the number of herds that became infected during 1970, 1971, and 1972, were minimal but increased markedly, particularly among dairy herds, during 1973-75.
During the period 1975-80, the number of newly infected herds found each year declined from the mid-decade peak and since then has remained relatively constant. In the 1970 to 1980 decade, a total of 168 dairy and 105 beef herds became infected, but the maximum number of herds under quarantine at any one time was 44 in December of 1975.

Table 2 shows the number of cattle tested, number of reactors found, number of infected herds found, reactors per herd, and prevalence of bovine brucellosis by year for each year in the two decades. During the 1960 to 1970 time period, 2,095,493 head of cattle were tested, with 1,845,996 of them having been tested during the years of 1960 to 1966. During the year in which certification was achieved, only some 34,000 required testing. Also, while approaching certification the number of infected herds and the number of reactors found per year dropped concurrently. Interestingly, the number of reactors found per herd remained relatively constant and low.

During the 10 year period from 1970 to 1980, all of these circumstances were reversed. The number of cattle tested per year was 41,476 the first year following certification. This reached 100,000 plus by 1973-74 and each year since then has been between 200,000 and 250,000 animals tested. The total number of animals tested during the 1970 to 1980 decade was 1,572,086. The cause for the increased testing was the continual increase of newly infected herds and increase in the number of reactors found. The peak year for number of reactors found was 1979-1980 when 1,389 were disclosed by serotesting. The number of reactors per head markedly increased in 1970 to 1980 compared to the previous decade. In fact, the least number of reactors per herd during 1970-1980 (7.63 in 1970-71) exceeded the highest number found per herd during 1960 to 1970 (5.95 in 1962-63). Since 1971, the trend in the number of reactor animals per herd has been upward and stood at 24.27 for the year 1979-80.

Table 3 shows the numbers of beef and dairy calves vaccinated per year for each year in the two 10 year periods. The number of dairy calves vaccinated per year remained relatively constant and above 200,000 for each year and the total number vaccinated during each of the two decades was similar—2,020,819 during 1960 to 1970 and 2,045,247 during 1970 to 1980. The number of beef calves vaccinated during 1960 to 1970 was 1,641,332. This dropped to 895,310 during 1970 to 1980.

Table 4 shows the number of beef cattle (stockers and feeders) and dairy females inshipped into California during each of the 10 year periods. The number of beef cattle inshipped was similar—17 million plus during 1960 to 1970 and 16 million plus during 1970 to 1980. In contrast, there was a marked difference between these two decades in the number of dairy females inshipped into California. During 1970 to 1980 there were almost twice as many (345,401) dairy replacement animals brought into California from out of state than in 1960 to 1970 (183,535). At the same time, total dairy female populations has remained
essentially constant during the entire 20 year period under study (see Table 5).

Tables 6 and 7 show the numbers of dairy replacement animals inshipped into California by year and by state of origin for 1960 to 1970 and 1970 to 1980. A perusal of these tables shows very clearly the shift that has occurred during these two decades in the numbers of dairy replacement females shipped into California and the state of origin of these animals.

Table 8 shows the slaughter price per hundred weight for cattle by year for 1970 to 1980, and Table 9 shows the chronological order of additions and revisions to the regulations of the Bovine Brucellosis Control and Eradication Program.

Figure 1 shows the number of newly infected dairy herds found per year, number of dairy cattle replacement animals inshipped by year, and the slaughter price per hundred weight for cattle during 1970 to 1980.

DISCUSSION

California has maintained its certification as free of bovine brucellosis and currently stands eligible for classification as an "A" state. However, bovine brucellosis slowly but surely has been increasing in prevalence during the 10 year period from 1970 to 1980—up from 0.38% to 0.60% at the end of 1979. In fact, as of September 30, 1982, the prevalence had risen to 0.86% and California is precariously close to failing to qualify for "A" state status. A review of the additions and revisions of regulatory measures (Table 9) certainly indicates that programatically all possible has been and is being done to reduce the prevalence of this disease and achieve true eradication. These regulations have been implemented and are being enforced. Additionally, except for a decline in the level of vaccination of beef calves following withdrawing a payment of vaccination fees, the vaccination and testing level has proceeded uninterruptedly at a steady and/or increased pace over the past two decades. Clearly, factors outside program control have a direct or indirect influence on the prevalence of bovine brucellosis.

This increase in prevalence since 1970, and particularly the abrupt increase in 1973, has been attributed largely to assigning all available veterinary personnel to handling the Exotic Newcastle Disease outbreak with a resultant decrease in surveillance and of vigilence at the border stations. These consequences of the Newcastle Disease outbreak may have contributed to the prevalence increase in 1973, but at the same time, there was a marked increase in the number of dairy females inshipped into California (Table 4). This also was the time period in which importation patterns of state of origin started to shift. For example, during the entire decade of 1960-1970, only 639 dairy females were imported from Texas, while just in 1973 in the 1970-1980 decade, there were 1,200 dairy females inshipped from Texas (Table 6, 7). In retrospect, it is not possible to attribute the initiation of the prevalence increase entirely to
the demands of the Newcastle Disease outbreak, nor can the continuing increase in prevalence relate back to that outbreak.

Other extra-program circumstances were explored to determine their possible direct or indirect influence on prevalence and several relationships were found.

In so far as the beef herds are concerned, the drought in California, which started in 1976, precipitated a sell-off of 200,000 head of beef animals (Table 5). As a consequence of the increased number of MCI tests, infected herds were found that otherwise might have remained undiscovered. The herd populations were replenished in 1978 when 600,000 more stocker and feeder animals inshipped than during 1977. Clearly, adverse weather conditions had a profound effect on disease discovery, which contributes to prevalence data. Weather obviously also had a direct relationship to the sale, movement, and importation of animals into beef herds.

Several circumstances outside the realm of program control were found to be directly associated with increased prevalence in dairy herds.

Except for a temporary decline in the dairy cattle population during 1971 to 1973, the population has remained essentially constant over the last two decades (Table 5). However, the number of herds has decreased and average size of each herd has increased. In 1980 the average size dairy herd contained 307 animals, up from 200 in 1970. However, in Southern California, the location of almost all of the infected herds, it is not unusual for herds to contain from 500 to 3,500 animals. Since it is not possible to provide pastures for herds this size, they are kept under dry lot style management and quartered on very modest acreage. In these herds, animals are precision culled and, in some herds, there is a complete turn over of animals every 2 to 3 years. This crowding, management style, increase herd size and rapid animal turnover has taken place in the same time period in which the prevalence has remained on the increase and simultaneously, the number of reactors found per herd has steadily increased (Table 2).

One result of rapid animal turnover has been a remarkable increase of inshipments of dairy female replacement animals—up to 345,401 in 1970-1980 from 183,535 during the 1960-1970 decade. Because of the availability of animals at a more desirable price, the states of origin of these replacement animals has changed considerably. In the 10 year period of 1970-1980, California imported over 20,000 females from Texas compared to 639 for the whole of the 1960-1970 decade. In addition, since 1970 an increasing number of animals were inshipped from Oklahoma, Mississippi, Florida, Arkansas, Alabama, and Kentucky. While these animals meet the vaccination, status of herd of origin, and pre and post entry testing requirements, it is obvious that California is buying brucellosis via the serologically “negative,” exposed animal in the incubation stage of the disease.
Other non-program factors explored for possible influence on the increase of prevalence included support prices for commodities such as cheese, butter, and milk, and slaughter price per hundredweight for cattle. Milk price supports increased gradually during the last decade in relation to inflation, but were unrelated to variations in number of animals imported or to variations in numbers of infected herds per year. Cheese and butter support prices also were unrelated to importation or disease prevalence. The one factor that was directly related was slaughter price per hundred weight for cattle (see Figure 1).

Clearly, environmental and economic factors well beyond the regulatory aspects of the bovine brucellosis control and eradication program have a direct influence on, and are directly related to the prevalence of brucellosis in the California cattle population. In fact, these non-programmatic factors account for the increase in prevalence and, in effect, "tip the scales" in favor of the disease. This effect could be modified, and ultimately eliminated, if it were possible to identify exposed animals who are in the incubation stage of the disease and are not yet reactive to the diagnostic tests. Until that time comes, California, and other importing states, will continue to buy the disease with the animal.

REFERENCES


CONTROL AND ERADICATION OF BOVINE BRUCELLOSIS 157

TABLE 1

NUMBER OF BEEF AND DAIRY HERDS IN CALIFORNIA THAT BECAME INFECTED DURING 1970-1980

<table>
<thead>
<tr>
<th>Year</th>
<th>Number Dairy Herds</th>
<th>Number Beef Herds</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1970</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>1971</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>1972</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>1973</td>
<td>38</td>
<td>5</td>
<td>43</td>
</tr>
<tr>
<td>1974</td>
<td>24</td>
<td>10</td>
<td>34</td>
</tr>
<tr>
<td>1975</td>
<td>36</td>
<td>31</td>
<td>77</td>
</tr>
<tr>
<td>1976</td>
<td>22</td>
<td>12</td>
<td>34</td>
</tr>
<tr>
<td>1977</td>
<td>13</td>
<td>12</td>
<td>25</td>
</tr>
<tr>
<td>1978</td>
<td>16</td>
<td>10</td>
<td>26</td>
</tr>
<tr>
<td>1979</td>
<td>12</td>
<td>12</td>
<td>24</td>
</tr>
<tr>
<td>Total</td>
<td>168</td>
<td>105</td>
<td>273</td>
</tr>
</tbody>
</table>
### Table 2


<table>
<thead>
<tr>
<th>Year</th>
<th>Number Cattle Tested</th>
<th>Reactors Found</th>
<th>Number Infected Herds</th>
<th>Reactors Per Herd</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1960-61</td>
<td>517,603</td>
<td>4,848</td>
<td>1,392</td>
<td>3.48</td>
<td>.94</td>
</tr>
<tr>
<td>1961-62</td>
<td>329,688</td>
<td>2,579</td>
<td>532</td>
<td>4.85</td>
<td>.78</td>
</tr>
<tr>
<td>1962-63</td>
<td>273,045</td>
<td>1,965</td>
<td>330</td>
<td>5.95</td>
<td>.72</td>
</tr>
<tr>
<td>1963-64</td>
<td>295,480</td>
<td>1,852</td>
<td>451</td>
<td>4.11</td>
<td>.63</td>
</tr>
<tr>
<td>1964-65</td>
<td>296,433</td>
<td>1,308</td>
<td>400</td>
<td>3.27</td>
<td>.44</td>
</tr>
<tr>
<td>1965-66</td>
<td>133,747</td>
<td>1,055</td>
<td>290</td>
<td>3.64</td>
<td>.79</td>
</tr>
<tr>
<td>1966-67</td>
<td>94,077</td>
<td>580</td>
<td>175</td>
<td>3.31</td>
<td>.62</td>
</tr>
<tr>
<td>1967-68</td>
<td>80,439</td>
<td>333</td>
<td>98</td>
<td>3.40</td>
<td>.41</td>
</tr>
<tr>
<td>1968-69</td>
<td>41,656</td>
<td>94</td>
<td>31</td>
<td>3.03</td>
<td>.23</td>
</tr>
<tr>
<td>1969-70</td>
<td>33,325</td>
<td>126</td>
<td>23</td>
<td>5.48</td>
<td>.38</td>
</tr>
<tr>
<td>1970-71</td>
<td>41,476</td>
<td>229</td>
<td>30</td>
<td>7.63</td>
<td>.55</td>
</tr>
<tr>
<td>1971-72</td>
<td>26,655</td>
<td>150</td>
<td>17</td>
<td>8.82</td>
<td>.56</td>
</tr>
<tr>
<td>1972-73</td>
<td>49,603</td>
<td>325</td>
<td>21</td>
<td>15.38</td>
<td>.65</td>
</tr>
<tr>
<td>1973-74</td>
<td>101,650</td>
<td>768</td>
<td>46</td>
<td>16.70</td>
<td>.45</td>
</tr>
<tr>
<td>1974-75</td>
<td>256,167</td>
<td>1,384</td>
<td>61</td>
<td>22.69</td>
<td>.54</td>
</tr>
<tr>
<td>1975-76</td>
<td>256,100</td>
<td>1,512</td>
<td>82</td>
<td>18.44</td>
<td>.59</td>
</tr>
<tr>
<td>1976-77</td>
<td>204,713</td>
<td>915</td>
<td>45</td>
<td>20.33</td>
<td>.45</td>
</tr>
<tr>
<td>1977-78</td>
<td>212,264</td>
<td>1,002</td>
<td>57</td>
<td>17.58</td>
<td>.47</td>
</tr>
<tr>
<td>1978-79</td>
<td>167,832</td>
<td>1,029</td>
<td>47</td>
<td>21.89</td>
<td>.61</td>
</tr>
<tr>
<td>1979-80</td>
<td>255,626</td>
<td>1,389</td>
<td>57</td>
<td>24.37</td>
<td>.60</td>
</tr>
</tbody>
</table>
### TABLE 3

**NUMBER OF BEEF AND DAIRY CATTLE VACCINATED BY YEAR IN CALIFORNIA DURING 1960 TO 1970 AND 1970 TO 1980**

<table>
<thead>
<tr>
<th>Year</th>
<th>No. Dairy Calves Vaccinated</th>
<th>No. Beef Calves Vaccinated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1960</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1961</td>
<td>242,563</td>
<td>168,693</td>
</tr>
<tr>
<td>1962</td>
<td>230,674</td>
<td>161,690</td>
</tr>
<tr>
<td>1963</td>
<td>237,363</td>
<td>195,615</td>
</tr>
<tr>
<td>1964</td>
<td>226,493</td>
<td>195,453</td>
</tr>
<tr>
<td>1965</td>
<td>219,267</td>
<td>204,936</td>
</tr>
<tr>
<td>1966</td>
<td>206,541</td>
<td>193,627</td>
</tr>
<tr>
<td>1967</td>
<td>215,612</td>
<td>188,406</td>
</tr>
<tr>
<td>1968</td>
<td>226,828</td>
<td>182,555</td>
</tr>
<tr>
<td>1969</td>
<td>215,478</td>
<td>150,357</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2,020,819</strong></td>
<td><strong>1,641,332</strong></td>
</tr>
<tr>
<td>1970</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1971</td>
<td>211,891</td>
<td>107,809</td>
</tr>
<tr>
<td>1972</td>
<td>232,575</td>
<td>77,117</td>
</tr>
<tr>
<td>1973</td>
<td>232,041</td>
<td>57,815</td>
</tr>
<tr>
<td>1974</td>
<td>257,000</td>
<td>83,970</td>
</tr>
<tr>
<td>1975</td>
<td>246,826</td>
<td>78,585</td>
</tr>
<tr>
<td>1976</td>
<td>243,528</td>
<td>87,268</td>
</tr>
<tr>
<td>1977</td>
<td>248,612</td>
<td>89,219</td>
</tr>
<tr>
<td>1978</td>
<td>251,745</td>
<td>69,959</td>
</tr>
<tr>
<td>1979</td>
<td>220,698</td>
<td>108,962</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2,045,247</strong></td>
<td><strong>895,310</strong></td>
</tr>
<tr>
<td>Year</td>
<td>Number Stocker and Feeder Cattle Inshipped</td>
<td>Number Dairy Females Inshipped</td>
</tr>
<tr>
<td>------</td>
<td>------------------------------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>1960</td>
<td>1,435,000</td>
<td>26,578</td>
</tr>
<tr>
<td>1961</td>
<td>1,454,000</td>
<td>18,924</td>
</tr>
<tr>
<td>1962</td>
<td>1,829,000</td>
<td>15,797</td>
</tr>
<tr>
<td>1963</td>
<td>1,749,000</td>
<td>14,382</td>
</tr>
<tr>
<td>1964</td>
<td>1,539,000</td>
<td>18,011</td>
</tr>
<tr>
<td>1965</td>
<td>1,984,000</td>
<td>17,848</td>
</tr>
<tr>
<td>1966</td>
<td>1,902,000</td>
<td>18,432</td>
</tr>
<tr>
<td>1967</td>
<td>1,863,000</td>
<td>16,775</td>
</tr>
<tr>
<td>1968</td>
<td>1,870,000</td>
<td>15,284</td>
</tr>
<tr>
<td>1969</td>
<td>1,850,000</td>
<td>21,504</td>
</tr>
<tr>
<td>Total</td>
<td>17,475,000</td>
<td>183,535</td>
</tr>
<tr>
<td>1970</td>
<td>1,728,000</td>
<td>22,306</td>
</tr>
<tr>
<td>1971</td>
<td>1,803,000</td>
<td>32,755</td>
</tr>
<tr>
<td>1972</td>
<td>1,901,000</td>
<td>41,265</td>
</tr>
<tr>
<td>1973</td>
<td>1,823,000</td>
<td>49,566</td>
</tr>
<tr>
<td>1974</td>
<td>1,465,000</td>
<td>25,480</td>
</tr>
<tr>
<td>1975</td>
<td>1,809,000</td>
<td>25,328</td>
</tr>
<tr>
<td>1976</td>
<td>1,323,000</td>
<td>22,802</td>
</tr>
<tr>
<td>1977</td>
<td>1,381,000</td>
<td>26,230</td>
</tr>
<tr>
<td>1978</td>
<td>1,915,000</td>
<td>44,786</td>
</tr>
<tr>
<td>1979</td>
<td>1,457,000</td>
<td>54,883</td>
</tr>
<tr>
<td>Total</td>
<td>16,605,000</td>
<td>345,401</td>
</tr>
</tbody>
</table>
### TABLE 5
CATTLE POPULATION (COWS AND HEIFERS THAT CALVED)
BY YEAR FOR 1960 TO 1970 AND 1970 TO 1980

<table>
<thead>
<tr>
<th>Year</th>
<th>Dairy Cattle Population</th>
<th>Beef Cattle Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>1960</td>
<td>899,000</td>
<td>851,000</td>
</tr>
<tr>
<td>1961</td>
<td>899,000</td>
<td>858,000</td>
</tr>
<tr>
<td>1962</td>
<td>881,000</td>
<td>854,000</td>
</tr>
<tr>
<td>1963</td>
<td>881,000</td>
<td>927,000</td>
</tr>
<tr>
<td>1964</td>
<td>867,000</td>
<td>956,000</td>
</tr>
<tr>
<td>1965</td>
<td>858,000</td>
<td>1,004,000</td>
</tr>
<tr>
<td>1966</td>
<td>849,000</td>
<td>1,035,000</td>
</tr>
<tr>
<td>1967</td>
<td>857,000</td>
<td>995,000</td>
</tr>
<tr>
<td>1968</td>
<td>848,000</td>
<td>973,000</td>
</tr>
<tr>
<td>1969</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1970</td>
<td>775,000</td>
<td>894,000</td>
</tr>
<tr>
<td>1971</td>
<td>758,000</td>
<td>916,000</td>
</tr>
<tr>
<td>1972</td>
<td>766,000</td>
<td>906,000</td>
</tr>
<tr>
<td>1973</td>
<td>789,000</td>
<td>915,000</td>
</tr>
<tr>
<td>1974</td>
<td>810,000</td>
<td>1,054,000</td>
</tr>
<tr>
<td>1975</td>
<td>800,000</td>
<td>1,097,000</td>
</tr>
<tr>
<td>1976</td>
<td>810,000</td>
<td>1,010,000</td>
</tr>
<tr>
<td>1977</td>
<td>809,000</td>
<td>991,000</td>
</tr>
<tr>
<td>1978</td>
<td>846,000</td>
<td>966,000</td>
</tr>
<tr>
<td>1979</td>
<td>860,000</td>
<td>930,000</td>
</tr>
</tbody>
</table>
### Table 6

**State of Origin of Dairy Cattle Inshipped to California by Year 1960 - 1970**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Arizona</td>
<td>1,298</td>
<td>1,111</td>
<td>598</td>
<td>425</td>
<td>1,296</td>
<td>1,559</td>
<td>376</td>
<td>1,336</td>
<td>499</td>
<td>430</td>
<td>7,728</td>
</tr>
<tr>
<td>Colorado</td>
<td>4,483</td>
<td>1,893</td>
<td>589</td>
<td>383</td>
<td>84</td>
<td>39</td>
<td>116</td>
<td>48</td>
<td>318</td>
<td>689</td>
<td>8,642</td>
</tr>
<tr>
<td>Idaho</td>
<td>5,016</td>
<td>4,167</td>
<td>5,140</td>
<td>4,342</td>
<td>4,809</td>
<td>7,667</td>
<td>8,567</td>
<td>5,432</td>
<td>3,753</td>
<td>6,542</td>
<td>55,444</td>
</tr>
<tr>
<td>Iowa</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>199</td>
<td>145</td>
<td>64</td>
<td>-</td>
<td>-</td>
<td>321</td>
<td>1,150</td>
<td>1,869</td>
</tr>
<tr>
<td>Kansas</td>
<td>4</td>
<td>60</td>
<td>182</td>
<td>297</td>
<td>56</td>
<td>-</td>
<td>2</td>
<td>-</td>
<td>71</td>
<td>36</td>
<td>708</td>
</tr>
<tr>
<td>Minnesota</td>
<td>57</td>
<td>55</td>
<td>40</td>
<td>208</td>
<td>403</td>
<td>30</td>
<td>1</td>
<td>5</td>
<td>459</td>
<td>1,475</td>
<td>2,733</td>
</tr>
<tr>
<td>Missouri</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>30</td>
<td>50</td>
<td>-</td>
<td>202</td>
<td>28</td>
<td>-</td>
<td>-</td>
<td>210</td>
</tr>
<tr>
<td>Montana</td>
<td>1,225</td>
<td>519</td>
<td>656</td>
<td>892</td>
<td>358</td>
<td>51</td>
<td>-</td>
<td>-</td>
<td>76</td>
<td>36</td>
<td>3,815</td>
</tr>
<tr>
<td>Nevada</td>
<td>1,788</td>
<td>2,316</td>
<td>1,074</td>
<td>953</td>
<td>1,511</td>
<td>1,002</td>
<td>1,926</td>
<td>2,240</td>
<td>1,644</td>
<td>1,211</td>
<td>15,665</td>
</tr>
<tr>
<td>Oklahoma</td>
<td>43</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>25</td>
<td>72</td>
<td>140</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Oregon</td>
<td>458</td>
<td>601</td>
<td>358</td>
<td>220</td>
<td>664</td>
<td>387</td>
<td>624</td>
<td>363</td>
<td>420</td>
<td>779</td>
<td>4,874</td>
</tr>
<tr>
<td>Texas</td>
<td>1</td>
<td>-</td>
<td>23</td>
<td>56</td>
<td>262</td>
<td>-</td>
<td>293</td>
<td>-</td>
<td>40</td>
<td>-</td>
<td>639</td>
</tr>
<tr>
<td>Utah</td>
<td>10,435</td>
<td>6,765</td>
<td>5,869</td>
<td>5,226</td>
<td>6,861</td>
<td>5,822</td>
<td>5,133</td>
<td>6,488</td>
<td>6,455</td>
<td>7,220</td>
<td>66,274</td>
</tr>
<tr>
<td>Washington</td>
<td>1,196</td>
<td>1,468</td>
<td>1,030</td>
<td>699</td>
<td>987</td>
<td>930</td>
<td>998</td>
<td>761</td>
<td>254</td>
<td>292</td>
<td>8,215</td>
</tr>
<tr>
<td>Wisconsin</td>
<td>98</td>
<td>11</td>
<td>102</td>
<td>195</td>
<td>459</td>
<td>72</td>
<td>134</td>
<td>42</td>
<td>268</td>
<td>445</td>
<td>1,826</td>
</tr>
<tr>
<td>Other</td>
<td>476</td>
<td>358</td>
<td>136</td>
<td>267</td>
<td>66</td>
<td>223</td>
<td>160</td>
<td>32</td>
<td>691</td>
<td>1,127</td>
<td>3,536</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>26,578</td>
<td>18,924</td>
<td>15,797</td>
<td>14,382</td>
<td>18,011</td>
<td>17,848</td>
<td>18,432</td>
<td>18,775</td>
<td>15,284</td>
<td>21,504</td>
<td>183,535</td>
</tr>
</tbody>
</table>
CONTROL AND ERADICATION OF BOVINE BRUCELLOSIS 163

### TABLE 7
STATE OF ORIGIN OF DAIRY CATTLE INSHIPPED TO CALIFORNIA
BY YEAR 1970 TO 1980*

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Arizona</td>
<td>149</td>
<td>1,701</td>
<td>687</td>
<td>3</td>
<td>-</td>
<td>34</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>2,574</td>
</tr>
<tr>
<td>Colorado</td>
<td>1,713</td>
<td>2,308</td>
<td>4,404</td>
<td>6,869</td>
<td>2,114</td>
<td>1,233</td>
<td>934</td>
<td>833</td>
<td>2,424</td>
<td>2,859</td>
<td>25,691</td>
</tr>
<tr>
<td>Idaho</td>
<td>6,637</td>
<td>11,282</td>
<td>14,261</td>
<td>16,085</td>
<td>12,468</td>
<td>12,730</td>
<td>10,195</td>
<td>12,165</td>
<td>13,507</td>
<td>13,039</td>
<td>122,369</td>
</tr>
<tr>
<td>Iowa</td>
<td>1,310</td>
<td>782</td>
<td>2,036</td>
<td>3,729</td>
<td>557</td>
<td>48</td>
<td>1,069</td>
<td>207</td>
<td>2,841</td>
<td>3,272</td>
<td>16,311</td>
</tr>
<tr>
<td>Kansas</td>
<td>129</td>
<td>-</td>
<td>205</td>
<td>39</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>362</td>
<td>2,275</td>
<td>2,420</td>
<td>5,431</td>
</tr>
<tr>
<td>Minnesota</td>
<td>260</td>
<td>537</td>
<td>847</td>
<td>1,180</td>
<td>120</td>
<td>23</td>
<td>198</td>
<td>12</td>
<td>-</td>
<td>2,092</td>
<td>5,269</td>
</tr>
<tr>
<td>Missouri</td>
<td>-</td>
<td>-</td>
<td>504</td>
<td>-</td>
<td>-</td>
<td>169</td>
<td>299</td>
<td>481</td>
<td>1,278</td>
<td>2,731</td>
<td></td>
</tr>
<tr>
<td>Montana</td>
<td>71</td>
<td>324</td>
<td>-</td>
<td>-</td>
<td>208</td>
<td>13</td>
<td>103</td>
<td>589</td>
<td>343</td>
<td>1,651</td>
<td></td>
</tr>
<tr>
<td>Nevada</td>
<td>1,648</td>
<td>2,837</td>
<td>2,587</td>
<td>3,317</td>
<td>21</td>
<td>1,974</td>
<td>2,426</td>
<td>3,697</td>
<td>1,988</td>
<td>1,981</td>
<td>22,478</td>
</tr>
<tr>
<td>Oklahoma</td>
<td>-</td>
<td>61</td>
<td>159</td>
<td>122</td>
<td>9</td>
<td>-</td>
<td>36</td>
<td>16</td>
<td>-</td>
<td>215</td>
<td>618</td>
</tr>
<tr>
<td>Oregon</td>
<td>568</td>
<td>1,205</td>
<td>1,024</td>
<td>1,942</td>
<td>447</td>
<td>719</td>
<td>842</td>
<td>860</td>
<td>469</td>
<td>475</td>
<td>8,551</td>
</tr>
<tr>
<td>Texas</td>
<td>188</td>
<td>523</td>
<td>788</td>
<td>1,205</td>
<td>769</td>
<td>1,091</td>
<td>832</td>
<td>965</td>
<td>3,759</td>
<td>9,917</td>
<td>20,037</td>
</tr>
<tr>
<td>Utah</td>
<td>7,572</td>
<td>8,771</td>
<td>10,760</td>
<td>8,871</td>
<td>4,602</td>
<td>6,158</td>
<td>4,964</td>
<td>4,807</td>
<td>5,421</td>
<td>4,818</td>
<td>66,744</td>
</tr>
<tr>
<td>Washington</td>
<td>176</td>
<td>56</td>
<td>277</td>
<td>88</td>
<td>100</td>
<td>420</td>
<td>67</td>
<td>84</td>
<td>897</td>
<td>325</td>
<td>2,490</td>
</tr>
<tr>
<td>Wisconsin</td>
<td>785</td>
<td>768</td>
<td>936</td>
<td>1,079</td>
<td>155</td>
<td>115</td>
<td>104</td>
<td>215</td>
<td>2,228</td>
<td>2,496</td>
<td>8,883</td>
</tr>
<tr>
<td>Other*</td>
<td>1,100</td>
<td>1,514</td>
<td>1,608</td>
<td>2,545</td>
<td>1,010</td>
<td>14</td>
<td>51</td>
<td>146</td>
<td>6,666</td>
<td>58</td>
<td>14,712</td>
</tr>
</tbody>
</table>

* See text for additional information on inshipments during this period.
TABLE 8

SLAUGHTER PRICES FOR CATTLE PER HUNDREDWEIGHT
BY YEAR 1970 - 1980

<table>
<thead>
<tr>
<th>Year</th>
<th>Price</th>
</tr>
</thead>
<tbody>
<tr>
<td>1970</td>
<td>$27.50</td>
</tr>
<tr>
<td>1971</td>
<td>30.40</td>
</tr>
<tr>
<td>1972</td>
<td>33.50</td>
</tr>
<tr>
<td>1973</td>
<td>42.90</td>
</tr>
<tr>
<td>1974</td>
<td>39.40</td>
</tr>
<tr>
<td>1975</td>
<td>36.30</td>
</tr>
<tr>
<td>1976</td>
<td>36.60</td>
</tr>
<tr>
<td>1977</td>
<td>36.00</td>
</tr>
<tr>
<td>1978</td>
<td>48.00</td>
</tr>
<tr>
<td>1979</td>
<td>65.80</td>
</tr>
<tr>
<td>1980</td>
<td>63.80</td>
</tr>
<tr>
<td>1981</td>
<td>57.30</td>
</tr>
</tbody>
</table>
CONTROL AND ERADICATION OF BOVINE BRUCELLOSIS 165

TABLE 9

REGULATIONS ADDED TO THE CALIFORNIA BOVINE BRUCELLOSIS
CONTROL AND ERADICATION PROGRAM 1960-1970
AND 1970-1980

1960 Market Cattle Testing Program adopted. All counties in California became included in control program.

1961 Dairy females inshipped required to have proof of calfhood vaccination.

1963 Federal "stop-fee" increased.

1964 Official vaccination age for dairy calves set at 4 to 8 months of age.

1966 Increased sensitivity BRT test introduced, as were the acidified plate antigen and rivanol supplemental tests.

1967 Two way back tag introduced.

1968 Minimum age for calfhood vaccination lowered from 4 months to 3 months. Federal "stop-fee" and per head fee for vaccination stopped.

1969 California became Certified Free of bovine brucellosis.

1970 Payment for vaccinations stopped.

1973 Unvaccinated beef animals for inshipment from non-brucellosis free states requires to have 30-day prior negative tests.

Animals for inshipment required to have permit prior to entry.
TABLE 9 (CONTINUED)

1974 Surveillance by MCI program increased (dairy breeds).

1974 All beef animals for inshipment, except those from neighboring states required to have prior 30-day negative test.

Animals inshipped required to have 30-day post importation test.

1976 Restrictions placed on movement of dairy animals in two counties in Southern California.

1976 Vaccination age lowered to 2 months.

1976 Vaccination required for intrastate movement (dairy breeds).

1977 Regulations imposed requiring cleaning and disinfecting of transport vehicles.

1978 30-day post importation test required on all cattle inshipped except those from certified free states.

Entry permit no longer required for native cattle of neighboring states or California cattle returning from neighboring states.
Figure 1

DAIRY CATTLE IMPORTS, SLAUGHTER PRICE PER CWT AND
HERDS NEWLY INFECTED WITH BRUCELLOSIS BY YEAR 1970 - 1980
STATUS OF THE COOPERATIVE STATE-FEDERAL
BRUCELLOSIS ERADICATION PROGRAM

Billy G. Johnson, D.V.M.
Hyattsville, Maryland

A major revision of the Brucellosis Eradication Uniform Methods and Rules (UM&R) became effective May 1, 1982. This revision changed both classification standards and movement requirements within and between areas.

The basis for these changes was the Brucellosis Technical Commission's report of August 1978. Based on recommendations in that report, the system was changed from county certification to State certification. The Commission's report recommended that a three-status system be adopted with Class A being those States free of brucellosis, Class B being those with low to medium incidence, and Class C those of highest incidence. Based on recommendations from the Brucellosis Committee of the United States Animal Health Association (USAHA), those proposals were amended to provide for four status areas titled Free, A, B, and C. The criteria for determining status included herd infection rates, infection found through the Market Cattle Inspection (MCI) Program, and the effectiveness of the surveillance programs. The original proposals were amended to allow a State to have up to two different areas under certain conditions.

SLIDE 1

As of November 1, 1982, there are 12 Class Free States, 25 Class A, 9 Class B, and 4 Class C States. Two States, Florida and Texas, requested and were granted permission to establish two classification areas; each State being divided between Class B and Class C.

Animal movement requirements differ for intrastate and interstate movements. Cattle from Class Free States may be moved without prior tests. A post-movement test is recommended. Cattle may move within a Class A State without test but must be tested negative within 30 days of an interstate movement. Cattle from Class B States must be tested within 30 days of movement and, if moved interstate, are to be quarantined and restested 45 to 120 days after movement. A permit is required for interstate movement. Movements within a Class C State must be made based on a negative test within 30 days of the movement. Vaccinated cattle moving interstate from a Class C State must be tested within 30 days of movement and nonvaccinates must be negative to two tests conducted at least 60 days apart. Both classes must have a permit for the interstate movement and must be quarantined and restested 45 to 120 days after test. Certain classes, such as steers, spayed heifers, cattle under 18 months, vaccinated dairy animals less than 20 months, or vaccinated beef cattle under 24 months of age from herds not under quarantine, are exempt from test.
For purposes of computing data for this visual, States were classified as they were prior to May 1, 1982. For the first time in three years there was significant reduction in the total number of infected herds. There were 11,597 infected herds found compared with 13,228 during Fiscal Year 1981.

This accounts for all herds with at least one reactor disclosed during the year. There were 39 States with either no infected herds during 1982 or fewer infected herds than found during the previous years.

Four Certified Free States had significant increases in infection. These were California, Indiana, Nevada, and Washington.

The percentage of infection in the 40 Free and low incidence States as compared to the Nation's total continues to decline. There were 30 States, each with less than 30 infected herds, representing only 1.5 percent of the total. An additional 10 States revealed 787 infected herds or 6.8 percent of the total. These 40 States accounted for 8.3 percent of the Nation's total. The remaining 10 States accounted for 10,635 infected herds (91.7 percent of the total). Four States, Texas, Florida, Mississippi, and Louisiana accounted for 59.8 percent.

The number of dairy herds found suspicious to the brucellosis ring test (BRT), the number of those herds tested and the number found infected, declined from last year. There were 263 infected dairy herds found as a result of BRT tests compared to 353 last year. This year's total is the lowest since the eradication program began.

A small increase was seen in the number of cattle tested under the MCI program. There were 12.3 million cattle tested in 1982 compared with 11.8 million in 1981. The increase occurred in cattle tested at slaughtering establishments. Standards added to the UM&R under the new classification system place increased emphasis on sample collection at slaughter. Increased efficiency in this area of the program will improve traceback procedures to infected herds.

The total cattle tested fell slightly from 1981 as fewer cattle were tested on farms. This resulted from the lower number of infected herds and less area testing. The reactor rate in cattle tested under the MCI program was 0.41 percent as compared to 0.42 percent last year. More card positive rivanol negative cattle were counted in 1982 causing a smaller drop in the MCI reactor rate than otherwise would have occurred. The reactor rate for all cattle tested was 0.90 percent.
The upward trend in vaccination of calves continued to a total of 7.5 million. This is the highest number of heifers ever vaccinated in one year in this country. Thirteen States had over 200,000 heifers vaccinated this year. Texas exceeded 800,000 while both Oklahoma and Wisconsin exceeded 400,000.

Fiscal Year 1982 was the first full year that the swine brucellosis program operated under the major revisions in the UM&R that went into effect April 1, 1981. These revisions were to provide flexibility to the program and to renew interest in States that considered the previous validation standards too rigid and virtually unattainable. There are indications that this is occurring. Three States, Maryland, Delaware, and Arizona, all utilized the new surveillance standards to qualify for validated-free status during the year and several others are close to that goal and expected to qualify in FY 1983.

The total number of swine tested for brucellosis decreased from 3.9 million in FY 1981 to 2.8 million in FY 1982. This total includes 2.2 million sows and boars tested under the Market Swine Testing (MST) Program and 529,000 tested on farms. The decrease in total animals tested is attributed to a 15 percent reduction in sow and boar slaughter from FY 1981, to marketing changes that shifted slaughter away from large volume plants with active contract blood collection programs and to the cessation of sampling in some skinning plants.

The MST reactor rate declined from 0.06 in FY 1981 to 0.046 in FY 1982. A similar decline occurred in the reactor rate of breeding swine tested on farms.

The April 1, 1981, UM&R introduced a new 3-stage validation program and early in FY 1982 each State was assigned a stage on the basis of their answers to a questionnaire on its program. Validated-free States automatically qualified for Stage III. Other assignments were based on compliance with UM&R standards and on meeting specific requirements designated as mandatory for each stage by the Swine Brucellosis Subcommittee at last year's meeting.

During the year three States, Arizona, Delaware, and Maryland, qualified for Stage III, or Validated Brucellosis-Free Area status, bringing the total number of Stage III States to 23. In addition, California, Colorado, Idaho, Iowa, Maine, Minnesota, Montana, Nevada, New Hampshire, North Dakota, Pennsylvania, Rhode Island, South Dakota, Utah, Vermont, Washington, Wisconsin, Wyoming, Puerto Rico, and the Virgin Islands held validated-free status at the end of year.
Nine States, Alabama, Arkansas, Connecticut, Georgia, Hawaii, Illinois, Louisiana, New York, and Virginia were in Stage II. Eleven States, Alaska, Florida, Kansas, Kentucky, Massachusetts, Michigan, New Jersey, North Carolina, Ohio, Oklahoma, and South Carolina were in Stage I, the entry stage. Designated as "no program" States were Indiana, Mississippi, Missouri, Nebraska, New Mexico, Oregon, Tennessee, Texas, and West Virginia. Some of these nine States are taking actions to correct deficiencies and should soon qualify for entry into Stage I of the program.

SLIDE 11

The number of validated herds decreased from 4,688 at the end of FY 1981 to 4,488 at the end of FY 1982.

Identification continues to be the major problem of the swine program. Current methods will permit eradication but the cost in time and money will be greater than if a universally accepted identification method was available. Until one is, program progress will require that we use the identification program we have with maximum effectiveness.
Cattle Brucellosis

State Classifications

- Free
- Class A
- Class B
- Class C

Sept. 30, 1982

Slide No. 1

Brucellosis Eradication

Number of Infected Herds Found
(According to State Classification)

State Classification
- Certified-Free
- Modified Certified
- Noncertified

Thousands

<table>
<thead>
<tr>
<th>Fiscal Year</th>
<th>Certified</th>
<th>Modified</th>
<th>Non-Certified</th>
</tr>
</thead>
<tbody>
<tr>
<td>1972</td>
<td>73</td>
<td>27</td>
<td>4</td>
</tr>
<tr>
<td>1973</td>
<td>77</td>
<td>27</td>
<td>1</td>
</tr>
<tr>
<td>1974</td>
<td>77</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>1975</td>
<td>78</td>
<td>22</td>
<td>0</td>
</tr>
<tr>
<td>1976</td>
<td>78</td>
<td>22</td>
<td>0</td>
</tr>
<tr>
<td>1977</td>
<td>78</td>
<td>22</td>
<td>0</td>
</tr>
<tr>
<td>1978</td>
<td>78</td>
<td>22</td>
<td>0</td>
</tr>
<tr>
<td>1979</td>
<td>78</td>
<td>22</td>
<td>0</td>
</tr>
<tr>
<td>1980</td>
<td>78</td>
<td>22</td>
<td>0</td>
</tr>
<tr>
<td>1981</td>
<td>79</td>
<td>19</td>
<td>0</td>
</tr>
<tr>
<td>1982</td>
<td>82</td>
<td>18</td>
<td>0</td>
</tr>
</tbody>
</table>

Slide No. 2
Brucellosis Eradication

Percent of Total Reactor Herds Found

- Fiscal Year 1982
  Total Herds: 11,597
  - 28.2% States: 1
    Herds: 3,000 (Texas = 3,270)
  - 31.6% States: 3
    Herds: 1,000 < 3,000
      (Florida, Mississippi, and Louisiana = 3,668)
  - 1.5% States: 30
    Herds: < 30
    (Remaining 30 States = 175)
  - 6.8% States: 10
    Herds: 30 < 300
      (Illinois, Virginia, Georgia, Colorado, Iowa, Kansas, Nebraska, South Dakota, California, and Idaho = 787)

- Estimated

Brucellosis Eradication

Milk Ring Test Results (BRT)

Fiscal Year

*Estimated
Brucellosis Eradication

Market Cattle Testing Program

<table>
<thead>
<tr>
<th>Fiscal Year</th>
<th>At Packing Plants</th>
<th>Other Plants</th>
</tr>
</thead>
<tbody>
<tr>
<td>1972</td>
<td>62.2%</td>
<td>37.8%</td>
</tr>
<tr>
<td>1973</td>
<td>63.3%</td>
<td>36.7%</td>
</tr>
<tr>
<td>1974</td>
<td>60.0%</td>
<td>39.4%</td>
</tr>
<tr>
<td>1975</td>
<td>70.0%</td>
<td>30.0%</td>
</tr>
<tr>
<td>1976</td>
<td>66.6%</td>
<td>33.4%</td>
</tr>
<tr>
<td>1977</td>
<td>67.5%</td>
<td>32.5%</td>
</tr>
<tr>
<td>1978</td>
<td>62.2%</td>
<td>37.8%</td>
</tr>
<tr>
<td>1979</td>
<td>54.9%</td>
<td>45.1%</td>
</tr>
<tr>
<td>1980</td>
<td>41.7%</td>
<td>58.3%</td>
</tr>
<tr>
<td>1981</td>
<td>42.0%</td>
<td>58.0%</td>
</tr>
<tr>
<td>*1982</td>
<td>44.9%</td>
<td>55.1%</td>
</tr>
</tbody>
</table>

*Estimated

Millions of Cows Blood Tested

Brucellosis Eradication

Blood Testing: Cattle

Farm or Ranch
MCT

<table>
<thead>
<tr>
<th>Fiscal Year</th>
<th>Thous Reactors Found</th>
</tr>
</thead>
<tbody>
<tr>
<td>1971</td>
<td>116</td>
</tr>
<tr>
<td>1972</td>
<td>158</td>
</tr>
<tr>
<td>1973</td>
<td>190</td>
</tr>
<tr>
<td>1974</td>
<td>250</td>
</tr>
<tr>
<td>1975</td>
<td>238</td>
</tr>
<tr>
<td>1976</td>
<td>241</td>
</tr>
<tr>
<td>1977</td>
<td>197</td>
</tr>
<tr>
<td>1978</td>
<td>195</td>
</tr>
<tr>
<td>1979</td>
<td>183</td>
</tr>
<tr>
<td>1980</td>
<td>174</td>
</tr>
<tr>
<td>*1982</td>
<td></td>
</tr>
</tbody>
</table>

Fiscal Year

*Estimated
Slide No. 7

Brucellosis Eradication
Calves Vaccinated

Slide No. 8

Swine Brucellosis
Animals Blood Tested
Swine Brucellosis

Infection Rate

Percent

0.5

0.4

0.3

0.2

0.1

0


Fiscal Year

Estimated

Swine Brucellosis Program Stages—Sept. 30, 1982
Swine Brucellosis

Validated Herds—Sept. 30, 1982

* Validated
State or Territory

- None
- 1-25
- 26-100
- Over 100

Total Herds 4488*  
*Estimated
A PROJECT TO DETERMINE FEASIBILITY OF UTILIZING CULL BREEDING BOARS AS SENTINEL ANIMALS FOR SWINE BRUCELLOSIS SURVEILLANCE

Granville H. Frye, D.V.M.; Phil Pickerill, D.V.M., Ph.D.; and Irvin T. Rhodes, D.V.M.

For over 20 years regulatory officials have considered the feasibility of testing only herd boars to locate brucella infected herds by slaughter surveillance. Since one of the principle means of transmitting brucellosis in swine is through breeding, the herd boar should be the animal with the greatest probability of reacting if the herd is infected. Limiting slaughter surveillance to boars only would reduce the number of swine tested by over 80 percent and cause a similar reduction in the funds and personnel needed for blood collection and tracing. Both are factors of increasing importance under the current conditions of reduced program resources.

Although it is generally conceded that the boar should be a good "sentinel" animal, considering the epidemiology of brucellosis in swine, no body of data exists to support this conclusion. On the contrary, limited work in the early 1960's casts some doubt on the validity of this assumption.

To resolve this question the Swine Brucellosis Subcommittee of the United States Animal Health Association, at last year's meeting, recommended that APHIS carry out a field trial to determine:

A. If the boar is a good "sentinel" animal for determining the brucellosis status of swine herds.

B. Whether it is possible to identify and trace a high percentage of slaughter boars to their herds of origin under current marketing methods.

APHIS accepted these recommendations and initiated a field trial to carry out the Subcommittee's request. The results of this field trial will be important in future Subcommittee deliberations on changing the current Market Swine Testing program to require the testing of boars only.

The low infection rate in swine made it impractical to combine into a single project an evaluation of the boar's value as a sentinel animal and our ability to trace boars to their herds of origin. Therefore, data on the health status of boars in infected herds is being accumulated from questionnaires completed on each herd found infected in the United States for the duration of the field trial. These questionnaires are retrospective examinations of the infected herds with particular attention being given to the length of time reactor and nonreactor boars have been in the herd, whether there had been recent boar sales, and if such sales were related to breeding problems.

The traceability of boars moving in today's complex marketing channels will be determined by means of a formal field trial which is
currently underway. In this trial buyers, buying stations, and auction markets handling slaughter breeding boars were selected in several midwestern States. Arrangements were made for them to identify the boars purchased with color coded eartags, generally either the metal All Weather bangle tag or the official cattle eartag. The identification devices would be recovered at the time of slaughter to determine percentage retained, average and maximum time between tagging and slaughter, and the ability to trace certain swine to their herds of origin. Swine to be traced would be selected by the Veterinary Services statistician to provide statistically valid results.

The trial was scheduled for completion October 14, 1982, allowing time for the data to be tabulated and analyzed for presentation at this year's USAHA meeting. However, a marked reduction in slaughter volume at the participating boar plant has made it necessary to extend the trial for an indefinite period.

To date usable questionnaires have been received on 15 infected herds (Chart I). Seven of these herds had reactor boars (46.7%) and in eight herds all of the boars were negative (53.3%). Boars reacting to the brucellosis test had been in the herd from 4 to 60 months, the average being 25.4 months. In herds where boars did not react, the boar had been in the herd from 2 to 24 months with, the average being 14 months. However, one of these herds (G-3) with a 50 percent reactor rate in sows was reported to have recently sold a boar with orchitis because of breeding problems.

Not included in this data are three infected herds which had all used the same "community" boar in their breeding programs. This boar was sent to slaughter without being tested for brucellosis so its role in this outbreak could not be verified.

These preliminary results are similar to data assembled from a number of infected herd test charts prior to last year's meeting. However, the information available at this time is not sufficient to draw conclusions on the value of the herd boar as a sentinel animal for brucellosis. The continuing field trial is expected to provide this data during the coming year.
<table>
<thead>
<tr>
<th>Herd</th>
<th>Number Sows Tested</th>
<th>Number of Reactor Sows</th>
<th>Number of Boars Tested</th>
<th>Number of Reactor Boars</th>
<th>Time Reactor Boars In Herd (Months)</th>
<th>Time Negative Boars In Herd (Months)</th>
<th>Supplemental Test Results (In Herd)</th>
</tr>
</thead>
<tbody>
<tr>
<td>G-1</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>18</td>
<td>-</td>
<td>Positive</td>
</tr>
<tr>
<td>G-5</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>36</td>
<td>-</td>
<td>Positive</td>
</tr>
<tr>
<td>G-7</td>
<td>11</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>60</td>
<td>-</td>
<td>Positive</td>
</tr>
<tr>
<td>G-8</td>
<td>8</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>36</td>
<td>-</td>
<td>Unknown</td>
</tr>
<tr>
<td>A-1</td>
<td>9</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td>24</td>
<td>Positive</td>
</tr>
<tr>
<td>M-3</td>
<td>11</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>12</td>
<td>36</td>
<td>Positive</td>
</tr>
<tr>
<td>M-6</td>
<td>381</td>
<td>57</td>
<td>15</td>
<td>1</td>
<td>12</td>
<td>12</td>
<td>Positive</td>
</tr>
<tr>
<td>G-2</td>
<td>40</td>
<td>4</td>
<td>6</td>
<td>0</td>
<td>-</td>
<td>12</td>
<td>Positive</td>
</tr>
<tr>
<td>G-3</td>
<td>10</td>
<td>5</td>
<td>1</td>
<td>0</td>
<td>-</td>
<td>12</td>
<td>Positive</td>
</tr>
<tr>
<td>G-4</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>-</td>
<td>24</td>
<td>Positive</td>
</tr>
<tr>
<td>G-6</td>
<td>88</td>
<td>34</td>
<td>4</td>
<td>0</td>
<td>-</td>
<td>14(1) 2(3)</td>
<td>Positive</td>
</tr>
<tr>
<td>M-1</td>
<td>16</td>
<td>9</td>
<td>1</td>
<td>0</td>
<td>-</td>
<td>12</td>
<td>Positive</td>
</tr>
<tr>
<td>M-2</td>
<td>17</td>
<td>8</td>
<td>1</td>
<td>0</td>
<td>-</td>
<td>12</td>
<td>Positive</td>
</tr>
<tr>
<td>M-4</td>
<td>30</td>
<td>20</td>
<td>1</td>
<td>0</td>
<td>-</td>
<td>24</td>
<td>Positive</td>
</tr>
<tr>
<td>M-5</td>
<td>19</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>-</td>
<td>2</td>
<td>Positive</td>
</tr>
</tbody>
</table>
REPORT OF SWINE BRUCELLOSIS SUBCOMMITTEE

Seven members of the Committee were present, along with 17 guests. Dave Meisinger of the National Pork Producers Council read a letter from Dr. Harry Mussman, Administrator of APHIS to Dr. Van Houweling of NPPC indicating that APHIS has no plans to reduce swine brucellosis program funding in fiscal '83 and that field work is expected to be maintained at or above the present level.

Dr. Granville Frye of APHIS reported that the first year of activity had been completed under the new standards recommended by this Subcommittee. Three states, Arizona, Delaware and Maryland have used the new standards of the program to advance to Stage III, the validated free phase, of the program during the past year. As a result, there are now 22 states, plus Puerto Rico and the Virgin Islands, in the third, validated free stage, 9 in Stage II; 11 in Stage I, and 9 states reported as having no program. Frye reported a reduction in sampling in 1982, a decline in infection rate to .045 in the MST program and a reduction in the number of validated herds. Identification continues to be a major problem of the program, he said. Responding to recommendations from the 1981 meeting of the Subcommittee, with regard to the request that APHIS carry out a field trial to determine (1) if the boar is a good sentinel animal for determining the brucellosis status of swine herds, and (2) whether it is possible to identify and trace a high percentage of slaughter boars to their herds of origin under current marketing methods, Frye offered the following report:

Data on the health status of boars in infected herds is being accumulated from questionnaires completed on each herd found infected in the country for the duration of the field trial, since the low infection rate makes it impractical to combine the two objectives into one trial. The results of those questionnaires to date indicate that in 15 herds in which they have been completed, in 7 the boars were positive and in 8 the boars were negative, obviously not enough data on which to base a conclusion. The study is being continued.

On the ability to identify and trace boars, that study is under way. Dr. Irvin Rhodes reported preliminary data on tests of 18,659 boars. The study was set up in a boar killing plant, but when all the pieces were in place for study, the plant discontinued slaughter of boars. Boar slaughter has since been resumed and the trial is continuing. He reported that of 20 samples selected on a random statistically valid basis, 17 could be traced. Dr. Phil Pickerill of APHIS, reported that a boar identification study using ear tags in Iowa, showed an increasing ability to trace boars slaughtered in an Iowa plant, from 13.5 percent in April of '82 to 44% in September, with about the same percentage in October. This involved a total of 53,772 boars, of which 24,464 originated in Iowa and were used in evaluating identification. These results indicate, he said, that if the tags are applied they will provide a better recovery rate than tattoo identification.
Frye reported that there have been no appeals by states from classifications assigned as a part of implementation of the new standards, so the recommendation of 1981 regarding appeals being routed through the Subcommittee is moot. The map showing status of the states requested by the Subcommittee has been prepared and distributed.

Chairman Doby called for reports from the nine states with no program. They were as follows:

Missouri—not in compliance with identification requirements. Dr. Badger reported there is little enthusiasm for the program in the swine industry; on-again, off-again sampling and testing, along with identification without sampling in the past, have created some bitter feelings. He said he hopes concentration can be placed on boars. There is the attitude that if identification is performed by someone else it will be done. The St. Joseph and Kansas City markets are major obstacles. He said a better, safer system for identifying boars is needed. He stressed that there is little swine brucellosis in Missouri. Tests were completed last year on 90,000 sows and boars in Missouri packing plants and 43,000 in out-of-state plants, with 14 reactors, all card positive-rivanol negative. Testing is being carried out, but identification is deficient.

Dr. Cobb of Georgia, a state with a great deal more swine brucellosis, reported that there are 40 herds under quarantine. Swine testing has led to several cases of human brucellosis and in a couple of occasions, human cases have led to discovery of swine infection. The most effective surveillance system in that state has been the testing of sows going through feeder pig sales, he said. Mississippi—deficiency involves lack of authority for a program. Dr. McCrory reported that the authority has now been obtained and asked that the state’s status be re-examined. He said no positives have been found in the small amount of surveillance being carried out and no infected herds are known. Tennessee—deficiencies include failure to collect MST samples at slaughter and lack of a system to report test results to states of origin. It was reported that no cases have been reported or discovered since 1968. There are 50 to 60 validated herds and testing is carried out at sales with no reactors. It is felt that a slaughter testing program would be for the purpose of satisfying trade barriers rather than eliminating disease. Dr. Frye pointed out that the $1½ million cost of the program could be compared with the $35 million value of exports of offal items to the EEC alone, indicating that satisfying trade requirements is an economically valid objective. It was agreed that the state’s status would be re-examined, since it may be free.

Nebraska—deficiency involves lack of MST sampling and a system for reporting results to other states. Dr. Spar reported no cases have been reported for at least 10 years. MST sampling was discounted by the federal government through what Frye agreed was a misunderstanding. He indicated it would be resumed in Nebraska packing plants. In the Nebraska program, test results are computerized and card positive-
rivanol negative samples are not traced until the computer matches two such test results from the same herd. Indiana—problems involved identification. No infected herds have been found for at least 5 years. If it wasn’t necessary to trace card positive-rivanol negative reactors, the state could be qualified as validated free. Oregon—once was classified as free, might qualify as validated free with a little additional effort.

No reports were received from other states with no program—New Mexico, Texas, and West Virginia.

Chairman Doby called for a discussion of the question of tracing only supplemental positive animals. Suggestions included a priority system requiring tracing of card positive-rivanol positive first, or alternatively, simply discontinuing the tracing of card positives which were negative on supplemental test, or of conducting only rivanol testing the latter was discarded as too costly and slow with the notation that the card test is needed as a screening test. Doby asked if there is any evidence of discovery of infection in a herd as a result of card positive-rivanol negative test results. Only one instance could be cited and in that case a card positive-rivanol positive sample would have resulted in a trace to that herd very soon. The epidemiologist and research scientists in the room agreed that samples negative to the supplemental test do not lead to disease and that not tracing them would not threaten success of the program.

Cobb moved, seconded by Black, that the program require tracing of card positive-rivanol positive test results only, with optional traceback on card positive-rivanol negative results. Motion carried.

Black moved, seconded, that the identification requirements be revised to specify external forms of identification only for boars moving to slaughter, eliminating the slap tattoo as an option for boar identification. It was pointed out that a high percentage, perhaps as many as 80%, of boars are skinned, making tattoos useless. In many cases the allegation is made that boars are tattooed when they may, or may not be, and there is no way to verify whether or not they are. Elimination of the tattoo as an option for boars, along with requirement of an external identification for boars, would greatly enhance enforcement of the boar identification requirement. Motion carried. In view of the lack of enforcement of identification requirements for sows and boars, especially prosecution of violations when they are reported, a resolution was approved for forwarding to the Resolutions Committee called for the Secretary of Agriculture to stimulate the Office of General Counsel to prosecute more vigorously reported violations of these regulations.

Hall moved, seconded by Black, that greater attention be given to reporting test results back to states of origin of animals. It was suggested that if there are problems with states of origin of sows and boars not receiving test results on those animals slaughtered outside the state, complaints be pursued either directly with officials of states where
the animals are slaughtered or through the appropriate federal regional office. Motion carried. The meeting was adjourned by the chairman, Paul Doby of Illinois.

The following members were in attendance: Vice Chairman Neal Black, Minnesota; Dr. John Cobb, Georgia; Dr. Granville H. Frye, Maryland; Dr. Robert E. Hall, Wisconsin; Dr. David Meisinger, Iowa; and Dr. Phillip Pickerill, Iowa.
REPORT OF USAHA BRUCELLOSIS COMMITTEE

Chairman: J.B. Armstrong, Kingsville, TX

Vice Chairman: L.C. Vanderwagen, Sacramento, CA

Committee members present: J.A. Acree, FL; J.W. Bitter, TX; Ramsey Burdett, OR; J.S. Cargile, TX; John Cobb, GA; C.H. Cole, MI; John R. Dahl, ND; M.L. Dierks, NB; Francis Drazek, NY; Tom Cook, D.C.; H.F. Embry, IL; B.H. Espe, OK; W.B. Fairchild, LA; D.E. Flagg, ND; Bill Gallagher, SD; Denny Gentry, NM; G.A. Hall, Ok; R.L. Hartin, OK; J.W. Holcombe, TX; John F. Hudelson, CO; C.N. Jewett, AR; B.G. Johnson, MD; Al W. Keating, IL; T.A. Kincaid, Jr., TX; W.D. Knox, WI; W. E. Lyle, WI; F.E. Mann, Jr., TX; W.F. Martin, NM; H.F. McCrory, MS; Joyce Mitteness, MN; Sid Moore, D.C.; W.G. Nelson, ID; R.E. Nelson, VT; B.F. Newcomb, MT; J.H. Niemi, SD; J.O. Pearce, Jr., FL; W.D. Prichard, OR; W.C. Ray, MD, A.J. Roth, VA; Larry Schaffer, NB; Raymond Schnell, ND; W.E. Stemler, IL; N.R. Swanson, WY; D.U. Walker, VT; Taylor Woods, MO

Comments by Mr. Armstrong—Briefly reviewed the status of brucellosis. 14 states are free of brucellosis, 26 states have 40 or fewer infected herds. Commented on the Texas Animal Health Commission report to the Governor and state legislature detailing the accelerated program now in effect in 32 contiguous counties in east Texas (Class C).

Report on the National Brucellosis Eradication Program—Dr. Billy Johnson. The full status report will be presented to the general session on Thursday. The report shows fewer total infected herds in 1982 than in 1981, fewer dairy herds found infected, a slightly lower MCI reactor rate, and an increase in the number of calves vaccinated.

The Brucellosis Information system is now progressing with equipment in place in 14 states and on order for 5 more. They hope to have the system operational in 18 states by the end of the year and the rest next year. Reduced dose Strain 19 vaccine is now being used in 35 states (about the same as last year) and adult vaccination is gradually being expanded in high incidence states.

The new classification system was placed in effect on May 1, 1982. All signs indicate progress is being made toward our goal of eradication.

The changes to Part 78 are nearing completion and hopefully will be published soon.

The Review Team that met this summer suggested a return to flat rate indemnities. Congress must be notified before indemnity rates are changed. It is expected those rates will be under review for possible changes during the next two months.

The FY 83 budget has been of concern. The administration's budget called for brucellosis appropriations of $59.896 million. The House appropriations bill provided approximately $77 million and the Senate bill
would fund the program at the 82 level of $91 million. APHIS is now operating based on a continuing resolution at the House level of $77 million.

*Report on Florida Program*—Dr. Clarence Campbell. Dr. Campbell reviewed the progress the accelerated program in Florida has made since it was started in October, 1980. The progress showed in the increase in calf vaccinations, reduction in infected herds, and an increase in number of certified free herds (11.4% of the herds in the state are now certified).

92% of the calves moving out of Florida go to 8 states. Under the current UM&R, officially vaccinated calves under 12 months of age may move from herds following a herd plan from any area into any area until Jan. 1, 1983. In view of the importance to Florida producers of exporting calves and the minimal effect they have, Dr. Campbell proposed that the committee recommended that those sections of the UM&R which pertain to the movement of those calf vaccinates be amended by substituting the deadline of Jan. 1, 1983 with the phrase “unless such move is prohibited by the State Veterinarian of the receiving area.” This was acted upon later in the meeting.

*Comparison of Factors Influencing the Control and Eradication of Bovine Brucellosis in California during two ten year periods, 1960-70 and 1970-80*—Dr. Margaret Meyer

Dr. Meyer reviewed the prevalence of brucellosis in California for the two previous decades, including the decrease until 1969 when California became certified free and the increase in 1973.

Adverse weather conditions, the change in importation patterns in the dairy industry, and the rapid turnover in those dairies make it clear that California has been buying brucellosis in the form of serological negative, exposed animals. Dr. Meyer stressed that non-program factors are involved.

Dr. Meyer's complete report will appear in the proceedings.

*Report of the Scientific Advisory Committee*—Dr. Margaret Meyer. The Advisory Committee recommended that the brucellosis committee adopt the following dosage for the reduced dose Strain 19 vaccine. Passed by Committee 11/9.

For calves (4-12 months of age) Optimum vaccination time — 5 months.
Minimum — 3 billion organisms per dose.
Maximum — 10 billion organisms per dose.
Optimum — 5 billion organisms per dose.
For adults (over 12 months of age)
Minimum — 300 million organisms per dose.
Maximum — 1 billion organisms per dose.
Optimum — 500 million organisms per dose.
The container size (multiple or single dose) should be worked out between APHIS and the companies involved.

The veterinarian administering the dose should be accountable for the dose administered.

Valid sampling procedures should be worked out to insure that dosage.

This subject was brought up later.

Dr. Meyer also addressed the subject of testing and regulations for goats with regard to brucellosis. The prevalence in goats is negligible or non-existent, therefore, the advisory committee recommended that test regulations should be left up to the individual states until such time that regulations are necessary.

Regulations New York has enacted to help protect its livestock population from Brucellosis—Dr. Francis Drazek

New York is a Class Free state and to remain that way they have recently instituted new import regulations in an effort to protect their livestock population from possible re-introduction of brucellosis. The import regulations are more strict than the UM&R. Animals from all states must be from a herd tested negative to a 30-day blood test. Cattle from B and C states must meet even more stringent requirements. Cattle not meeting NY state requirements shall be returned to the state of origin, slaughtered or remain under extended quarantine. Dr. Drazek emphasized that these regulations minimize the chances of NY becoming reinfected.

RECOMMENDED CHANGES TO THE UM&R

Dr. Fred McCrory—recommended to clarify the qualifications needed for certifying a herd as free when an entire herd or part of a certified free herd is purchased to start a new herd. (handout) The discussion was tabled until Tuesday. (See motions number 13 to 20 on Pages 6, 7)

Dr. Robert Hartin—Recommended a change concerning the MCI reactor rate. This included discussion on animals that were card positive, but rivanol negative. These animals stay in the MCI rate as reactors until the herd is tested and found negative.

The UM&R states that the MCI reactor rate between B and C states will decrease from 0.3% to 0.2% on January 1, 1983. Some were concerned that this should not be dropped as soon as scheduled.

The other concern about the MCI rate was related to reactors found in a whole herd sell-out. That herd may produce numerous reactors, all of which are counted in the MCI rate.

These recommendations were sent to a sub-committee to report Tuesday. (See motions number 10, 11 & 12 on page 6)

Report of Identification sub-committee—Jack Dahl, Chairman; Dr. M. L. Dierks; Dr. B. F. Newcomb; John Cargile; Dr. John Cobb

1. The Nebraska suggestion to allow multiple test tags. Vote after much discussion—failed to pass.
2. The ID sub-committee recommended approval of the following: That APHIS immediately explore the use of a large, easily readable, plastic bangle tag as an alternate ID method to presently used metal tag. Passed by the committee.

3. NCA proposal requesting that APHIS, USDA improve the official ear tag as to color durability, structural strength and legibility of numbers. Also, proposed an educational program for teaching the proper application of ear tags and tattoos. Passed by committee.

4. ID of spayed heifers—Recommend that the jaw brand with a hot iron of the spade figure (as on a playing card) be designated as the universal identification of a spayed heifer. Passed as amended to say—with an open spade on either or both jaws.

5. Recorded ownership brands—Recommendation that Part 1, CC should include a provision that recorded ownership brands shall be acceptable for interstate movement identification requirements in addition to individual animal registered breed association brand number as provided in Part 1. Motion failed. This subject was brought up again Tuesday. (See motion #26, page 8)

6. Identification of vaccinated heifer calves from infected herds for interstate movement—Recommended that if this class of animals is required to be identified, that vaccinated heifer calves from infected herds with an individual herd plan be allowed to move interstate with the identification of a hole in the left ear at least ¾ inch in diameter in lieu of the S brand. This recommendation was referred to a sub-committee made up of Dr. Cobb, Mr. Dahl and Dr. Acree to report on Tuesday (See motion #9 on page 6 when it was passed by the committee.)

**Uniform brand for spayed heifers**—Dr. W. G. Nelson. Reported on brands used in some western states. The spade figure (as on a playing card) with an open spade on either jaw or both jaws. The discussion resulted in the amendment to Mr. Dahl’s recommendation which passed.

**Report on the 16 points presented by epidemiologists in 1981**—Dr. David Walker. Dr. Acree was the chairman of a sub-committee that recommended at the 1982 mid-year meeting, that these points not be included in the UM&R. That recommendation was approved by the full committee. Other subcommittee members were Dr. Flagg and Dr. Newcomb.

Dr. Walker made the following motion: That Dr. Acree’s report on the 16 points be resubmitted to his sub-committee and that the sub-committee be expanded to include an epidemiologist, an industry representative and a member of the brucellosis advisory committee with the charge that:

1. A determination be made whether or not some of these recommendations are now part of the UM&R and, if so, recommend they remain a part of the UM&R.
2. A determination be made whether some of these recommendations should be incorporated into evaluation of state-federal regulatory performance.

3. A determination be made whether any of these 16 points, on individual merit, should be recommended on the basis of scientific soundness, practicality and flexibility to the livestock industry, for implementation into the UM&R.

The committee passed this and the sub-committee was expanded to include Bill Gallagher, Dr. Bill Pritchard and Dr. Garry Adams. (See page 8).

Report of sub-committee (assigned at mid-year meeting) on proposed changes to the UM&R—Dr. Brian Espe Sub-committee members: Dr. Espe, Dr. William B. Fairchild and Dr. Gregg Nelson.

Suggested amendment of UM&R regarding disposition of suspects at auction markets and subsequent disposition of remaining animals in consignment. Change would delete second half of Part II, 4 which calls for a hold order on such animals.

Discussion was tabled until Tuesday, (See motion #21 on page 7) and passed at that time.

Definition of a Reactor—suggested the deletion of one line to correct MCI reactor inequalities. Discussion tabled until Tuesday, (See motion #22 on page 7) when it was passed by the committee. It now classifies MCI reactors on the same serological test interpretations as a routine reactor.

Definition of a finished-fed heifer—Suggested it be changed to read "Heifers in a quarantined feedlot which have been on feed for a sufficient period of time to achieve the flesh condition desired by the slaughter establishments." The change removes the requirement of a certain number of days on feed. Discussion was tabled until Tuesday. (See motion #23 on page 5) Passed Tuesday.

Report of Educational Advisory Sub-committee—Dr. Clint Jewett.

The sub-committee had the following recommendations:

1. Improve communication with the Office of Management and Budget.

2. Re-evaluate the information officers in each state. A survey conducted by APHIS (Sid Moore) to information officers in each state resulted in only 15 replys from all 50 states.

3. The brucellosis committee should change its approach to be more positive. Say we are 99% free of brucellosis rather than 1% infected.

4. An important question—How do we reach the producer that has no intention of listening to anyone anyway? No answer.

5. The sub-committee noted a problem with relationships between VMO's and practitioners and producers. There is often not sufficient dialogue between technicians and practitioners. These problems vary in different areas.
6. Dr. Walker's sub-committee of several years ago on Standards of Performance should be reviewed by the committee again. Problems in education could be helped by peer group review that was suggested by that sub-committee.

7. Recommend to expand industry involvement in information effort.

SUMMARY OF ACTION TAKEN 11-8-82

MOTION #1—Testing requirements for goats, no longer necessary. Committee passed recommendations dropping such interstate requirements.

MOTION #2—Reduced Strain 19 dose levels suggested by Dr. Meyer—Page 2. (Passed)

MOTION #3—Motion to allow movement of vaccinated heifers from infected herds—as suggested by Dr. Campbell—(Page 2) with approval of receiving state. Motion failed. (See motion #9 on page 6) Reworded and passed 11-9-82 to include ear punch.

MOTION #4—Motion to allow multiple test tags. Motion failed.

MOTION #5—Motion to explore use of plastic bangle tags—Page 3. Motion passed.

MOTION #6—Motion to improve official ear tag—Page 3. Motion passed.

MOTION #7—Motion on identification of spayed heifers—Page 3. Passed as amended.

MOTION #8—Motion on herd brand to be used for identification—Page 3. Motion failed. (See motion #26 on page 8) Later reworded and passed.

MOTION #9—Motion on identification of vaccinated heifers from infected herds with ¾ inch hole in ear. Page 4. Sent to sub-committee (Dahl, Cobb, and Acree) to report Tuesday. (See page 6) (Motion 9).

SECOND SESSION, NOV. 9, 1982

MOTION #2 (page 2 of 11/8 report) was brought up. It was recommended that the dose levels suggested by Dr. Meyer yesterday be adopted. The motion passed.

MOTION #9—Change in UM&R Part II, section M, last sentence. Regarding officially vaccinated calves from an infected herd under herd plan. The motion recommended that these animals be identified with a ¾ inch hole in the left ear. This motion had been sent to a sub-committee (Dahl, Cobb and Acree) yesterday. Motion passed.—see attachment number 1 for exact wording.

This motion also changes part 6, sec. 3E; Part 7, sec. 3E; and Part 8, sec. 3F.

Dr. Vanderwagen addressed the three questions regarding MCI rates
originally brought up on Monday. (See page 3 of first report) (Dr. Hartin's presentation)

MOTION #10—Motion to permit reclassification of MCI reactor: MCI Reactor may be reclassified according to Part II, 1.9 “Reclassification of Reactors” and will be included in the MCI reactor rate according to final classification.” Motion passed.

MOTION #11—Postpone the Jan. 1, 1983, scheduled reduction of the adjusted MCI prevalence rate from .30% to .20% in class B and C states until Jan. 1, 1984, with an annual review by the Brucellosis committee prior to date of change. Motion passed.

MOTION #12—In the definition of MCI prevalence rate include “deleting from the numerator all market cattle reactors from a herd in which all test eligible animals have been eliminated by slaughter.” Motion passed. The motion was amended at Wednesday's session to add: “That herd will be designated as an infected herd and included in the appropriate statistics.”

The following actions refer to certified Free herds discussed yesterday by Dr. McCrory. (See page 3 of first report) Attachment 5)

MOTION #13—Change UM&R Part IV, A. Qualifying Methods, Add 3. Purchase of a Certified Free Herd: (a) No negative herd blood test is required when the cattle remain on the premise. A new certificate will be issued in the owner's name. The anniversary date and the herd number will remain the same. (b) “All or part of a Certified Free Herd purchased and moved directly to a premise without other cattle may qualify without a test. A new Certification number will be issued. The anniversary date of the new herd is established by the test date of the herd of origin or by a new herd test of the purchased cattle.” A negative blood test and a complete epidemiological investigation may be used to resolve a suspicious BRT in qualifying a herd. Motion passed.

MOTION #14—Change UM&R Part IV, B. Qualifying, 8. Movement of cattle into a Certified Brucellosis-Free Herd, Sec. d. Reword to read: “From A, B, or C status areas or states—Test Eligible cattle from herds not under quarantine must be blood tested negative for brucellosis within 30 days prior to the date of being moved. In addition, shipping requirements must be met if the cattle are moved interstate. Animals added to a Certified free herd under this provision shall not receive new herd status for sale purposes until they have passed a 60-120 day post entry retest and have been included in an anniversary herd test.” Motion passed.

MOTION #15—UM&R Part IV, Section B. number 8e. Motion to delete entire paragraph. Motion passed.

MOTION #16—Change UM&R Part IV, Section A. Qualifying methods; number 1. Milk ring test. Initial certification: Change “three” to “four.” “A minimum of four consecutive negative milk ring tests conducted at not less than 90-day intervals, followed by a
negative herd blood test conducted within 90 days after the negative milk ring test.” Motion passed.

MOTION #17—Change UM&R Part IV, Section B. number 3. Testing period, Initial Certification: “10 to 14 months (minimum of 6-months if BRT is used).” Motion changed “6” to “9.” Motion passed.

MOTION #18—Change UM&R Part IV, Section B. number 7, paragraph 2. Motion adds sentence at end of Initial certification paragraph. “If the second qualifying test for a herd undergoing initial certification reveals only one reactor the certification will be delayed until all provisions for release of quarantine have been met, and upon recommendation of an epidemiologist.” Motion passed.

MOTION #19—Part IV, Section B. number 8c.
“From Certified Brucellosis-Free herds of Free Status States or Areas — No test requirements on breeding or dairy cattle originating from Certified Brucellosis-Free Herds or Free Status States or Areas — Animals added to a Certified Brucellosis-Free Herd under this provision shall not receive new herd status for sale purposes until they have passed a 60-120 day postentry retest.” (new language is underlined) Motion passed.

MOTION #20—Part IV, Section B. Add the following: “9. Proof of qualifying as a certified brucellosis free herd.
a. Initially a certificate will be issued.
b. Recertification will be done by a renewal certificate showing certified free herd number, number of animals and owner only.” (All new language) Motion passed.

MOTION #21—By Dr. Brian Espe
Part II, M4—Disposition of suspects at auction markets and subsequent disposition of remaining animals in a consignment. See Attachment number 2 for deletions. Motion passed. Permits free movement of negative animals in the lot.

MOTION #22—By Dr. Espe—Change UM&R Part I,A
Definition of a reactor. Motion to delete one sentence to correct MCI reactor inequalities. See Attachment number 2 for deletion. Motion passed.

MOTION #23—Definition of a finished-fed heifer to read:
“Heifers in a quarantined feedlot which have been on feed for a sufficient period of time to achieve the flesh condition desired by the slaughter establishments.” Motion passed.

Joe Huff from Colorado Serum Co. spoke regarding the reduced dose adopted in MOTION #2. He believes a vaccine can be produced in the limits set by the committee. However, laws must be changed to allow them to produce a vaccine with less than 10 billion organisms. Dr. Atwell was present and assured the committee that APHIS-VS will move as fast as possible to get the regulations changed.

Dr. Peitz said Burroughs-Wellcome Co. has produced 5 trial lots of the
reduced dose vaccine and it looks like they can produce it within those limits. He also said sampling and testing procedures shouldn't be a problem.

Dr. Hartin expressed concern that there may be shortage of vaccine during the transition period to the new vaccine. The committee was assured that this would not be a problem.

_Subcommittee's report on 16 points_ (see page 4 on Monday's report)

Dr. Acree reported that 7 of the 16 points were either included in the UM&R or dealt with satisfactorily by other sub-committees. The points they addressed and made recommendation on follows: (using original numbers)

1. Test all cows entering quarantined feedlots:  
   Recommendation: States should deal with this individually. Added surveillance is good if it can be done.

2. Standard Plate test not to be used as screening test at markets.  
   Recommendation: No disagreement. Do not use.

3. Testing and retesting procedures established for suspects.  
   Recommendation: A sub-committee should be formed to develop a definition for "Recommended Epidemiological Procedures." This could cover suspect herds, contact herds and infected herds.

4. Quarantine of contact herds.  
   Recommendation: Recommend 2nd test on contact (adjacent) herds without holding in quarantine. We do not feel that we have authority to hold a negative tested adjacent herd under quarantine.

5. Establish a deadline for testing.  
   Recommendation: Already covered in UM&R or proposals previously considered, except for re-test of suspect cattle which should be handled either under definition of "Epidemiological procedures" or in the herd plan.

6. Testing of all cattle herds of one owner when on-farm reactor is found. Recommendation: This is dealt with in the herd plan and would be determined by epidemiological investigation. Where more than one herd exists without contact or commingled herds, this should be documented by an owner affidavit included in the herd records.

7. Release herd from quarantine after 10 months instead of 6 months. Recommendation: There is merit to holding herds longer than 120 days. Recommend this whole area be reviewed by the Brucellosis Technical Committee for their recommendations and facts regarding re-occurrence of infection in previously known, infected herds.

8. APHIS rewrite Part II, Definitions D and F (Pages 19-20 of UM&R)  
   Recommendation: No disagreement on handling of herds with Strain 19 isolation. In herds where Brucellosis Strain 19 is not
isolated, recommend that this be spoken to in definitions of "Recommended Epidemiological Procedures."

16. Include in UM&R that standard tube test, BAPA, RIV, CF will be conducted on all market or slaughter reactors with titers of \(+25\) or above on plate test or whose samples are card positive. Recommendation: All available tests be used on slaughter samples. Market samples already covered under requirement for confirmation test. There is an inconsistency in new UM&R in that all supplemental tests are not considered in designating MCI reactors.

MOTION #24—To adopt all the points and recommendations in Dr. Acree's report. Motion passed.

MOTION #25—Because of point 16 above—
Change UM&R Parts V, VI, and VII (definition of reactor)—strike "or without." Found on pages 17, 23, 30 and 38 of UM&R. Motion passed. Requires supplemental tests of MCI samples.

MOTION #26—Amend Part 1, CC "Certificates," by adding the sentence—"Herd brands may be used as identification on certificates for cattle being shipped interstate where brucellosis or other official tests are not required. Motion passed.

Sub-committee on Fraudulent Practices

Dr. Clint Jewett announced the formation of this sub-committee.

Members are:

Dr. Jewett, Chairman
J. S. Cargile
Dr. Frank Mann
Dr. Joe Bitter
Dr. Rhodes
Dr. Dean Flagg

Jack Dahl
J. O. Pearce
Dr. MacKery
Dr. Deisch

The sub-committee on Standards of State and Federal Regulatory Performance submitted its report to the brucellosis committee and this report was approved by the brucellosis committee and made a part of the Brucellosis committee report previously.

MOTION #27—Dr. Jewett moved that the report and its recommendations be implemented by USDA. Motion passed. (See page 5, para 6)

MOTION #28—Dr. Jewett also brought up the following recommendation:

Since screening of cattle is a serious problem in the spread of brucellosis, and veterinarians, technicians, dealers and others have been in-criminated, and this committee consists of delegates representing the AVMA, AABP, NCA, Marketing and regulatory services, this committee recommends that—

1. Guidelines be established that can be used by veterinary examining boards and state associations and veterinarians for implementing plans to stop these fraudulent activities.
2. Statements be released to dealers and markets concerning violations of the UM&R and CFR whenever their agents are involved in illicit activities.

3. The state veterinarian notify examining boards of all violations of accreditations.

4. Investigate and determine the source and use of illicit antigens and cards and increase efforts to determine if any of these sources are from other countries.

5. A committee be named to implement the above outlined points. Motion passed.

The committee agreed to meet Wednesday, November 10th at 9 a.m.

THIRD SESSION — NOVEMBER 10, 1982

Call to order by Vice chairman Dr. Vanderwagen — 9:20 a.m.

It was moved to amend MOTION #12 (second session, page 6) to read: "That herd will be designated infected and included in the appropriate statistics."

The committee reconvened today to consider changes suggested by the state of Louisiana and worded by the sub-committee. Dr. Margaret Meyer pointed out that the Scientific Advisory Committee met with the subcommittee and others and is abstaining from any recommendations because of language differences and the short time they had to review it.

MOTION #29 — The committee moved to table the motion regarding classification standards for the Rivanol test until the next meeting. Motion passed.

MOTION #30 — (See Attachment #3)
Motion was made to accept deletions and additions in R. Whole Herd Vaccination Plan, paragraph 2.

MOTION #31 — Motion was made to change UM&R Part VIII Class C States, Section D — Movement of cattle within or from Class C states for the following purposes. 3. Breeding cattle, section b. (See Attachment #4) Motion passed.

MOTION #32 — It was moved to hold a mid-year meeting next summer. Motion passed.

A sub-committee will be appointed to decide on a date and place.

Meeting was adjourned at 10:30 a.m.

ATTACHMENT #1
Motion #9 — Passed

PROPOSED CHANGES TO UM&R

PART II, SECTION M, LAST SENTENCE:

However, officially vaccinated calves under 12 months of age and steers and spayed heifers of any age in a herd known to be
affected by which is following an approved herd plan may move from any area into any area, provided that the intact vaccinated heifers will be further identified with at least a ¼" diameter hole in the left ear, unless such move is prohibited by the State Veterinarian of the receiving area.

PART VI, SUBSECTION 3e:

PART VII, SUBSECTION 3e:

PART VIII, SUBSECTION 3f:

Official vaccinates under 12 months of age from quarantined herds with an approved written individual herd plan may move without restriction, provided that the intact vaccinated heifers will be further identified with a ¾" diameter hole in the left ear, unless such move is prohibited by the State Veterinarian of the receiving area.

ATTACHMENT #2

Report of Subcommittee on Proposed Changes to the UM&R

Dr. Brian Espe
Dr. William B. Fairchild
Dr. Gregg Nelson

MOTION #21 — Passed

Part II, M4 — Disposition of suspects at auction markets and subsequent disposition of remaining animals in a consignment.

In a single consignment of cattle, which are from a producer's herd of origin, the owner shall either return the vaccinated suspect(s) under quarantine to the herd of origin until the suspect(s) is negative to the card test, declared a stabilized suspect by a designated epidemiologist after subsequent test(s) conducted in not less than 30 days, or classified a reactor on a test; or sell the suspect(s) to a quarantined feedlot, quarantined pasture, or to slaughter, identified with an "S" brand. Card negative cattle in this consignment may return to the herd of origin or move to another premise within the State or interstate with permit.

MOTION #22 — Passed

CHAPTER I — BOVINE BRUCELLOSIS

PART I

A. REACTOR'

Official vaccinates of dairy breeds 20 months of age and over and official vaccinates of beef breeds 24 months of age and over (as evidenced by the presence of the first pair of permanent incisor teeth) or official vaccinates under these ages that are parturient (springers) or post-parturient are classified serologically as reactors when they disclose complete standard plate test (SPT) or standard tube test (STT) agglutination reactions in the blood titer dilution of 1:200 or higher; are
USAHA BRUCELLOSIS

positive to the brucellosis card test where it has been the only test conducted, disclose titers of 25 percent fixation (1 plus) in a dilution of 1:40 or higher on the complement fixation (CF) test; disclose a complete agglutination reaction in the 1:25 dilution or higher on the rivanol test; or are found infected by other diagnostic procedures such as isolation of Brucella abortus.

MOTION #23 — Passed

PART I

FF—Heifers in a quarantined feedlot which have been on feed for a sufficient period of time to achieve the flesh condition desired by the slaughter establishments.

ATTACHMENT #3

R. Whole Herd

Vaccination Plan

2. Herds which are not Certified Brucellosis Free—Persons owning herds at high risk of becoming infected with brucellosis which are not Certified Brucellosis-Free, may also request “whole herd” vaccination. The individual herd plan may then include the use of whole herd vaccination in addition to other recognized procedures for control and elimination of the disease. All eligible calves shall also be calfhood vaccinated and identified. Whole herd vaccination may be initiated only upon written permission of the state and federal officials directly responsible for program activities in the states in which the herd is located. All animals so vaccinated shall be identified in addition to an official eartag by an open-ended “V” hot iron brand on the right jaw, or an official “AV” (adult vaccination) tattoo in the right ear or both. Following vaccination, only the adult vaccinated cattle will be placed under quarantine. Herds shall resume test no later than 6 months following the initial whole herd vaccination (excepted as provided by Part II W. Deadline exceptions) Post vaccinated Card test negative animals, may move without restriction, provided they meet intrastate and interstate requirements, from herds not known to be affected and which were negative at the time of vaccination. The herd will be eligible for release from quarantine with a negative test 120 days or more following vaccination.

ATTACHMENT #4

MOTION #31 — Passed

Part VIII Class C. States

D. Movement of cattle within or from class C states for the following purposes

3. Breeding Cattle

b. Test-eligible nonvaccinated cattle from other nonquarantined herds may move interstate if they have two consecutive negative
tests at least 60 days apart. The second negative test must be
within 30 days prior to movement. The first test is valid for 12
months. If the first test includes all the test eligible animals in
herds that have been intact 120 days or more such animals may
move immediately following the negative herd test. The negative
herd test is valid for 12 months. Animals moving from such
negative herds must be tested negative within 30 days of
movement. A certificate is required from the state of origin and
a permit for entry is required from the receiving state prior to
interstate movement from a farm of origin directly to the farm of
destination. Cattle may be moved interstate from a farm of origin
without a certificate and prior permit for entry directly to a
Specifically Approved Stockyard for the second negative test,
provided State-of-destination requirements are met. A certificate
is required for such test negative cattle subsequently moving
interstate from a Specifically Approved Stockyard. A permit for
entry is required from the receiving State prior to movement
from the stockyards. The cattle must be quarantined with a
retest 45-120 days postmovement.

ATTACHMENT #5
(Motions 13-20, pages 6,7)

Recertification: No change.

Herd retest for quarantine release and to fulfill the provisions required
under Part IV, A, 1 and 2, may be conducted concurrently.

8. Movement of cattle into a Certified Brucellosis-Free Herd
   a. no change
   b. no change
   c. From Certified Brucellosis-Free herds or Free Status States or
      Areas—No test requirements on breeding or dairy cattle
      originating from Certified Brucellosis-Free Herds or Free Status
      States or Areas. Animals added to a Certified Brucellosis-Free
      Herd under this provision shall not receive new herd status for
      sale purposes until they have passed a 60-120 day postentry
      retest. Motion 19
   d. (Motion 14)
   e. (Motion 15)

9. Proof of qualifying as a certified brucellosis free herd.
   a. Initially a certificate will be issued.
   b. Recertification will be done by a renewal certificate showing
      certified free herd number, number of animals and owner only
      (Motion 20)
USAHA BRUCELLOSIS
ATTACHMENT #5
(Motions 13-20, pages 6, 7)
PROPOSED AMENDMENTS
BRUCELLOSIS ERADICATION UNIFORM METHODS AND RULES

Part IV

Certified Brucellosis-Free Herds of Cattle

A. Qualifying Methods A herd may qualify by one or more of the following methods:

1. Milk ring test (BRT)
   Initial certification: A minimum of four consecutive negative milk ring tests conducted at not less than 90-day intervals followed by a negative herd blood test conducted within 90 days after the last negative milk ring test (Motion 16)

   Recertification — No change

2. No change

3. Purchase of a Certified Free Herd
   (a) No negative herd blood test is required when the cattle remain on the premise. A new certificate will be issued in the new owner's name. The anniversary date and the herd number will remain the same.

   (b) Part of a certified free herd purchased and moved to a premise without other cattle may qualify with a test 60-120 days after purchase. A new certificate number will be used. The anniversary date will be established by the test date.

   A negative blood test and a complete epidemiological investigation may be used to resolve a suspicious BRT in qualifying a herd. (Motion 13)

B. Qualifying

1. through 2. — No change.

3. Testing period
   Initial Certification: 10 to 14 months (minimum of 9 months if BRT is used). (Motion 17)
   Recertification — No change.

4. through 6. — No change

7. Herd status, if infection occurs — When one or more reactors are disclosed in a certified herd or in a herd under test for initial certification, it shall be considered affected and the quarantine and retest provisions in Part II, D and E shall apply.

   Initial certification: A herd qualifies for initial Certified Brucellosis-Free Herd status when free of infection, all provisions for release of quarantine have been met, and the additional provisions required under Part IV, A, 1 and 2 have been met. If the
second qualifying test for a herd undergoing initial certification reveals only one reactor the certification will be delayed until all provisions for release of quarantine have been met. (Motion 18)

ATTACHMENT #6

MINUTES

Meeting of the Subcommittee on Swine Brucellosis
of The Committee on Brucellosis
of the
United States Animal Health Association
Sheraton St. Louis Hotel—Room 48
St. Louis, Missouri
October 14, 1981

The following members of the Subcommittee were present: G. H. Frye, Hyattsville, Maryland; Neal Black, South St. Paul, Minnesota; David Meisinger, Des Moines, Iowa; Robert E. Hall, Madison, Wisconsin, and Paul B. Doby, Chairman, Springfield, Illinois. Sarah Hurley, Madison, Wisconsin, represented Willis E. Lyle.

Dr. Phillip Pickerill presented two proposals: (1) identification system for boars utilizing plastic ear tags attached to the ear by a hog ring or other appropriate devices, (2) boars be considered as monitoring animals for the Market Swine Testing (MST) program. Dr. Pickerill's proposals are based on the inefficiency of the slap tattoo system. This is due to the increase in skinning of swine at slaughter rather than scalding and dehairing. He reported on a field trial using the new ear tag system. When a plastic tag was applied with a hog ring, a high percentage of the tags were still attached at slaughter.

The Subcommittee instructed the Chairman to appoint a task force to conduct an indepth study of all aspects of converting the MST program to apply to boar testing only. Such a study would include a field investigation. The task force would be asked to report to the Subcommittee during the 1982 annual meeting of USAHA.

Dr. G. H. Frye discussed a number of problems he has encountered with the new Brucellosis Eradication Uniform Methods and Rules relative to the eradication of swine brucellosis.

The Subcommittee adopted the following motions as a result of Dr. Frye's presentation:

- A separate report be made on the disposition of each card and rivanol positive swine brucellosis reactor.
- Personnel of Veterinary Services, USDA will decide the appropriate classification for respective states as a result of a request from the state for program classification. State officials will be given the opportunity to
appeal to the Subcommittee if they feel the allocated classification is not appropriate.

- Personnel of Veterinary Services, USDA will prepare a map to illustrate each state's classification status in the Swine Brucellosis Eradication Program. This map will be distributed to farm publications.

- States should meet all requirements and prerequisites for the stage they are assigned to in the 3-stage validation program, but the following requirements are mandatory and not to be waived when assigning States to these stages.

(a) All individuals and firms (in the State) responsible for identifying swine under 9 CFR Part 78 are in compliance.

(b) Quarantine and promptly test all suspicious herds to establish status. Depopulation or initiate an active retest program for those determined to be infected.

(c) Tag and slaughter reactors within the established time limits.

(d) Comply with all provisions of the UM&R, Chapter II, that are not dependent upon action by, or in, another State.

(e) Have an active surveillance program.

(f) Have a system for reporting MST results back to States of origin of eligible swine tested in their State.
INTRODUCTION

Theileriosis continues to be a severe disease problem of bovids in many parts of the world. Though traditional cattle rearing in the lesser developed countries is adversely affected by a multitude of factors, it seems clear that in East Africa, East Coast Fever (ECF) has a profound effect not only on the present rather low productivity but also is a major constraint on the improvement and development of a more useful cattle industry.

In East Africa, ECF is now considered to be a complex of diseases which is caused by three transmitted Theileria species. T. parva (parva), which causes the classical form of ECF, is transmitted between cattle by Rhipicephalus appendiculatus. T. parva (lawrencei) is also transmitted by R. appendiculatus but from African buffalo to cattle. Although the biology of this parasite in cattle is different, it is now believed to be the same species as the previous Theileria spp. T. mutans, which is transmitted by Amblyomma spp., is also common in African buffalo and cattle and, though previously thought to be nonpathogenic in cattle, has been shown to cause disease.

The economic impact of ECF is difficult to establish definitively because of a general lack of reliable data. Calf mortality rates of 15% to 30% have been attributed to ECF; lower, and perhaps more realistic estimates, have been put at 1% to 2%. The variation in estimates probably relates to the endemicity of disease in the areas under consideration. In addition to mortality, a chronic theileriosis syndrome causing retardation of growth and lowered productivity is being increasingly recognized. Exotic cattle are commonly imported into tropical countries in attempts to upgrade productivity; within the range of R. appendiculatus, this frequently results in high losses. As a consequence, a policy of total dipping is adopted with the concommitant disadvantages of high expenses, acaricide resistance and potential epidemic instability. Disease control based on immunization against Theileriosis could obviate many of these dangers.

IMMUNIZATION WITH SPOROZOITES

To date, the most successful method of immunization against ECF is the infection and treatment method. Cattle are infected by injection with...
a stablate of sporozoites prepared from triturated infected ticks. Early studies\textsuperscript{10} showed that the severity of clinical response to an inoculum of sporozoites was dependent on the quantum injected. Recipients of certain low doses may recover; however, the reactions varied with individual recipient animals and with different stablate preparations. The proportion of deaths from this regimen was both unpredictable and unacceptable. However, these adverse reactions could be controlled by simultaneous administration of tetracyclines\textsuperscript{11,12}. This method was further refined by using more effective and long acting tetracycline preparations\textsuperscript{9,13}.

Two problems relate to this method of immunization. First, the antibiotic protection can be overwhelmed by a high infective dose, and not all tetracyclines are effective. Second, strain differences of parasite occur which result in breaks during field challenge with clinical signs that are irreversible by tetracycline treatment and generally result in death.

A number of new therapeutic drugs with antitheilerial activity have now been identified\textsuperscript{14,15}. Using an experimental compound \textsuperscript{993c} (Parvaquine\textsuperscript{*}) a successful field immunization trial has recently been completed\textsuperscript{16}. Forty-six improved Boran cattle were immunized with \textit{T. parva} (\textit{parva}) and \textit{T. parva} (\textit{lawrencei}) stablates and \textit{T. mutans} infected blood. All three parasite strains were derived from isolates collected in, or near, the trial area. The cattle were exposed to a very severe natural field challenge with over 1,000 adults and greater numbers of nymphs of \textit{R. appendiculatus} feeding on each animal. Two of the immunized animals died (4\%) whereas 50\% of the challenge control animals succumbed. Under reduced challenge, where reasonable tick control is practiced, it is likely that the immunized cattle would have undergone subclinical reinfections which would have been controlled by their acquired immunity without the need for drug intervention. The breakthrough infections in this trial were all of the \textit{T. parva} (\textit{lawrencei}) type. The cattle in the trail area cohabited with a large population of African buffalo. This first of a series of field trials augurs well for a practical method of immunization.

Consideration of the concept of vaccination with irradiated sporozoites has been renewed and initial studies, reviewed by Irvin and Gill\textsuperscript{17}, indicate some promise. Sporozoite neutralizing antibodies have been identified and shown to have activity \textit{in vitro}\textsuperscript{18} and \textit{in vivo}\textsuperscript{19}. Using adjuvant to stimulate the production of highly avid antibodies this method of immunization might block the initial invasion of the parasite. Whether this method could also be modified to protect against the pathogenic macroschizont stage of the parasite if successful infection of the bovine lymphocyte occurs has yet to be determined.

**IMMUNIZATION WITH MACROSCHIZONT**

The most likely effector immune mechanisms identified to date appear to be cell mediated\textsuperscript{20}. Non-specific killer (NK) cell activity against the

* Mention of a trademark does not indicate endorsement by the USDA
macroschizont infected lymphoblastoid cell has been demonstrated\textsuperscript{21} and also specific cytotoxic lympholysis\textsuperscript{21,22}. These latter effector cells show histocompatibility restriction and only recognize infected autologous cells.

This histocompatibility restriction is extended to the infectivity of \textit{T. parva}-infected lymphoblastoid cells. Morrison, et. al.\textsuperscript{23} showed that $10^2$ autologous cells can infect and immunize but it requires $10^7$ or $10^8$ allogenic cells to achieve the same effect. In contrast, immunization with \textit{T. annulata} can be achieved in allogenic recipients with $10^4$ cells\textsuperscript{24}. The bovine histocompatibility system can be defined by bovine lymphocyte antigens (BoLA) and it has been shown that schizont-infected cells retain their BoLA type\textsuperscript{25}. Recent preliminary experiments\textsuperscript{26} have shown that $10^3$ or $10^5$ BoLA matched donor cells immunized 50\% and 66\% of recipients, respectively.

Experiments are under way in an attempt to immunize BoLA half matched animals. If this can be done, a schizont-infected cell vaccine could be used in herds with single or limited sire background.

Additional experimentation may circumvent the histocompatibility restriction. Cell types other than T-lymphocytes have been reported to be infected with schizonts. In the case of another intracellular protozoan, \textit{Leishmania} spp., the expression of major histocompatibility is deleted from the parasitized host macrophage. In this context, it is of particular interest that \textit{T. parva} (parva) will infect macrophages\textsuperscript{27}.

A combined regimen of immunization with schizont-infected cell lines and sporozoite stabilitates has been reported, and successful control of \textit{T. annulata} has been achieved in this way\textsuperscript{24}. Also, attenuated strains of this parasite have been used for immunization\textsuperscript{24}; however, similar attenuation of \textit{T. parva} (parva) results in reduced immunogenicity\textsuperscript{17}.

Strain differences appear to be a minor problem in \textit{T. annulata} immunization but are of greater importance in ECF infections, particularly when \textit{T. parva} (lawrencei) is part of the endemic situation. Exclusion of the African buffalo from cattle populations will effectively reduce this problem and the recrudescence of disease and the carrier state might be quite insignificant in a solely \textit{T. parva} (parva) endemic area.

A master cross-immunity strain from Uganda has been described\textsuperscript{17} but this has yet to be fully documented. If such strains exist, they will prove invaluable in ECF immunization.

**IMMUNIZATION AGAINST TICK VECTOR**

Acquired resistance of cattle to ticks has been extensively reported by Australian workers\textsuperscript{28,29} and similar resistance has been observed against \textit{R. appendicalatus}\textsuperscript{30}. Moreover, preliminary experiments have indicated that transmission of \textit{T. parva} (parva) in tick resistant cattle is reduced\textsuperscript{30,31}.

Because of the problem of ECF strains, it is unlikely that immunization alone will control ECF. Also, ECF cannot be considered apart from other
THEILERIOSIS

tick-borne diseases and tick toxicosis. However, the encouraging advances in research on acquired resistance to ticks, on immunization and chemotherapy against *Theileria* spp. and the strategic use of acaricides point the way to an integrated control of ticks and tick-borne diseases. By means of such control, the profound constraints imposed by ECF on livestock production in Africa could be alleviated.

REFERENCES

5. Duffus, W. P. H. 1977, IBID.
7. Young, A. S., Leich, B. L. and Newson, R. M., IBID. pp. 60.
27. Moulton, J. E. Personal communication.


31. Cunningham, M. P. Personal communication.
INFECTIONOUS DISEASES OF CATTLE

Chairman: J. B. Young, Austin, TX
Vice Chairman: V. A. Seaton, Ames, IA

A. A. Anderson, MD; H. L. Arnold, WVA; R. P. Azelton, MO; D. E. Bartlett, WI; Joe Bearden, MS; D. L. Brinkmeyer, IA; L. N. Brown, TX; E. A. Carbrey, IA; C. S. Card, PA; Pablo Correa Giron, DF Mexico; R. P. Crawford, TX; G. L. Crenshaw, CA; J. F. Evermann, WA; R. W. Fulton, LA; G. D. Gurss, KS; R. E. Horton, NJ; Jerry Houck, SD; N. W. Kruse, NE; G. Lambert, IA; A. J. Luedke, CO; C. S. McCain, WA; A. W. McClarkin, IA; C. A. Mebus, NY; Joyce Mitteness, MN; M. A. Mixson, AL; B. F. Newcomb, MT; P. A. O'Berry, IA; B. I. Osburn, CA; J. O. Pearce, Jr., FL; M. W. Peterson, CA; L. F. Roth, NE; S. L. Reynolds, TX; J. A. Schmitz, OR; R. D. Schultz, AL; W. L. Sippel, FL; Richard Smith, KS; P. L. Spencer, IL; Dan Suther, CA; N. R. Swanson, WY; D. H. Tice, FL; M. VanderMaaten, IA

The committee met at 1:30 PM on November 10, 1982 at the Radisson Plaza Hotel, Nashville, Tennessee. There were 25 members and guests present.

Dr. G. D. Gurss presided as Acting Chairman in the absence of Dr. J. B. Young, Chairman, and Dr. V. A. Seaton, Co-Chairman.

The following reports were presented:
Subcommittee on Artificial Insemination, Dr. H. J. Bearden, Chairman. The subcommittee on Artificial Insemination has continued to monitor recommendations relative to health standards for bulls used in artificial insemination, but has no recommended changes at this time. It was proposed that the work of the subcommittee be continued.

CALICIVIRUS INFECTION OF CATTLE

Dr. Alvin Smith, Corvallis, Oregon

Dr. Smith reported the isolation of a calicivirus from calves with a history of respiratory problems. The isolate was made from a fecal swab cultured on Vero and bovine turbinate cell cultures. The herd was located in Tillamook County, Oregon that is on the Pacific Coast. The calicivirus caused vesicular lesions in experimentally infected pigs. Dr. Smith stressed the widespread distribution of calicivirus. These viruses have been isolated from marine animals, mammals as well as fish, dogs, cats, pigs and cattle.

Trichomoniasis—Dr. G. D. Gurss, Kansas stated that he had been asked by Mr. Bert Hawkins of Ontario, Oregon to report on a severe problem with trichomoniasis that caused a 35% reduction in the calf crop due to poor conception rate.

Dr. W. L. Sippel, Texas—Trichomoniasis is widespread. A survey of approximately 100 bulls at slaughter was made and 8% infection was detected. He described a treatment which was effective, but difficult to
apply. However, he stated that the conception rate will improve without treatment because a degree of immunity is developed. Unfortunately, the annual addition of heifers to the herd will bring about a return of the problem. The diagnosis is most effectively confirmed by carrying the inoculation medium to the field and inoculating it there.

Dan Suther, California — Northern California has problems with trichomoniasis. Diagnosis was a problem because of the long distance to the nearest laboratory. Treatment and control measures were ineffective. Those examinations were required to confirm the negative status of a bull. From the economic point of view this was not satisfactory.

Dr. D. E. Bartlett, Wisconsin — Trichominoisis is a problem of longstanding. Treatment with effective herd management is required to control it. However, the treatment is so expensive it may be more economical to purchase replacements. The use of artificial insemination will eliminate the trichominonisis problem.

PASTEURELLA HEMOLYTICUM INFECTION

Dr. C. S. Card, Pennsylvania — He reported on a peracute pneumonia due to *P. hemolyticum infection* in which a rapid respiratory rate was observed shortly before sudden death. A necrotic pneumonia was observed at necropsy. Prompt treatment with antibiotics, especially, chloranphenical, was effective. Culture for other etiologic agents was negative. The condition occurred primarily in housed, dairy cattle.

Dr. W. L. Sippel, Texas — He suggested it might be helpful to have a *P. hemolyticum* bacterin available as well as the *P. multocida* bacterin.

Dr. George Lambert, Iowa — Dr. Lambert stated that the commercial production of a good bacterin is very difficult.

*Hemophilus sominus* Infection

Dr. J. A. Schmitz, Oregon — Dr. Schmitz raised a question as to the importance of *Hemophilus* infection as a cause of disease in cattle. His laboratory has not been successful in isolating the organism but has detected serologic titers.

Dr. Bob Jones, Colorado — Dr. Jones corroborated this observation and suggested caution in interpreting serologic results. Nearly 90% of cattle have titers. A significant rise in titers should be obtained with paired, acute-convalescent, serums before confirming infections with *Hemophilus*.

There being no further business, the committee adjourned.
HEBDOMADIS SEROGROUP LEPTOSPIRES IN FLORIDA CATTLE

F.H. White and K.R. Sulzer

The first evidence of bovine infections with leptospires of the Hebdomadis serogroup in the United States was obtained from 1951 to 1954 in Florida, when dairy cattle were observed with symptoms of fever, loss of appetite, reduced milk production, frequently having thickened yellowish, bloody milk, and sometimes aborting (Galton et al., 1956). Serum specimens submitted to the Veterinary Division, Army Medical Service Graduate School, agglutinated antigens of the Hebdomadis serogroup to high titers. The most sensitive reactions were obtained with an antigen of *Leptospira interrogans* serovar *sejroe* (Yager, 1953).

After the serologic incrimination of serovar *sejroe* in bovine leptospirosis, that serovar was added to diagnostic antigen batteries, and many reactors were found in cattle in Florida and elsewhere in the United States. However, all attempts to isolate *sejroe* or other Hebdomadis serogroup members from affected cattle failed.

A milestone in our understanding bovine leptospirosis was the first isolation of *hardjo* in Louisiana by Roth and Galton (1960). This serovar, in the Hebdomadis serogroup, was isolated with great difficulty using the leptospiral cultural media available at that time. Isolations of *hardjo* from cattle by Clark, et al., (1961), Sulzer et al., (1964), and Robertson et al., (1964), confirmed the difficulty of recovering these organisms from infected cattle.

Following the first isolation of *hardjo* from cattle, Alexander and Evans (1962) conducted serologic studies on sera from cattle in ten states, and found reactors to numerous serovars of the Hebdomadis serogroup. Those studies revealed that the most sensitive test antigen for Hebdomadis reactors was serovar *wolffi*; however, cross-agglutinin absorption studies provided presumptive evidence of infections with *hardjo*. There was no evidence that Hebdomadis serogroup titers in bovine sera were due to infections with *sejroe*. After these developments, *hardjo* was added to the leptospiral diagnostic antigen battery in many laboratories, and *hardjo* reactors were commonly detected in cattle sera.

By using an improved medium, a bovine albumin polysorbate 80 medium (Ellinghausen and McCullough, 1965), and gerbil or hamster inoculation, Hanson et al. (1965) isolated *hardjo* from cattle in Illinois, in one case from a cow in a herd where abortions had occurred. However, few isolations of *hardjo* were made from cattle even after the development of the improved medium, perhaps because of the continued common reliance on serologic diagnosis of leptospirosis.

The isolation of another Hebdomadis serogroup member, serovar *szwajizak*, was reported from dairy cattle with leptospiral symptoms in
Oregon by Glosser et al. (1975). Since then, *szwajizak* infected herds have not been reported.

In addition to *hardjo* and *szwajizak* infections in cattle in the United States, serovar *balcanica*, another Hebdomadis serogroup member, was recently isolated from both dairy and beef cattle in Florida (Martone and Kaufmann, 1979; and White, et al., 1982).

Numerous reports of cultural and serologic evidence of Hebdomadis infections in cattle associated with abortions and mastitis, some considered to be *sejroe* infections, were reported from other countries, including Japan (Yamamoto, 1951), Denmark (Borg-Petersen and Fennestad, 1956), Belgium (Van Riel et al., 1957) Israel (Van der Hoeden 1964), Russia (Semenora et al., 1965), Scotland (Michna and Campbell, 1969), and Ireland (Ellis, et al., 1976). It is probable that many of these *sejroe* serologic findings, actually were caused by *hardjo*, as was found in the United States.

Following the reports of *hardjo* infections in cattle in the United States and Canada, *hardjo* infections in cattle were reported from Australia (Sullivan and Stallman, 1969), Argentina (Myers and Jelambi, 1975), England (Orr and Little, 1979), New Zealand (Flint and Liardel, 1980), Columbia (Aycardi et al., 1980), and Ireland (Ellis et al., 1982).

Dairy cattle were a source of *hardjo* infections for dairy farm milkers in New Zealand (Christmas, et al., 1974a and 1974b), Israel (Shenberg et al., 1977), and Florida (White et al., 1981).

**LEPTOSPIRAL INFECTIONS IN DAIRY COWS AND DAIRY WORKERS IN FLORIDA**

Our studies to identify leptospiral infections in Florida cattle began in 1978 when serum specimens from dairy milkers with symptoms compatible with leptospirosis were found to contain antibodies against *hardjo*. The outbreak soon involved 11 of 17 persons employed on 3 dairy farms, totaling over 2,000 cows. It also became evident that *pomona* played a minor role in the infections on one farm. Symptoms of the ill dairy workers included, in order of frequency, fever, malaise, nausea and vomiting, myalgia and coryza, chills, enlarged lymph glands, pharyngitis, anorexia, stiff neck, photophobia, arthralgia, diarrhea, conjunctival suffusion, and skin rashes. The frequency of these symptoms ranged from 100% to 27%.

Blood specimens for culture and serology, were obtained from the ill dairy workers. Follow-up specimens for culture and serology were obtained from some of the patients.

Blood was collected from cattle in the 3 involved dairy herds for serologic studies, and urine was obtained from one herd for culture and hamster inoculation.

All cultures were made in bovine albumin polysorbate medium, using a serial tenfold dilution method (1:50, 1:500, 1:5000) to reduce chances of
HEBDOMADIS SEROGROUP LEPTOSPIRES

bacterial contamination in urine specimens. For serologic studies, the improved microscopic-agglutination test (Cole, et al., 1973) was utilized, incorporating 15 leptospiral serovars.

Reliable clinical information on the three herds was not available, but undocumented histories indicated that mastitis and abortion problems had occurred.

Herd A contained about 1000 Holstein cows that had not been vaccinated against leptospirosis. Sixty-seven blood specimens were collected for serology. Twenty-four urine specimens were cultured directly and 20 were inoculated into hamsters in attempts to isolate leptospires.

Herd B contained about 1000 Holstein cows that, according to the owner, had been vaccinated 6-8 months previously with a bacterin containing both pomona and hardjo organisms. Two-hundred blood specimens were collected for leptospiral serology.

Herd C consisted of about 100 Holstein cows that, according to the owner, had been vaccinated 5 months previously with a bacterin containing serovars pomona, hardjo, and grippotyphosa. Twenty-eight blood specimens were collected for serology.

Results

Dairy farm A. The 4 ill employees on this farm had serum titers for hardjo ranging from 1:200 to 1:6,400. One patient also had a titer of 1:800 to serovar tarassovi. Follow-up serology on 2 of these patients showed an increase of the hardjo titer in one, while one remained at 1:3,200.

Seventy-eight percent of 67 cows tested in Dairy A had serologic titers to hardjo ranging from 1:100 to 1:1,600. Serologic titers to non-Hebdomadis serogroups were absent or at very low levels. Two isolations of hardjo were made from the urine of cows in the herd.

Dairy farm B. Three of the 5 ill employees on this farm had serum titers for hardjo ranging from 1:200 to 1:3,200. A sero-conversion from negative to 1:800 to tarassovi occurred in one patient who also had a hardjo titer. Serovar pomona was isolated from the blood of one of the hardjo reactors, suggesting that a mixed or second infection had occurred. Particularly since the hardjo titer was high at the time the blood specimen was cultured. Serovar pomona was isolated from the blood of another dairy worker at this farm, and the fifth patient had serologic evidence of pomona infection, as well as a sero-conversion with tarassovi from negative to 1:400.

The most prevalent serologic reactions among 200 cows in Dairy B were to hardjo (64%) at titers ranging from 1:100 to 1:3,200. Serologic reactions to non-Hebdomadis serogroups were most prevalent to tarassovi (42%) at titers from 1:100 to 1:800, and to pomona (33%) at titers from 1:100 to 1:3,200.

Dairy farm C. There were only 2 employees on this 100 cow dairy, and both had leptospiral symptoms. One had a serum antibody titer to hardjo
of 1:12,800 at the time his blood was cultured, and the titer subsequently dropped to 1:6,400 in 27 days. Leptospires were not isolated. However, hardjo was isolated from the blood of a serologic negative specimen from the second patient on this farm, and the serum titer converted to 1:800 in 14 days. This represents the first isolation of hardjo from man in the Americas.

Leptospiral reactors were found at serum dilutions of 1:100 or higher in 24 (84%) of the dairy cows tested on farm C. Eleven cows (36%) had titers of 1:100 to 1:800 to pomona. Six reacted to hardjo at titers of 1:100 to 1:1,600, and 6 reacted to grippotyphosa at titers of 1:100 to 1:800.

LEPTOSPIRAL INFECTIONS IN BEEF CATTLE IN FLORIDA (1980)

Recently we have isolated hardjo, balcanica, and pomona from beef cattle at slaughter in Florida (White et al., 1982). In that study, one kidney and a matching blood specimen were collected from 306 cattle at a slaughtering plant during the summer of 1980. The cattle originated from feedlots, livestock markets, and ranches from throughout Florida. The previously described leptospiral cultural and serologic methods were used. Leptospiral vaccination histories were not available.

Results

Leptospires were isolated from 82 (36%) of 226 kidneys that were otherwise uncontaminated. Forty-five of the isolates were serotyped as hardjo, 3 as balcanica, and 3 as pomona. Two isolates could not be identified, and 15 could not be grown to a sufficient density for serotyping. Fourteen isolates were lost during attempts to adapt them to a liquid medium for serotyping.

Leptospiral antibodies were found in sera of 218 (71%) of 306 of the cattle at titers of 1:100 to 1:1,600. Serum titers ranging from 1:100 to 1:1,600 to hardjo were found in 162 (53%) of the cattle. Of 45 matching serum specimens from cattle from which hardjo was isolated, 43 had titers from 1:100 to 1:1,600 to that serovar. Serovar hardjo was isolated from 2 seronegative cows.

The 3 cows from which balcanica was isolated were not examined for balcanica antibodies, but had low titers to hardjo (1:200, 1:100, 1:400).

Serum titers from 1:100 to 1:1,600 to pomona were found in only 28 (9%) of the cattle. The 3 cows from which pomona was isolated had titers to pomona (1:1,600, 1:1,600, 1:800), but also titers to hardjo (1:1,600, 1:800, 1:200). Many of the other pomona reactors also reacted to hardjo at similar dilutions. Only 8 sera were serologically positive to pomona alone. Low serologic titers to tarassovi were found in 14 (4.5%) of the cattle.

LEPTOSPIRAL INFECTIONS IN BEEF CATTLE IN FLORIDA (1981)

The leptospiral cultural and serologic studies of cattle at slaughter in
Florida were continued in 1981, with cattle originating from similar widespread locations.

Results

Leptospires were isolated from 37 (18%) of 210 otherwise uncontaminated kidney cultures. To date, nine of the 37 leptospiral isolates have been identified as hardjo and one as pomona. The serotyping of 27 isolates remains to be completed.

Leptospiral antibodies were found in the serums of 172 (66%) of the cattle with titers ranging from 1:100 to 1:1,600. Serologic titers from 1:100 to 1:1,600 to hardjo were found in 136 (52%) of the cattle. Serovar balcanica was added to the antigen battery for this study, and 105 (40%) of the cattle carried titers to that serovar (1:100 to 1:400). As expected, there were many cross-reactions between hardjo and balcanica.

Serologic titers from 1:100 to 1:1,600 to pomona were found in only 13 (5%) of the cattle. Only 4 cattle were serologically positive to tarassovi in this sampling.

SUMMARY AND COMMENTS

Leptospirosis was diagnosed in 11 of 17 dairy workers on 3 farms in Florida. Serologic evidence incriminated serovar hardjo in 9, and serovar pomona in 2 of the patients. Serovar pomona was isolated from 2, and hardjo from one patient. This was the first reported isolation of hardjo from man in the Americas. Serologic evidence also incriminated hardjo and pomona in the dairy cows, and hardjo was isolated from 2 cows on one farm.

During 1980 and 1981, leptospires were isolated from the kidneys of 121 (28%) of 436 beef cows at slaughter in Florida. Fifty-four of the isolates were identified as hardjo, 4 as pomona, and 3 as serovar balcanica. The additional 60 isolates have not yet been identified. Serologic titers in the cattle were largely to hardjo.

The inadequacy of presently available leptospiral media appears to limit the growth of many isolates in these studies, causing delays in serotyping. Despite this, the currently available media are far superior to those previously used and should be utilized to more adequately identify leptospirosis in animals and man.

The leptospires that remain unidentified, and many isolates that do not grow well enough to identify, may represent new serogroups and serovars of leptospires in cattle populations. Improvement of isolation media appears to be needed.

Serologic reactors to serovar tarassovi in both dairy and beef cattle remain unexplained. It was particularly interesting that tarassovi antibodies were detected in serums of both dairy cows and dairy farm milkers on 2 farms in Florida, and appeared in hamsters that had been inoculated with bovine urine on one farm. Serovar tarassovi infections
are known to occur in cattle elsewhere in the world, and it is possible that these findings may indicate the presence of that serovar in Florida. However, serologic cross reactions to some other serovars cannot be ruled out as a cause of the tarassovi titers. Identification of the poor growing unidentified isolates may answer these questions.

REFERENCES


HEBDOMADIS SEROGROUP LEPTOSPIRES


From the Department of Preventive Medicine, College of Veterinary Medicine, University of Florida, Gainesville, FL 32610 (White), and the Bacterial Immunology Branch, Leptospirosis Laboratory, Centers for Disease Control, Atlanta, Georgia 30333 (Sulzer).
LEPTOSPIRA GRIPPOTYPHOSA INFECTION IN THE DOG
D.N. Tripathy, L.E. Hanson and Jan Davis
College of Veterinary Medicine, University of Illinois,
Urbana, Illinois

Leptospiral infections are common in dogs in the United States. The incidence is, however, quite low in confined dogs and rather high in stray dogs. In most cases the evidence of infection is based upon the serological studies as isolations are often not attempted. *Leptospira canicola* is believed to be the predominant serovar infecting dogs, (Diesh, *et al.* 1976) but reports indicate higher incidence of *icterohemorrhagiae* in some urban populations (Thiermann, 1980). Other less common serovars infecting dogs are *pomona, ballum* and *grippotyphosa*. First isolation of serovar *grippotyphosa* from dogs in the U.S. was recently reported by Cole *et al.* (1982). This report describes the second isolation of serovar *grippotyphosa* in another geographic region from a naturally infected dog in which leptospiral infection was suspected because of typical clinical signs. Although leptospires were observed on initial darkfield microscopic examination of the urine, some difficulties were encountered in isolation of the organisms.

HISTORY

A single urine sample from a 5½ year Irish Setter was submitted on July 7, 1980 for darkfield microscopic examination. The dog was showing signs of hematuria, anorexia, malaise and had a temperature of 105°F. Under the dark field live leptospires were observed in the urine sample and a diagnosis of leptospirosis was made. A serum sample from the dog was not available at that time.

MATERIAL AND METHODS

(a) *Isolation in the media.* Various combinations of bovine albumin polysorbate 80 (BAP 80) media used were: liquid, semisolid, liquid with 5% normal rabbit serum, liquid with sodium pyruvate (100 μg/ml), semisolid with 5% normal rabbit serum and solid BAP 80 agar medium. A total of 30 tubes and two solid agar plates were used for isolation.

(b) *Hamsters.* Each of the two weanling hamsters was inoculated intraperitoneally with 0.5 ml of urine. Both the hamsters were killed 14 days after inoculation. Their sera were tested for antibody by the microscopic agglutination (MA) test and kidneys were cultured in liquid and semisolid media.

(c) *Response of hamsters, gerbils and rabbit to isolated leptospiral culture.* Four hamsters, two gerbils and a New Zealand rabbit were inoculated with the semisolid culture following isolation from the urine. Kidneys and in some cases other tissues of the animals were cultured in the media following death or when sacrificed. Sera were tested for antibody by the MA test.
RESULTS

Viable leptospires (15 to 20 per field) were observed in the dog urine sample examined under the dark field microscope. However, a positive leptospiral isolation was made in only one tube of semisolid medium which had been supplemented with 5% normal rabbit serum. This tube also had fungal contamination. Light confluent growth also occurred in one of the solid agar plates.

Both the weanling hamsters inoculated with the urine of the dog were culturally and serologically negative 14 days after inoculation.

Leptospires were isolated from kidneys of all four hamsters inoculated with the leptospiral culture which had mold contamination. Two hamsters died 6 days after inoculation. Their kidneys were enlarged and hemorrhagic. Histopathologic examination of the liver revealed marked congestion. The kidneys showed congestion, dilated tubules, and mild perivasculitis on microscopic examination. One hamster died 15 days after inoculation. One hamster killed 28 days after inoculation had an MA titer of 1:100 against serovar *grippotyphosa*. Interstitial nephritis and perivasculitis were observed on histopathologic examination of the kidney of this hamster.

Both gerbils inoculated with the culture exhibited signs (depression and dehydration) 16 days after inoculation, and leptospires were isolated from their kidneys.

The adult New Zealand rabbit had an MA titer of 1:10,000 against serovar *grippotyphosa* 15 days after intraperitoneal inoculation with the culture, which rose to 1:100,000, 20 days after inoculation. It also had a titer of 1:1000 to serovar *autumnalis*. The rabbit was sacrificed 22 days after inoculation. Leptospires were not isolated from kidneys, brain, liver and urine of the rabbit.

DISCUSSION

Most reports on the incidence of leptospirosis in dogs and other species are based on the serological studies because of limited attempts towards isolation due to the fastidious nature of some leptospires. In the present case clinical signs were suggestive of leptospirosis and, therefore, the urine sample was submitted for darkfield microscopy. However, a serum sample was not submitted until after recovery in September, 1980 along with a second urine sample. This serum was positive against *grippotyphosa* at 1:1000 with 1:100 titer against *autumnalis* and *icterohemorrhagiae*. The second urine sample was negative culturally, as well as under darkfield microscopy. The isolation in only one out of a total of 30 tubes indicates the fastidious nature of this serovar. With approximately 15 to 20 leptospires per field in the urine, we had anticipated isolations in the different media. Difficulties in isolation of leptospires, especially of the fastidious serovars, have been encountered quite frequently. Poor growth of serovar *grippotyphosa* cultures from kidneys of hamsters and gerbils in liquid BAP 80 medium has been observed.
(Tripathy and Hanson, 1974). Since rabbit serum or sodium pyruvate supplemented BAP 80 medium has been beneficial for growth of fastidious leptospires of hebdomadis group (Tripathy et al., 1980), addition of either rabbit serum or sodium pyruvate to BAP 80 media can be useful during isolation from clinical specimens. Although the hamsters inoculated with the initial urine sample did not develop positive cultural or serologic response, isolations were made from gerbils and hamsters inoculated with the culture. The isolate was pathogenic for hamsters and gerbils.

The culture of the isolate as well as the rabbit serum against it were submitted to the Center for Disease Control, Atlanta, Georgia. The isolate was confirmed as *grippotyphosa* in a two way cross agglutination absorption test.

Acknowledgements: The authors thank Dr. D. Lypton and Dr. J. D. Lykins for submitting various samples, and Beecham Laboratories, White Hall, Illinois for providing funds.

SUMMARY

Serovar *grippotyphosa* was isolated from a naturally infected dog showing clinical manifestation of leptospirosis. The organism was quite fastidious as isolation was only possible in rabbit serum enriched bovine albumin polysorbate 80 medium. This is the second isolation of *grippotyphosa* from a dog, but from a different geographical region in the U.S.

REFERENCES


REPORT OF THE 1982 COMMITTEE ON LEPTOSPIROSIS

Chairman: S. L. Diesch, St. Paul, MN
Vice Chairman: H. C. Ellinghausen, Jr., Ames, IA

C. S. Card, PA; J. J. Cecil, IA; J. R. Cole, Jr., GA; B. J. Edmundson, WA; John Finnell, IL; R. F. Hall, GA; L. E. Hanson, IL; Rube Harrington, Jr., IA; C. M. Hibbs, NM; P. B. Kimsey, CA; R. L. Morter, IN; Carl Norden, NB; L. A. Rosener, MO; H. L. Rubin, FL; J. G. Songer, AZ; A. B. Thiermann, IA; D. N. Tripathy, IL; F. J. Wedam, OR; F. H. White, FL; J. M. Williams, MO

The USAHA Leptospirosis Committee has again reaffirmed last year's resolution on the establishment of a National Animal Leptospirosis Research Reference Center. This year, the committee feels there is an even greater need for such a Reference Center since the Centers for Disease Control, USPHS no longer is authorized to type leptospiral organisms of animal origin. Most typing of leptospiral isolated in North and South America have been conducted by CDC. This year the National Cattlemen's Association members strongly reaffirmed this committee's 1981 resolution and emphasized the critical need for such a Research Reference Center. The American Association of Laboratory Diagnosticians Executive Board endorsed the resolution on November 9, 1982. Support for this resolution will be again brought to the attention of other livestock groups.

A sub-committee, (Dr. Ellinghausen, IA; Dr. Cole, GA; and Dr. Rubin, FL), are preparing monograph III which describes the serological characterization of the microscopic agglutination tests (MAT) and plan to publish this 3rd paper in the 1983 USAHA proceedings. It will emphasize the use of the MAT in cross-agglutination patterns (homologous and heterologous agglutinin absorption) and treatment of sera with chemicals to detect the presence of early antibody.

The continuing occurrence of leptospirosis in man emphasizes the importance of Leptospira as a zoonotic agent. It is important to alert the health professions to the opportunities of leptospiral transmission to humans from livestock, wildlife, and their environments. The disease in humans is often overlooked because of lack of unique symptomatology. Therefore, the association between man and clinical and sub-clinical leptospirosis in animals should be brought to the attention of health professionals.

Current documentation indicates that an extensive distribution of leptospiral serovars exist in Central and South America. These are a potential threat to the U.S. livestock industry. At least 12 serovars of the Hebdomadis serogroup, at present not identified in U.S. livestock, have been isolated from animals and man in Central and South America. Constant surveillance of our livestock through careful diagnostic techniques currently not conducted, is imperative to monitor the prevalent leptospiral serovars in the United States.
Dr. Tripathy of IL presented a summary of a paper on isolation of serovar *grippotyphosa* in an IL dog. Dr. White of FL summarized his paper concerning Hebdomadis serogroup isolated from Florida cattle. These are published in this USAHA Proceedings.

Dr. Kirkbride, SD discussed his findings with direct immunofluorescence (FA) for the diagnosis of leptospirosis from kidneys of aborted fetuses. Eleven of 1300 fetal kidneys submitted to the diagnostic laboratory were leptospiral positive by FA. Limited serological studies have detected antibody reactions to serovar *bratislava* in horses and pigs and *tarassovi* in cattle and humans. Further surveillance is essential to determine the significance and distribution of these serovars.

Drs. Kirkbride and Thiermann, IA reported on their results of experimental ELISA tests as a new diagnostic procedure for leptospirosis. Although the test was found more sensitive, and detects some infected animals responding negative to the agglutination test, it also detected most vaccinated animals. The ELISA test does not differentiate titers resulting from vaccination or natural infection.

Further leptospirosis research in these and other areas are essential. The committee strongly recommends that adequate support be provided to meet these needs, including the development of laboratory diagnostic tests to differentiate vaccination from infection.

1983 GOALS OF THE USAHA LEPTOSPIROSIS COMMITTEE

1. Continued effort in the justification and establishment of a National Animal Leptospirosis Reference Center.

2. Completion of Monograph III entitled Characterization Procedures. This will compliment Monograph I, Laboratory Diagnosis of Leptospirosis of Domestic Animals, and II, Isolation Procedures.


4. Further characterization of the signs and pathology of leptospirosis in all species of animals.

5. Encouragement of practitioners and diagnostic laboratories to selectively isolate and identify leptospires in order to detect emerging serovars.

6. Improvement of existing methods for the control and prevention of leptospirosis.

7. Further documentation of the need for increased support for leptospirosis research, which is presently inadequately supported.
Mastitis is a serious problem in the dairy industry that affects quality of dairy farm life, efficiency of milk production, yield of dairy products and consistency of dairy product quality.

The bad news is that currently a small proportion of dairymen are implementing an effective mastitis prevention program. This results in an unnecessary cost of infected quarters and decreased value of infected cows in addition to the previously listed costs.

The good news is that dairymen with a mastitis prevention program can consistently maintain a herd with low infection levels and produce milk of highest quality. Progress in udder health occurs when dairymen direct most attention on prevention techniques every day of the year. Keep uninfected quarters uninfected. Treatment during lactation will often result in quarters remaining in subclinical situation.

Sell all chronic mastitis cows!! The infected mammary gland can influence new infection in the herd since it is the major reservoir of microorganism casing mastitis. The infected cows produce milk less efficiently and increase the bulk tank somatic cell count, decrease milk casein and milk composition making the milk susceptible to development of off flavors. The manufactured products made from this milk will yield less product and will not be of the high quality and maintain shelf life as nonmastitic milk would have provided.

Establish a scheduled milking equipment service program with a qualified service representative. Proper installed and operating milking equipment is essential to harvest milk efficiently from producing cows.

It is important to organize the milking routine to take advantage of the milk let-down. The following practices are recommended for rapid and complete milking with minimum irritation to the udder. Sanitize the machine and your hands prior to milking.

1. **Proper Stimulation For Maximum Milk Let-Down**
   Use individual paper towels to wash the udder and teats with warm water containing a sanitizer. Change the water frequently, depending on the cleanliness of the cows. In many milking parlors, warm water is piped to each stall and the milker can use his hand to wash the udder. Allow 30 seconds to wash and stimulate each udder. Wipe teats and udder dry.

2. **Check For Abnormal Milk**
   The use of a strip cup at each milking is recommended to check for abnormal milk. Some research has shown that the strip cup should be used before washing the udder. This avoids movement of milk from the
teat cistern which may be high in bacteria into the gland cistern and cause mastitis.

Draw a few streams of milk from each teat into a strip cup to clean the teat opening and check for mastitis and abnormal milk. The preferred method is to close the top of the teat first and squeeze. This prevents bacteria in the teat from being forced into the udder. The first few streams of milk are generally high in bacteria and should be kept out of the milk supply.

3. Attach Milking Machine When Let-Down Occurs

Apply the machine when the teats are firm, indicating milk let-down (approximately one minute after starting udder stimulation.)

4. Machine Adjustment

Adjust the teat cups during milking to insure that the quarters milk out properly.

5. Machine Strip Quickly—Do Not Overmilk

To machine strip apply downward pressure on the claw and massage each quarter in gentle downward motion. Do not squeeze the inflation milk tube to detect milk flow. This will cause vacuum fluctuation at the teat end.

If the cow was properly stimulated for milk let-down and the milking machine attached when let-down occurred, then machine stripping should be done in 15 seconds or less in most cows. Some cows require no machine stripping.

6. Remove The Machine As Soon As Milk Flow Stops

Remove the teat cups by shutting off the vacuum as soon as milk flow stops.

7. Teat Dip—Disinfect Teat Ends

Immediately after teat cups are removed the teats should be dipped with a product proven to be safe and effective. Teat dips have shown a decrease of new infections during lactation of more than 50%.

The majority of infections occur as a result of microorganism entry into the test cistern between milkings. Dipping teats immediately after milking with an effective teat dip destroys most of the microorganisms on the teat end and provides residual protection.

Somatic cell count monitors udder health

Maintain low somatic cell count for Milk quality, yield and minimal mastitis. Accurate somatic cell counting by milk plants and DHIA Milk testing programs can provide a dairyman for the first time a routine monitor concerning udder health status of the dairy herd. Over a period of several years the dairyman can gain the benefits of providing a desirable housing and milking environment. The months when SCC increase can point out when cows are challenged by factors influencing cell count and provide the dairymen and his advisors a signal concerning
opportunities for change so the cows are challenged less next year if the unfavorable conditions are changed.

Mastitis has always been a problem of the dairy industry. It is complex because of the many interacting factors that can allow an organism to enter a sterile quarter of a cow. SCC monitoring of herds and cows can assist dairymen in becoming more aware of the extent of mastitis in his herd and the extent of financial loss occurring due to hidden mastitis as indicated by SCC.

DHI and milk plants can communicate the financial effect of high SCC to lost milk production on the farm and in addition the effect on milk quality and yield of dairy products when the milk plant processes it. DHI now alerts dairymen to consider cows above 200,000 on a monthly test as probably infected and losing milk production.

Studies in 350 Wisconsin herds involving almost 5000 cows find that milk production average milk yield during a lactation was reduced by 400 pounds when cell counts increased from 50,000 to 100,000 for the lactation. Each time the lactation SCC doubled the yield was reduced by another 400 pounds. The study indicated a reduced milk yield of 1600 pounds for cows averaging 800,000 SCC and 1200 pounds when lactation average SCC was 400,000. On a daily basis each doubling means a 1.35 lb. reduction in milk yield. Findings of a research study at Virginia Polytech shows similar trends.

Use of somatic cell count:

About 2 million cows are tested monthly in the 38 states now offering the somatic cell testing program. Forty two percent of all cows on DHIA are routinely being monitored by SCC.

The greatest value of the SCC program may be as an educational tool creating an awareness of the amount of subclinical mastitis in a herd and the significant milk loss when SCC increases. Hopefully dairymen will then consistently apply research proven mastitis control procedures.

— Proper managed milking procedure with functionally adequate milking machines
— Dip teats after milking with effective teat dip.
— Administer promptly a full series of recommended treatments to all clinical cases.
— Dry cow treat with specially formulated, commercially available dry cow antibiotic preparation.
— Cull animals with chronic infection that do not respond to treatment.
— Provide a dry environment for clean cows.

Monthly SCC monitoring program will allow the herdsman to observe changes in udder health status and may gain understanding of management decisions affecting SCC.
One of the keys is keeping the low SCC uninfected cow uninfected. Milk them first or with machines only for uninfected cows (unless you have a backflush system.) Pinpoint the problem cows. The SCC report will point out the chronic infected cow to be culled. It will identify cows over 200,000 to be milked last or with a machine to be used on infected cows.

Consider drying off of infected cows early and dry cow treat them. Dry cow treatment is considerably more effective in removing organism than lactation treatment. Saleable milk is not contaminated with antibiotics. The effect on the lactation herd is the same as culling as it reduces lactation herd infection and spread to the uninfected cow.

Recent U of W research shows that lactation treatment is only about 20-25 percent effective in removing organisms from the infected gland. An exception is if cows are infected with Streptococcus agalactiae then a 90-95 percent success can be expected when treated with 100,000 units penicillin.

Culture of 8-10 cows with SCC over 200,000 that have not recently been treated can provide valuable information concerning the organisms invading previous uninfected quarters. This information can be valuable in making recommendations concerning items to consider for correcting and for lactation and dry cow therapy.

TABLE 1

SOMATIC CELL COUNT AFFECTS ROLLING HERD AVERAGE, MILK.

<table>
<thead>
<tr>
<th>SOMATIC CELL COUNT</th>
<th>DAILY AVERAGE</th>
<th>PERCENT UNDER 400,000</th>
<th>WEIGHTED AVERAGE SOMATIC CELL COUNT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greater Than 20,000</td>
<td>66</td>
<td>89</td>
<td>210,000</td>
</tr>
<tr>
<td>18-20,000</td>
<td>60</td>
<td>81</td>
<td>280,000</td>
</tr>
<tr>
<td>16-18,000</td>
<td>55</td>
<td>79</td>
<td>340,000</td>
</tr>
<tr>
<td>14-16,000</td>
<td>51</td>
<td>76</td>
<td>400,000</td>
</tr>
<tr>
<td>12-14,000</td>
<td>47</td>
<td>70</td>
<td>460,000</td>
</tr>
<tr>
<td>Less Than 12,000</td>
<td>42</td>
<td>68</td>
<td>530,000</td>
</tr>
</tbody>
</table>

1982 Wisconsin DHI
### TABLE 2

**SOMATIC CELL COUNT AFFECTS DAILY HOLSTEIN MILK PRODUCTION**

<table>
<thead>
<tr>
<th>Wisconsin DHI Number Holsteins</th>
<th>Somatic Cell Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>107</td>
<td>210</td>
</tr>
<tr>
<td>94</td>
<td>260</td>
</tr>
<tr>
<td>84</td>
<td>300</td>
</tr>
<tr>
<td>74</td>
<td>330</td>
</tr>
<tr>
<td>64</td>
<td>370</td>
</tr>
<tr>
<td>54</td>
<td>400</td>
</tr>
<tr>
<td>45</td>
<td>440</td>
</tr>
<tr>
<td>35</td>
<td>530</td>
</tr>
<tr>
<td>25</td>
<td>640</td>
</tr>
<tr>
<td>14</td>
<td>900</td>
</tr>
</tbody>
</table>

6/82

**LEND YOUR SUPPORT TO THE NATIONAL MASTITIS COUNCIL**

The National Mastitis Council (NMC) is looking for your active support. Since 1950, the NMC has been coordinating research and educational interests which can help dairymen control the annual 2 billion dollar plus losses and costs caused by bovine mastitis. By bringing together dairymen, processors, extension professionals, veterinarians, researchers, educators, dairy cooperatives and dairy supply and manufacturing interests, a strong and successful base organization has been created to control and solve the problems caused by mastitis.

Because it's going to take a strong concentrated group effort, we need your support and involvement to help expand and accelerate the NMC plan of action. Your membership dues contribute to educational and research efforts, seminars, in-group presentations, administration, on-site training, committee work and the annual meeting. Just to be continually updated on mastitis by NMC is an important part of you or your organization becoming “active” in promoting the national drive to control mastitis.

If you are interested in becoming an active part of our national plan of action to control mastitis, or would like a list of our NMC publications and audio/visual materials, please contact National Mastitis Council

1840 Wilson Blvd.
Arlington, VA 22201
MASTITIS CONTROL: PRESENT TECHNOLOGY AND FUTURE PROSPECTS
J. S. McDonald
National Animal Disease Center
P.O. Box 70
Ames, Iowa 50010

SUMMARY
At the present time, the only proven-effective mastitis control program is the teat dip-dry cow therapy program. The program is effective mainly against the gram-positive cocal forms of mastitis. It seems that a different type of effective mastitis control program may not appear in the near future.

Improvement in mastitis control will result through further reduction in the rate of new intramammary infection, increase in the rate of elimination of existing intramammary infection, and by enhancement of the naturally-occurring protective mechanisms of the cow.

INTRODUCTION
Today and in the future, our mastitis control programs must have a broad spectrum of activity. Control programs aimed at eradication of a specific organism only, e.g., *Streptococcus agalactiae*, should be minimized. Even though several of these specific programs are technically feasible, they cannot be justified on economic grounds. However, there are specific types of serious intramammary infection (IMI), e.g., mycoplasma, that require a special type of eradication program.

A program that remains effective for a long period should not require intensive supervision by specialists. The herd owner, manager and/or milkers should be able to carry out simple, routine procedures that are necessary in a control program.

CURRENT STATUS
At the present time, the only proven-effective, broad-spectrum mastitis control program is the teat dip-dry cow therapy (TD-DCT) program. The program consists of dipping of all teats of all lactating cows in a concentrated disinfectant solution after each milking. In addition, following the last milking of each lactation, all glands of all cows are injected with a long-lasting antibiotic preparation. Treatment of existing infection is most important early in the program while prevention of new
IMI is more important later in the program. The program is primarily aimed at control of gram positive cocci infections. This program may eradicate *S. agalactiae* and *Staphylococcus aureus* from many herds over a 2 or 3 year period.

Some of the advantages of the TD-DCT program include the decreased need for a microbiology laboratory, the apparent absence of a problem with selection of antibiotic resistant strains, and eradication of *S. agalactiae* and *S. aureus*.

There are some disadvantages of the TD-DCT program. A herd problem can appear following removal of easily-eradicated infections followed by the appearance of infections that are more difficult to eradicate, eg., *Streptococcus uberis* and enterococci. Also, the program has little effect on the rate of coliform infection. In addition, the program does not protect glands during milking or during the last half of the dry period when the injected antibiotic has disappeared from the gland. Also, there is no protection of glands before first lactation. During the last 15 years, a change has occurred in the types of microorganisms that cause IMI. In many herds, *S. uberis* and coliforms, mainly *Escherichia coli* and *Klebsiella sp.*, have become common causes of IMI.

It appears that a different type of effective mastitis control program may not appear in the near future. Therefore, the hope in mastitis control in the near future is for the improvement of the existing program. The rest of the paper will address this point with additional comments on control of IMI that is not controlled by the TD-DCT program.

**IMPROVEMENTS IN CURRENT CONTROL PROGRAM**

In the consideration of future prospects in mastitis control, three broad areas will be discussed: 1. Reduction of the rate of new IMI; 2. Increase in the rate of elimination of existing IMI; and 3. Enhancement of the naturally-occurring protective mechanisms of the cow.

**REDUCTION OF THE RATE OF NEW IMI**

Disinfectant teat dipping is the single most important method for killing the microorganisms that contact the teat end. By decreasing microbial exposure of the teat end, a decrease in the rate of new IMI will result. Teat dipping after each milking kills those organisms that are transferred to the teat skin during milking and prevents bacterial colonization of the teat skin and teat canal. By lowering the number of microorganisms on the teat skin, the transfer of organisms by fomites at subsequent milkings is minimized. Currently marketed teat dips are only 50 to 80% efficient in reducing the rate of new IMI. Effective teat dips should reduce the microbial population on the teat skin by over 95%.

There are several ways to increase teat dip efficiency. However, it may not be practical to increase concentration of the active ingredients as this may increase teat skin irritation. By reformulation, increased efficiency may result. For example, iodine teat dips become more ef-
fective when ionization and volatility are increased. Increased adhesion to the teat skin and greater residual activity should increase efficacy. Effective automatic teat dipping or teat spraying would reduce the milker's role in the mastitis control program and assure more uniform application.

Another effective method to decrease exposure of the teat end is to prevent microbial build-up in the environment. The cow's environment should be kept as dry as possible and bedding maintained in a sanitary manner. Proper temperature and relative humidity is necessary. Near 50°F with low humidity and proper ventilation appear to be ideal.

Segregation of the herd into groups of noninfected and infected cows with infected cows being milked last will decrease teat-end exposure to microbes. Segregation should be based upon periodic microbiological examination of milk samples from each gland of each cow in the herd.

Teat injuries should be prevented as they decrease resistance and increase microbial exposure. Cows should have correct size stanchions and free stalls where a soft, dry, and comfortable surface is provided for lying down. There are advantages in removal of dew claws from newborn heifer calves to reduce teat injuries.

Earlier detection and treatment of mastitis and avoidance of the accompanying high microbial counts in secretions from infected glands will decrease exposure. It would be desirable to measure milk somatic cells or electrical conductivity when each cow is milked for comparison with levels at previous milkings. These procedures will permit earlier mastitis detection and may be automated in the near future.

Use of an adequate amount of warm, nonpoluted running water and the bare hand is recommended to wash and stimulate glands. Use of a disinfectant in the udder wash water is of little value in reduction of microbial exposure of the teat ends but regulations require addition of sanitizer for its effect on milk quality. Following washing, allow 30 seconds for dripping. Then dry the udder and teats thoroughly. These procedures will decrease exposure of the mammary gland to transient bacteria, mainly the coliforms. "Squirting" of milk from all glands prior to or following each milking has not been proven to decrease the rate of the IMI.

Currently, the only proven effective method for prevention of new IMI in the dry cow is injection of an antibiotic preparation into each gland. Available preparations are effective for only the first half of the dry period. More effective preparations that will last throughout the dry period should be developed. Injection of irritants like endotoxin into dry glands to increase the somatic cell count and increase the rate of involution may decrease the rate of new IMI. Teat dipping throughout the dry period has not been proven to be effective in decreasing the rate of new IMI.

Differences in milking machine design and operation influence transfer
of microorganisms between cows and from infected to noninfected glands within each cow. Fabrication of flexible milk collection tubes from silicone or other nonporous materials could be advantageous. Back-flushing and/or disinfection of the cluster between cows should decrease fomite transfer from cow to cow. Installation of teat cup shields in the short milk tubes and individual long milk tubes from each gland will further decrease fomite transfer. Operation of the milking machine with minimal vacuum fluctuation and prevention of inflation slippage along with complete relief of vacuum prior to inflation removal will decrease the rate of new IMI.

ELIMINATION OF EXISTING IMI

A second way to improve mastitis control is to increase the rate of elimination of existing IMI. Presently, the most effective way to accomplish this is through the use of dry cow antibiotic therapy. There are some arguments against the use of dry cow antibiotic preparations in all glands of all cows after the last milking of each lactation. Nevertheless, dry cow therapy is the most cost-effective procedure in mastitis control procedures and selective therapy is not economical. Most of the benefit is from increased milk production following elimination of IMI.

There are some efficacious dry cow antibiotic preparations on the market. Few of these products maintain effective levels in the dry gland for more than 30 days and consequently there is little protection against new IMI during the last half of the dry period. There is a need for development of a dry cow antibiotic preparation that will maintain effective levels within the gland throughout the entire dry period, have rapid milkout after parturition, and leave no milk or carcass residues. Many new IMI occur during the 2 weeks before parturition; these should be prevented. Most of the new IMI that occur at this time are caused by \textit{S. uberis} and the coliforms, mainly \textit{E. coli}.

Lactational antibiotic therapy will eradicate many streptococcal IMI but will only eliminate 20 to 50\% of \textit{S. aureus} from infected glands. A big disadvantage of lactational therapy is the cost involved in discarding the antibiotic-adulterated milk following therapy. Compared with dry period therapy, lactational therapy is relatively ineffective, does not eradicate IMI in the majority of cases, and is more costly; therefore lactational therapy should be minimized. Nevertheless, clinical cases of mastitis must be treated early to speed clinical recovery and to allow early sale of a marketable product from the glands. Lactational therapy can be minimized by maintaining the milking system in a properly functioning condition, by proper milking technique, by effective teat dipping and by efficacious dry cow therapy.

An important method of eliminating IMI from a herd is by culling of cows with chronically infected and scarred glands. Culling of cows with infected glands helps to reduce the reservoir of IMI within the herd. However, there is little long-term benefit from culling because of the
contagious nature of IMI and the loss of genetic material in the herd. A small number of IMI are eliminated by the cow without antibiotic therapy.

ENHANCEMENT OF PROTECTIVE MECHANISMS

The third way to improve mastitis control is to increase the cow's protection against new IMI by activating or enhancing her naturally-occurring protective mechanisms. The first line of defense against new IMI is the teat canal. Cows with tightly constricted canals and low maximum milk flow rates are less susceptible to new IMI. However, low maximum milk flow rates are genetically related to decreased milk production so it would not appear desirable to select for low maximum milk flow.

Even though it is not obvious, much research on the teat canal should be carried out. We know little about the anatomy and physiology of the teat canal, about the keratin lining, about epithelial cell turnover rates, epithelial colonization, and reverse flow through the teat canal. A great potential for benefit and success lies with research on the teat canal. High milking vacuum and high inflation massage force will remove most of the keratin from within the teat canal.

There are several growth stimulatory/inhibitory factors in mammary secretion that influence microbial growth. Lactoferrin, especially in dry gland secretion is bacteriostatic to iron-requiring organisms if the citrate concentration remains low. Lactoferrin also influences the function of macrophages, lymphocytes and neutrophils by influencing the characteristics of the cell surface. Cystine increases in dry gland secretion and stimulates the growth of S. agalactiae. The function of the lactoperoxidase/thiocyanate/hydrogen peroxide system is not predictable, especially in the presence of cystine. Function of lysozyme, complement, and xanthine oxidase have not been determined in mammary secretion. The high level of the immunoglobulin IgG, in colostrum appears to have little effect on new IMI.

An increase in the number of neutrophils in mammary gland secretion will offer some protection against new IMI. If the neutrophil count in the secretion is \( >5 \times 10^5 \), some protection is present. Higher neutrophil levels offer more protection. Placement of an intramammary device (IMD) such as a coiled polyethylene loop in the lactiferous sinus will increase the neutrophil content of foremilk and strippings and some protection against new IMI results. However, the IMD should not be inserted into infected glands and its presence decreases milk production. These disadvantages may remove the IMD from consideration in mastitis control.

Phagocytic efficiency in mammary secretion varies from cow to cow and with the stage of lactation. Factors that would promote opsonization, ingestion, and intracellular killing of microorganisms would increase the cow's resistance to new IMI.
One of the important roles of vaccination is to increase phagocytic efficiency by increasing specific and nonspecific opsonization. Systemic vaccination to date has failed to achieve significant levels of protective antibody in bovine mammary secretion. Intramammary immunization has some obvious advantages but its efficacy and the effects on milk production are not clear. Selection of antigens for vaccination is a difficult task; however, a more complete knowledge of the pathogenesis and the role of the bovine immune system in mastitis may offer some clues. Because of the numerous microbial causes of IMI, antigenic specificity, and other disadvantages of vaccination, classic vaccination may not be economical in mastitis control. Nevertheless, in some cases, eg., chronic staphylococcal mastitis, vaccination may increase phagocytic efficiency and decrease the severity of IMI. Also, in acute mastitis, there appears to be a need to decrease the inflammatory response of the cow. In this regard, the role of vaccination is not apparent.

Biological competition by IMI with microbes of low virulence may protect the mammary gland against IMI by virulent microbes. Glands infected with organisms such as the coagulase-negative staphylococci, eg. *Staphylococcus epidermidis*, and *Corynebacterium bovis* appear to have increased resistance to other types of new IMI. This is a unique concept in mastitis control and much research should be done in the area. Practical aspects, such as methods for producing infection and effect on milk production, should be evaluated. These bacteria appear to colonize epithelial surfaces and exclude other microorganisms. Infected glands show increased neutrophil counts and this may be the mechanism of increased resistance.

Genetic selection has largely been ignored in mastitis control because of selection for increased milk production and the lack of selection criteria for mastitis resistance. Today, bulls are being culled if their daughters have high somatic cell counts, as a result of increased non-clinical mastitis, and a high incidence of clinical mastitis. The ultimate development of genetic engineering, embryo transfer, and embryo selection may offer tools for genetic selection for mastitis resistance. We should be looking for some genetic markers which are detectable early in the life of heifers that will indicate mastitis resistance. Perhaps histocompatibility markers, bovine serum albumin levels, β-lactoglobulin phenotypes, various blood types, blood cell and plasma protein polymorphisms, and serum immunoglobulin types and levels are some markers that could be examined.

Future prospects for improved mastitis control procedures may not result from a breakthrough in only one area. Improvement will most likely result from progress in several areas. Hopefully, each improvement will be additive and complementary to progress in the other areas and the result will be considerable improvement in mastitis control in the future.
REFERENCES


REPORT OF THE COMMITTEE ON MASTITIS

Chairman: Dr. Clarence A. Jordan, Morgan Center, VI
Vice Chairman: Dr. John S. McDonald

J. B. Adams, D.C.; R.W. Bennett, GA; Robert Bushnell, CA; Carl Graham, MO; D.E. Jasper, CA; C.N. Jewett, AR; C.A. Kirkbride, SD; W.E. Lyle, WI; J.S. McDonald, IA; W.W. Menz, ID; T.G. Murnane, DF; R.S. Sechrist OH; F.E. Sterner, CO; G.H. Swenson, MI; D.U. Walker, VT; R.F. Weidner, IL; Francis D. Gregerson, CO; Dr. Edward Sterner, CO; Ralph F. Hall, TN; Dr. Thomas G. Murnane, Mexico; Jack H. Hagler, TX; Ted Hickerson, TX; Dr. Don Bosman, WI; Dr. Allen Bringe, WI; Dr. David U. Walker, VT; Richard Sechrist, OH; Dr. Max Crandall, D.C.; Dr. Thelma Njaka, W.Va; Dr. John E. Post, CT; Dr. James P. Quigley, GA; Dr. Robert K. Nelson, GA.

The October 14, 1981, Committee report was read and approved with one minor correction.

The five goals developed last year was reviewed and the following progress reported:

1. Have successfully established a Joint Mastitis Committee made up of representatives of USAHA, National Mastitis Council, A.V.M.A. and American Association of Bovine Practitioners. This joint committee met during the 1982 Annual Meeting of the National Mastitis Council where they agreed to issue and promote a common set of recommendations with regard to the prevention of Antibiotic drug residues in meat and milk.

2. Reviewed the status of various Mastitis programs including:

   (a) Wisconsin—Dr. Don Bosman of their Department of Agriculture reported that their 8 year old State program is both regulatory and service related with herd cell counts of over a million somatic cells triggering the regulatory action. The program works through the dairyman's veterinarian requesting the help of Dr. Bosman and the Wisconsin program to improve udder health, reduce cell counts and improve the dairy operation of that particular herd. The program emphasizes:

       (1) proper sanitary procedures, (2) good milking equipment properly installed and operated, and (3) a total environmental and bacterial evaluation. This program is free to the dairyman.

   (b) Dr. David Walker, Vermont State Veterinarian reported that Vermont is seeking to implement a State program with Somatic cell limits of less than a million perhaps as low as 600,000 in bulk tank milk samples being cause for suggesting to the offending dairyman that he join the State Mastitis Control program. Vermont's program will be tailored after the New York and Nebraska programs with a request for funding to be presented to the legislature this winter.

   (c) Dr. Post of Connecticut stated that in the past they have had a very
active program which may need to be reactivated if Somatic cell count maximums for bulk tank milk are lowered.

(d) Mr. Francis Gregerson, Colorado dairyman and Director of the Mountain Empire Dairy Cooperative outlined the features of their very successful monetary Incentive Milk Quality Program whereby producers, whose milk meets the qualifications, receives 8 cents per c.w.t. extra. 71% of their producers now receive this bonus which is paid out monthly. Their handlers have become very quality conscious as they seek to increase the flavor and keeping qualities of their bottled milk and are happy to pay extra for milk of very high quality.

3. Dr. Max Crandall of the Bureau of Veterinary Medicine and Mr. John Adams of the National Milk Producers Federation reported on the status of FDA's updating of the "Mastitis Product Guidelines." The guidelines will be issued in three parts: (1) Product Labeling, (2) Animal Safety and Effectiveness, and (3) Human Safety with the labeling guidelines soon to be issued.

4. Dr. Jordan reported that he had enlisted the aid of the Pharmaceutical Committee in encouraging the development of new products to combat Mastitis.

5. Dr. Jordan reported that he had been appointed to the National Mastitis Council Board of Directors as USAHA's representative and had been invited to the A.V.M.A.'s Mastitis Committee meeting last February.

It was agreed that the committee should pursue the following goals and objectives:

1. Education of individuals attending USAHA meetings by making available the latest educational information on Mastitis Control.

2. Seek permission to man the National Mastitis Council's display during future USAHA Annual Meetings.

3. Emphasize "Preventative Maintenance" as the best management recommendation to prevent Coliform Mastitis.

4. Challenge State Veterinarians, Extension Veterinarians and Extension Specialists to become more actively involved in Mastitis Control programs, invite them to attend our committee meetings, and the annual meeting of the National Mastitis Council to be held February 21-23, 1983, at the Executive West, Louisville, Kentucky.

5. Encourage more basic research in Mastitis.

6. Invite representatives of the Goat Industry to join our committee.

Richard Sechrist, Executive Secretary of the National D.H.I.A., reported that there are now over two million cows in the D.H.I.A. program that are being screened for Somatic cells. This represents 42% of all cows on test.

The committee reviewed and discussed papers to be presented to the
General Session Friday morning by Doctors John McDonald and Allen Bringe. All agreed that Mastitis Control Programs work better if the handler offers a monetary incentive.

It was voted to recommend to the USDA that they support a resolution asking for an increase in basic Mastitis research. The resolution was sent to the Resolutions Committee of USAHA for approval.

The committee voted to meet again sometime during the 1983 Annual Meeting of the National Mastitis Council with the exact time, place and agenda to be developed by the chairman.

The meeting adjourned at 5:00 p.m.

Respectfully submitted

Clarence Jordan, Chairman
Selenium is one of the nutritionally required trace elements that may exhibit toxicity. Its nutritional and therapeutic effects will be described as they may affect toxicoses induced by the aflatoxins and pyrrolizidine alkaloids from plants, *Crotalaria spectabilis* and *Senecio erraticus*. Selenium, Se, is required in the diet of most animals, including mankind, at levels of 0.1-0.2 μg/g or ppm. Chronic toxicity is induced at 3-5 μg/g and acute toxicity at 10 μg/g.

Selenium is an essential constituent of glutathione peroxidase, GSH.Px, and evidence suggests there is a homeostatic mechanism regulating blood Se levels where dietary intake is adequate to provide this critically important element. The FDA.NRC guidelines suggest levels of 0.1/μg/g for humans, broilers, layers and cattle, 0.2 μg/g for turkey pouls and 0.3 μg/g for swine for best growth and performance. (See table I)

The enzyme GSH.Px is present in various cell types and tissues including erythrocytes, phagocytic leucocytes, e.g., granulocytes, neutrophils, basophils and eosinophils, monocytes and alveolar and peritoneal macrophages, and in the liver, kidneys, lungs and muscles. This enzyme catalyzes reduction reactions utilizing reduced glutathione, GSH, thus protecting cells from peroxidative damage initiated by the metabolites hydrogen peroxide, lipid peroxides and singlet oxygen. Presence of foreign objects, such as bacteria, stimulate phagocytosis, the object is surrounded in the phagolysosome; and peroxide formation increases which causes death of the invading bacteria. GSH.Px present in the animal cells, protects against the peroxides.

Selenium deficiency is associated with liver necrosis which, in turn, predisposes to decreased bile flow and improper absorption of vitamins A, D, E, and K. The pancreas is affected adversely with decreased protein and fat digestion. The deficiency also induces “white muscle” disease in cattle, sheep, poultry and rabbits. Selenium deficiency decreases milk levels further and babies are predisposed to “sudden infant death syndrome” (SIDS). Human milk would contain twice the selenium level of cow’s milk normally; mother’s milk, low in selenium has also been associated with SIDS. (Table 3. Selenium Concentrations).

Vitamin E deficiency has interrelated actions with selenium. Deficiency may predispose to cardiac muscle necrosis, hemolysis and hemorrhage, testicular degeneration, uterine atrophy and abortions, hemorrhagic lesions in the lungs and embryotoxicity or even neonatal toxicity. (Table 4, Selenium-Vitamin E Interrelationships). Thus, both selenium and vitamin E deficiencies may predispose to increased severity of aflatoxicosis.

Aflatoxin has been associated with human primary liver cell cancer in
Aflatoxin B<sub>1</sub> levels in dairy cattle feeds greater than 150 ng/g result in aflatoxin M<sub>1</sub> levels greater than 0.5 ng/g, the established FDA guideline level. Such milk must be destroyed. Because of the importance of providing foods, milk and eggs relatively free of aflatoxin B<sub>1</sub> and M<sub>1</sub>, Federal guideline levels of less than 20 ng/g have been established for all feeds except cotton seed meal, CSM. Since CSM is used in feeds for non-lactating animals at low levels, i.e. about 10-11% of the ration, a 300 ppb level has now been accepted as the guideline level for cottonseed products. Inclusion of CSM in dairy cattle rations would be limited to an aflatoxin B<sub>1</sub> levels <20 ng/g. (Federal Register July 30, 1982.).

Burguera<sup>2</sup>, 1981, compared the toxic effects of aflatoxin B<sub>1</sub>, and monocrotaline, the active principle of <i>Crotalaria spectabilis</i> to determine if there was an additive toxic effect between aflatoxin B<sub>1</sub> and monocrotaline in turkey poults. It was also of interest whether the levels of toxicants administered would result in detectable residues in poult tissues.

One hundred eighty healthy one-day old male turkey poults were assigned at random to 12 treatment groups, 15 per group: Groups I, V and IX received 0.1, 5.0 or 10 ug/g of selenium in the diet (control groups); Groups II, VI and X received 250 ng/g of aflatoxin B<sub>1</sub>, plus 0.1, 5.0 or 10 ug/g dietary selenium, respectively; Groups II, VII and XI received 2500 ug/g <i>Crotalaria spectabilis</i> seeds plus 0.1, 5.0 or 10 ug/g dietary selenium, respectively; Groups IV, VIII and XII received 250 ng/g aflatoxin B<sub>1</sub> and 2500 ug/g <i>Crotalaria spectabilis</i> seeds plus 0.1, 5.0 or 10 ug/g dietary selenium, respectively.

Body and liver weight loss plus pathologic lesions, with lowered values in TP and A, aG, βG, and high values in γG in the groups receiving crotalaria, were induced by monocrotaline toxicity.

The similar or higher values in total proteins, A, aG, βG and liver weights in groups receiving aflatoxin B<sub>1</sub> plus selenium indicated the protective effect of selenium against aflatoxin toxicity; selenium may have stimulated the production of glutathione peroxidase, decreasing the concentration of the peroxide metabolites.

Residues of aflatoxin B<sub>1</sub> and M<sub>1</sub> were found in the kidneys of poults re-
ceiving aflatoxin B₁ also dehydroretronecine, the metabolite of monocrotaline, was detected in livers of poults receiving *Crotalaria spectabilis* seeds.

The favorable results reported by Burguera suggested research be performed to determine the effect of decreasing the concentrations of selenium (Se 0.1, 2, 4 ug/g) and increasing aflatoxin B₁ (AFB₁ 0, 500 ng/g) in the diet for 18 days followed by a recovery period in young turkey poults. Treatment groups were: Group I, 0.1 ug/g Se, 0 ng/g AFB₁; Group II, 2 ug/g Se, 0 ng/g AFB₁; Group III, 4 ug/g Se, 0 ng/g AFB₁; Group IV, 0.1 ug/g Se, 500 ng/g AFB₁; Group V, 2 ug/g Se, 500 ng/g AFB₁; Group VI, 4 ug/g Se, 500 ng/g AFB₁. Goldstein⁸, Dec., 1981.

Aflatoxin B₁ exposure in Groups IV, V and VI caused a depression in body and liver weights; total leucocyte counts were reduced during the AFB₁ exposure period. Serum concentrations of TP, A, α and β globulins, as well as serum complement titers, were progressively depressed during the AFB₁ exposure period.

Aflatoxicosis in Group IV included histopathologic changes in the liver and kidney. Their percentage of lymphocytes was depressed while heterophils increased during the AFB₁ exposure period; serum (serum γ-gamma) G concentrations were increased.

The acute toxic effects resulting from AFB₁ exposure were diminished by the addition of 2 ug/g Se in the diet in Group V. Body and liver weights at the end of the trial were increased in Groups II and V as compared to corresponding Groups I and IV.

The presence of 4 ug/g Se in the diet was determined to be toxic for poults in Groups III and VI. Histopathologic changes were observed in the liver and kidney of poults in Group III. Leucocyte numbers were reduced and hematocrit values increased during the last week of Se exposure in Group III. Toxic effects in Group VI were similar to or more severe than those observed in Group IV.

Recovery from all changes induced by AFB₁ exposure, except for body and liver weights, occurred during the recovery period in Group IV, V and VI receiving added dietary selenium.

Increased hematocrit values or hemoconcentrations occurred in Group IV during the AFB₁ exposure period. Phagocytic activity of heterophils, expressed as percent and mean phagocytosis, was greatly depressed in Group IV, but was only slightly reduced in Groups V and VI. Additions of selenium to the diet afforded increased phagocytosis as a part of the immuno-protective mechanism.

Wozniak, 1980, investigated the immune response in piglets given aflatoxin B₁, at 0.2 mg AFB₁/kg B.W. for 5 days. Several immunological parameters were used, including complete blood hematology, lymphocyte transformation, complement titers, electrophoresis, inoculating with sheep RBC and then antibody titration. Liver and mesenteric lymph node biopsies were taken and serum enzymes including arginase, SGOT and
SGPT were measured. To test for delayed hypersensitivity, *Candida albicans* was injected intradermally. There was a delay in hypersensitivity in piglets in the test group receiving aflatoxin $B_1$.

The preliminary results indicated a decreased immunocompetence in the piglets with aflatoxicosis. Antibody production and WBC numbers were not decreased, yet the *decreased* complement titers indicated that *antibody function* and *phagocytic* capacity were *impaired*. Electrophoretic studies during the trial showed altered A/G ratio, a decrease in a globulins, no change in $\beta$ globulins but an increase in $\gamma$ globulins which supports the complement determination results. The serum enzyme levels for SGPT, SGOT and arginase were increased in the aflatoxin exposed group. There was also a marked increase in bile duct proliferation as well as lymphocytic infiltration in the aflatoxin $B_1$ group.

Araya, like Goeger et al, 1982, described the toxicity of *Senecio erraticus* in sheep. The leaves and seeds contained pyrrolizidine alkaloids in both the fresh and dried plants. These alkaloids have been shown to be toxic for animals and the metabolites are excreted in milk affecting growth and performance of the offspring. Thus, meat and milk may contain residues hazardous for mankind. (Table 2 Some Plants Continuing Pyrrolizidine Alkaloids)

The animals were each fed 200 g. of dried *Senecio* for 60 days. Although no clinical signs were observed, there were indications of hepatotoxicity including increases in glutamic dehydrogenase, bilirubin as detected by the BSP test, as well as megalocytosis, fat and lymphocytic infiltration. Araya and others have found animals consume such contaminated feeds for 15 to 30 days but decrease intake thereafter. Araya reported sheep to be more resistant to *S. erraticus* than in his earlier research with calves.

Research is underway to evaluate the protective effects of selenium and vitamin E in young calves exposed to aflatoxin $B_1$. Dosages of aflatoxin $B_1$ at 1 mg/kg bodyweight given as a single dose or as 5 divided doses, 0.2 mg/kg, have induced aflatoxicoses. Protection was provided by use of selenium alone or in combination with injectable Vitamin E. These results will be published at a later date.
In Foods:

- Fish products: 1 ug/g
- Grains—seleniferous area: 1 ug/g
- Animal meats: 0.2 ug/g

In Serum (ug/ml):

<table>
<thead>
<tr>
<th></th>
<th>Deficient</th>
<th>Adequate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equine</td>
<td>0.05</td>
<td>0.22</td>
</tr>
<tr>
<td>Bovine</td>
<td>0.02</td>
<td>0.19</td>
</tr>
<tr>
<td>Porcine</td>
<td>0.06</td>
<td>0.21</td>
</tr>
<tr>
<td>Ovine</td>
<td>0.02</td>
<td>0.29</td>
</tr>
</tbody>
</table>

SUMMARY

1. Aflatoxin B₁ and the pyrrolizidine alkaloids in *Crotalaria* and *Seneclio* (sp) induce acute hepatotoxicoses with anorexia, decreased weight gains, prolonged prothrombin times with hemorrhage, immunosuppression and death.

2. The hepatotoxicoses result in decreased bile flow, decreased absorption of essential vitamins and altered protein synthesis.

3. Selenium deficient rations, as may occur in the S.E. United States or in other areas of the world should be supplemented to provide at least 0.1 ug/g for broilers, hens, cattle, 0.2 ug/g for turkeys and 0.3 ug/g for swine.

4. Added selenium at 0.5 to 2.0 ug/g stimulated production of glutathione peroxidase reducing the toxicities of peroxides, the metabolites of aflatoxin B₁ and the pyrrolizidine alkaloids.

5. It is suggested that this added GSH.Pₓ would afford protection against 100-500 ng/g AFB₁ in rations.
TABLE 1

INTERPRETATION OF SELENIUM VALUES

<table>
<thead>
<tr>
<th></th>
<th>DEFICIENT</th>
<th>MARGINAL</th>
<th>ADEQUATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equine</td>
<td>Serum</td>
<td>.050</td>
<td>.051 - .139</td>
</tr>
<tr>
<td></td>
<td>Liver</td>
<td>.160</td>
<td>.161 - .299</td>
</tr>
<tr>
<td>Bovine</td>
<td>Serum</td>
<td>.020</td>
<td>.021 - .069</td>
</tr>
<tr>
<td></td>
<td>Liver</td>
<td>.120</td>
<td>.121 - .249</td>
</tr>
<tr>
<td>Porcine</td>
<td>Serum</td>
<td>.060</td>
<td>.061 - .119</td>
</tr>
<tr>
<td></td>
<td>Liver</td>
<td>.120</td>
<td>.121 - .299</td>
</tr>
<tr>
<td>Ovine</td>
<td>Serum</td>
<td>.030</td>
<td>.031 - .079</td>
</tr>
<tr>
<td></td>
<td>Liver</td>
<td>.150</td>
<td>.151 - .249</td>
</tr>
</tbody>
</table>

TABLE 2

Common Names and Distribution in North American of Some Plants Containing Pyrrolizidine Alkaloid

<table>
<thead>
<tr>
<th>Botanical Name</th>
<th>Common Names</th>
<th>Location of Plant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amsinckia intermedia</td>
<td>Fiddleneck</td>
<td>Pacific coast states</td>
</tr>
<tr>
<td></td>
<td>Tarweed</td>
<td></td>
</tr>
<tr>
<td>Crotalaria retusa</td>
<td>Crotalaria</td>
<td>Florida</td>
</tr>
<tr>
<td>Crotalaria sagittalis</td>
<td>Rattlebox</td>
<td>Atlantic &amp; Gulf coast states</td>
</tr>
<tr>
<td>Crotalaria spectabilis</td>
<td></td>
<td>Southeastern states</td>
</tr>
<tr>
<td>Heliotropium europaeum</td>
<td>Heliotrope</td>
<td>Atlantic coast states</td>
</tr>
<tr>
<td>Senecio glabellus</td>
<td>Bitterweed</td>
<td>Florida, Illinois, North Carolina, Texas</td>
</tr>
<tr>
<td>Senecio jacobaea</td>
<td>Tansy ragwort</td>
<td>Pacific coast states</td>
</tr>
<tr>
<td></td>
<td>Stinking willie</td>
<td></td>
</tr>
<tr>
<td>Senecio longilobus</td>
<td>Thread-leaf groundsel</td>
<td>Arizona, Colorado, Nebraska, New Mexico</td>
</tr>
<tr>
<td></td>
<td>Wooly groundsel</td>
<td>Texas, Utah</td>
</tr>
<tr>
<td>Senecio spartioides</td>
<td>Broom groundsel</td>
<td>Arizona, Colorado, Nebraska, Texas, Wyoming</td>
</tr>
<tr>
<td>Senecio vulgaris</td>
<td>Common groundsel</td>
<td>Pacific coast states</td>
</tr>
<tr>
<td>Symphytum sp.</td>
<td>Comfrey</td>
<td>Widely distributed</td>
</tr>
</tbody>
</table>
TABLE 3
Selenium concentration in U.S. Swine Feeds.\(^1\)

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Origin</th>
<th>No. of samples</th>
<th>Range (ppm)</th>
<th>Mean (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn</td>
<td>Ill.</td>
<td>31</td>
<td>0.02-0.15</td>
<td>0.05</td>
</tr>
<tr>
<td>Corn</td>
<td>Ind.</td>
<td>17</td>
<td>0.01-0.15</td>
<td>0.04</td>
</tr>
<tr>
<td>Corn</td>
<td>Iowa</td>
<td>25</td>
<td>0.02-0.16</td>
<td>0.05</td>
</tr>
<tr>
<td>Corn</td>
<td>Kan.</td>
<td>1</td>
<td>0.99</td>
<td></td>
</tr>
<tr>
<td>Corn</td>
<td>Mich.</td>
<td>17</td>
<td>0.01-0.09</td>
<td>0.03</td>
</tr>
<tr>
<td>Corn</td>
<td>Minn.</td>
<td>23</td>
<td>0.02-0.19</td>
<td>0.09</td>
</tr>
<tr>
<td>Corn</td>
<td>Mo.</td>
<td>4</td>
<td>0.02-0.09</td>
<td>0.05</td>
</tr>
<tr>
<td>Corn</td>
<td>Nebr.</td>
<td>6</td>
<td>0.04-0.19</td>
<td>0.05</td>
</tr>
<tr>
<td>Corn</td>
<td>N.Y.</td>
<td>1</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>Corn</td>
<td>N. Dak.</td>
<td>5</td>
<td>0.09-0.26</td>
<td>0.19</td>
</tr>
<tr>
<td>Corn</td>
<td>Ohio &amp; Ind.</td>
<td>5</td>
<td>0.09-0.15</td>
<td>0.09</td>
</tr>
<tr>
<td>Corn</td>
<td>S. Dak.</td>
<td>9</td>
<td>0.11-2.03</td>
<td>0.40</td>
</tr>
<tr>
<td>Corn</td>
<td>Wisc.</td>
<td>5</td>
<td>0.02-0.13</td>
<td>0.04</td>
</tr>
<tr>
<td>Meat meal</td>
<td>Iowa</td>
<td>1</td>
<td>0.84</td>
<td></td>
</tr>
<tr>
<td>Meat meal</td>
<td>Ohio</td>
<td>2</td>
<td>0.13-0.24</td>
<td>0.18</td>
</tr>
<tr>
<td>SBM (44)</td>
<td>Ill.</td>
<td>2</td>
<td>0.20-0.21</td>
<td>0.20</td>
</tr>
<tr>
<td>SBM (44)</td>
<td>Iowa</td>
<td>1</td>
<td>1.04</td>
<td></td>
</tr>
<tr>
<td>SBM (44)</td>
<td>Ohio</td>
<td>4</td>
<td>0.05-0.13</td>
<td>0.10</td>
</tr>
<tr>
<td>Limestone</td>
<td>Ohio</td>
<td>1</td>
<td>0.09</td>
<td></td>
</tr>
</tbody>
</table>

1. From information presented by Ullrey (1974).
**TABLE 4**

Selenium-vitamin E interrelationships.  

<table>
<thead>
<tr>
<th>Condition</th>
<th>Animal</th>
<th>Prevented by Vitamin E</th>
<th>Selenium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reproductive failure:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Embryonic degeneration</td>
<td>Female, Hen,</td>
<td>Turkey Yes</td>
<td>No 3</td>
</tr>
<tr>
<td></td>
<td>Ewe No 2</td>
<td></td>
<td>Yes 3</td>
</tr>
<tr>
<td>Sterility</td>
<td>Male: Dog</td>
<td>Cock, Pig Yes</td>
<td>No</td>
</tr>
<tr>
<td>Liver, blood, brain &amp; capillaries:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver necrosis</td>
<td>Pig Yes</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>Erythrocyte destruction</td>
<td>Chick Yes</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Blood protein loss</td>
<td>Chick, Turkey Yes</td>
<td></td>
<td>Yes 3</td>
</tr>
<tr>
<td>Encephalomalacia</td>
<td>Chick Yes</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Exudative diathesis</td>
<td>Chick, Turkey Yes</td>
<td></td>
<td>Yes 3</td>
</tr>
<tr>
<td>Steatitis</td>
<td>Mink, Chick, Pig Yes</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>Nutritional myopathies:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nutritional muscular dystrophy</td>
<td>Duck, Chick</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Turkey Yes</td>
<td></td>
<td>Partially</td>
</tr>
<tr>
<td>Stiff lamb</td>
<td>Lamb, Kid No 2</td>
<td></td>
<td>Yes 3</td>
</tr>
<tr>
<td>White muscle disease</td>
<td>Calf, Sheep No 2</td>
<td></td>
<td>Yes 3</td>
</tr>
<tr>
<td>Myopathy of gizzard &amp; Heart</td>
<td>Turkey poult No 2</td>
<td></td>
<td>Yes 3</td>
</tr>
</tbody>
</table>

3. When added to diets containing vitamin E.
EDDS AND BORTELL

REFERENCES


CURRENT STATUS OF TOXICOKINETICS AND RESIDUE DETECTION OF TRICHOTHECENE MYCOTOXINS IN SWINE, CATTLE AND FEEDSTUFFS

Beasley, V. R., Swanson, S. P., Reynolds, R. D., Coppock, R. W., Corley, R. A., Cote, L. M., and Buck, W. B.

There are at least fifty trichothecene mycotoxins, however most of these are the product of laboratory isolations from *Fusarium* species and have not been detected in field samples. These mycotoxins are most often encountered in grains in temperate regions, when high moisture and cool temperatures prevail especially when harvests are delayed or grains are improperly dried. Three members of this group which have been incriminated in significant illness in livestock are T-2 toxin, diacetoxyscirpenol (DAS) and deoxynivalenol. The latter compound has also been called vomitoxin and less often Rd toxin. Herein we consider the likelihood of encountering these mycotoxins in feeds and animal tissues in order that the diagnostician may logically assess the probability of these mycotoxins being present in feed samples or animal specimens.

The trichothecene mycotoxins have dermonecrotic effects, cause decreased feed consumption, may cause vomition or diarrhea, may depress resistance to infectious disease and may affect coagulation of blood. T-2 toxin was apparently responsible for the widespread outbreak of the syndrome known as alimentary toxic aleukia in humans in the Soviet Union in the 1940's. Hsu et al reported on a hemorrhagic bowel syndrome in dairy cattle associated with the presence of 2 ppm T-2 toxin in corn. However, similar effects have not been associated with the administration of T-2 toxin except in higher doses or by parenteral injection. Recently, however, Osweiler et al showed that, at sufficiently high doses, T-2 toxin administered orally to cattle may cause inhibition of clotting function and diminution of several immunologic parameters.

Diacetoxyscirpenol has been similarly implicated in hemorrhagic gastrointestinal disorders of swine and cattle. Finally, deoxynivalenol has been repeatedly incriminated in feed refusal, vomition and failure to gain in swine.

This paper is based upon 3 areas of endeavor: first of these is our toxicokinetic research on these mycotoxins in swine and cattle. Second is a survey of the occurrence of these compounds based upon laboratory submissions of feedstuffs. Finally, we have the preliminary results of a feeding study of a ration formulated with corn naturally contaminated with deoxynivalenol in weanling swine.

**T-2 TOXIN**

In toxicokinetic studies, T-2 toxin was administered to swine at 0.3, 0.6,
and 1.2 mg/kg intravascularly, at 2.4 mg/kg orally and at 2.4 and 4.8 mg/kg dermally. The 1.2 mg/kg dose comprises an approximate LD₉₀ dose. T-2 toxin was eliminated according to a two compartment open model with an "elimination phase" half-life of from 11 to 15 minutes for the 3 intravascular doses. Therefore, in spite of the fact that a potentially lethal (1.2 mg/kg) intravascular dose was administered, no T-2 toxin could be detected in plasma by two hours after administration. The quantities of T-2 toxin in urine after intravascular administration accounted for less than 0.1% of the dose in either species. Previous studies using radiolabelled T-2 toxin have suggested that significant portions of metabolites are excreted in the urine but that a greater portion of metabolites of T-2 toxin leave in the feces.

When swine were euthanized at 1, 2, 3, and 4 hours after administration of intravascular doses of T-2 toxin the highest concentration was present in lymphoid tissues (spleen and mesenteric lymph nodes) however all tissues had virtually eliminated the parent compound by 4 hours post-dosing. No T-2 toxin was detected in the liver, even at 1 hour. Animals not euthanized survived at least until 7 hours post-dosing, and no residues were detected in tissues of swine or calves that died or which were euthanized at 24 hours. Thus the syndrome leading to lethality progresses in spite of major degradation of the parent compound.

After topical administration of T-2 toxin, no systemic effects were observed even at 4.8 mg/kg. As expected, inflammatory and degenerative changes in the skin occurred in the area in which the toxin was administered. At a single oral dosage of 2.4 mg/kg in two swine, no parent T-2 toxin was present in plasma or urine at any time nor in tissues at times of death, 18 and 19 hours post-dosing. Large concentrations were present in stomach contents, but less than one tenth this concentration was present in other portions of gastrointestinal tract.

The plasma disappearance of T-2 toxin in cattle was similar to that in swine following a two compartment open model. Calves receiving 0.6 and 1.2 mg/kg intravenously had "elimination phase" half-lives of approximately 17 and 18 minutes respectively. Other calves received single oral doses of 0.6, 1.2, 2.4, and 3.6 mg/kg. In spite of the severe clinical manifestations at the higher doses, including weakness, somnolence, cold mucous membranes and finally diarrhea, these animals all survived and none ever had detectable T-2 toxin in plasma or urine at a limit of detection of 40 ppb. No T-2 toxin was present in any tissue after the death of one of the 1.2 mg/kg IV dosed calves at 8 hours post-dosing, nor in any oral or intravenously dosed animals euthanized at 24 hours after administration.

**DIACETOXYSCIRPENOL**

Diacetoxyscirpenol (DAS) disappears from plasma at approximately the same rate as T-2 toxin when doses of 0.1 and 0.5 mg/kg are administered intravenously to swine. However, tissue levels persist
slightly longer than for T-2 toxin with spleen, the tissue which had the highest amount containing 30 to 130 ppb of DAS when animals receiving the 0.5 mg/kg dose were euthanized at 8 hours post-dosing. Slightly more DAS was excreted in urine than in the case of T-2 toxin but urinary excretion of the intact compound is not a major route of detoxification.

DEOXYNIVALENOL

Of the three mycotoxins T-2, DAS, and deoxynivalenol, only the latter can be described as prevalent in the Midwest United States. Vesonder and Hesseltine reviewed the literature in 1980 and cited 18 scientific reports of deoxynivalenol contamination at levels of 0.1 to 40 ppm, 5 reports of T-2 toxin at 0.02 to 25 ppm, 3 reports of DAS at 0.5 to 32 ppm and 2 of nivalenol, only one of which was quantitative at 4.8 ppm. Although deoxynivalenol is considerably less toxic than T-2 toxin or DAS, it is so much more prevalent that it is of greatest agricultural concern at the present time.

Deoxynivalenol is produced primarily by *Fusarium roseum* which is, depending upon the classification system, also referred to as *F. graminearum* var. *roseum*, *F. equiseti*, *F. scirpi*, *F. avenaceum*, *F. culmorum* or *Gibberella zeae*. This organism is, perhaps, better known for its ability to produce zearalenone, an estrogen-like mycotoxin. Deoxynivalenol has been reported in complete feeds, corn, wheat, oats and barley. The toxin is produced as the result of field infection but in grains improperly dried, stored, or shipped (as for analysis), the production of toxin may persist.

The minimal oral emetic dose of deoxynivalenol in swine has been reported as 0.1-0.2 mg/kg. In other studies, 3.6 ppm of purified deoxynivalenol added to swine feed resulted in a 20% decrease in feed consumption and 40 ppm in a 90% decrease. Naturally contaminated feed was far less palatable than feed to which purified toxin was added suggesting other effects of the fungal agent on palatability or appetite.

Several outbreaks of feed refusal, poor growth and vomition in swine have been attributed to deoxynivalenol. For example, in Southern Austria, 60 samples of corn were analyzed. Fifty-six of the 60 were from swine farms affected with feed refusal and all these were positive for deoxynivalenol at from 1 to 20 ppm. No T-2 toxin and no DAS were detected in any sample. Of 4 control corn samples analyzed, all were negative for all 3 mycotoxins.

In another study, 4 analyses of corn were described as a result of feed refusal by swine. Deoxynivalenol was present at levels of 1.3, 7.9, 7.9, and 28 ppm. The first two were from Austria, the third from Canada and the fourth from Indiana. No T-2 toxin and no DAS were found in any sample.

In September, 1977, in northwest Ohio, after realizing that corn was damaged by *F. Roseum*, a survey performed on affected corn showed the reddish mold that usually begins observable growth at the ear tip. Of 52 field samples, 24 were positive for DON at from 0.5 to 10 ppm.
In an outbreak of swine feed refusal, infertility, and hyperestrogenism associated with 2 lots of corn in France, zearalenone was present at 10 and 25 ppm, nivalenol at 4.28 and 1.18, deoxynivalenol and 0.6 and 0.14 ppm and T-2 toxin at 0.02. None of these was found in control corn.

In association with high rainfall and humidity in summer, 1980, portions of the Ontario winter wheat crop were found to contain deoxynivalenol. Of samples intended for export, the mean concentration of deoxynivalenol was 0.15 ppm with the highest level being 0.42 ppm. Of samples from Ontario elevators most samples had less than 1% affected kernels but one sample was more heavily infected with a resultant deoxynivalenol concentration of 8.5 ppm. Incidents of feed refusal and feed refusal plus vomition were reported in association with this outbreak. Gilts fed the contaminated wheat in a complete feed (1 ppm deoxynivalenol) refused feed the first 3 days and the average daily gain was 71.6% of control animals. No vomition was noted and no gross lesions were seen at necropsy.

When poultry were fed up to 0.7 ppm deoxynivalenol in feed there was no change in egg production and there was a slight but possibly economically significant decrease in egg weight and shell thickness.

ILLINOIS FEED SURVEY

In a recent survey conducted at the Illinois Regional Diagnostic Laboratory at Centralia and the University of Illinois, College of Veterinary Medicine, Laboratories of Diagnostic Medicine, a total of 342 samples were analyzed. Of these, 274 or 80.12% were positive for deoxynivalenol. This was not a random sampling of feedstuffs but was a result of feeds submitted either with a request for mycotoxin analysis or with a history suggestive of a mycotoxin problem. Thus, the sample is biased in favor of finding deoxynivalenol.

These positive samples were comprised of 49% feed, which was primarily mixed complete feed but which also included silage and some haylage specimens. Other positive specimens included 47% corn; 2.5% oats; and 1.5% wheat. The mean vomitoxin residue in feed was 2.7 ppm with a range of 0.1 to 22 ppm. The mean concentrations of deoxynivalenol for corn, oats and wheat were 3.1, 3.8, and 18.6 ppm and the respective ranges of deoxynivalenol concentration were 0.23-4.6; 0.22-13.3; and 0.14-36.7 ppm. The sample included only 7 oats and 4 wheat specimens, and may not be representative of moldy oats and wheat in this year. Most feedstuffs analyzed were of the 1981 harvest.

Fifty-four percent of the submissions did not state what species to which the feedstuff was being offered. Of the remaining 46%, three quarters was being used for swine and approximately 1/9 was fed to cattle and 1/9 to horses. Approximately 2% was being fed to sheep and only 1% was reported as being fed to birds.

Of 28 cases with reasonably detailed histories, reproductive problems, if lumped together, were prevalent being mentioned in 50% of the
complaints. Prolapses occurred as the most frequent reproductive problem with lesser numbers of infertility, swollen vulvas and abortion. These signs are very likely attributable to zearalenone. Of the 28 cases, feed refusal was mentioned in 12, weight loss or poor gain in 7, diarrhea in 5, death in 4 and vomition in 3. In some cases in which estrogenic signs were mentioned, zearalenone if present, was undetected at a limit of detection of 100 ppb. This could easily be attributable to sampling error. Of course, other causes of certain of these signs may have gone undiagnosed. One may also consider the possibility of the influence of undetected relatives of zearalenone in the feed or, alternatively, an interaction between low levels of zearalenone and deoxynivalenol.

DEOXYNIVALENOL TOXICOKINETICS

Returning to kinetics, the plasma disappearance of deoxynivalenol from swine over time was slower than in the case of T-2 toxin or DAS. Approximately 25% of the total dose of deoxynivalenol administered intravenously was accounted for in the urine. The concentration in urine peaked at 6 hours and by 24 hours virtually no more deoxynivalenol was being excreted in the urine.

WEANLING PIG STUDY

In the deoxynivalenol feeding study 3 groups of 18 weanling pigs at 5 to 6 weeks of age were fed rations formulated with corn naturally contaminated with deoxynivalenol. The concentrations in the finished feed of deoxynivalenol in ppm were 0.7, 3.1 and 5.8. Urine collected from swine at 0.7 ppm in feed contained .25 to .40 ppm and at 5.8 ppm in feed, 1 to 3 ppm.

Weight gain and feed efficiency at the 5.8 ppm level were virtually wiped out the first week and were poor the third week. These animals developed rough hair coats and appeared somewhat gaunt. Animals in the intermediate dose group gained 2/3 as much as those in the low dose group the first two weeks and were less feed efficient during the first week. During the fourth and fifth weeks the two higher dose groups began catching up to the low dose groups in body weight. There was only occasional vomition or diarrhea in any group. The liver, muscle and kidney residues of a pig fed the 5.8 ppm ration were all approximately 30 ppb and spleen was approximately 13 ppb after two weeks of feeding.

SUMMARY

Deoxynivalenol is less potent but much more prevalent that T-2 toxin or DAS in the United States and probably the world. Specimens for analysis should include, first as the preferred specimen, feed. We generally request a 10 lb. sample of feed as representative as possible of the entire feed or feed component. The specimen should be shipped so as to arrive in good condition. Therefore, high moisture feeds should be shipped either frozen or by courier fast enough to prevent further mold growth. One could analyze stomach or rumen contents for any of these mycotoxins provided that the animals are eating up to the time of death.
which of course will not often be the case. Therefore, this specimen would be obtained only rarely, and would be desired only when representative feed was unavailable. In animals receiving deoxynivalenol in feed, urine could be analyzed to detect the presence of this compound. However, at the present we cannot clearly correlate the significance of given levels of deoxynivalenol in the urine except to say that the presence indicates exposure. Deoxynivalenol testing should not await the onset of vomition as this sign is not among the most prevalent even in swine. For this reason the term vomitoxin should perhaps be abandoned. Finally, it is common to find both zearalenone and deoxynivalenol together although in given cases the presence of estrogenic effects may occur when zearalenone is undetected. More than likely all the estrogenic effects described are due to zearalenone or similar metabolites. However, the potentiation of zearalenone by deoxynivalenol may be worth consideration.

REFERENCES

Chairman: G. D. Osweiler, Columbia, MO
Vice Chairman: W. B. Bixler, Rockville, MD

The Environmental Residues Committee met Monday November 8 at 1:30, with approximately 20 members and guests in attendance. Discussion was organized around the problem of inadvertent residues in food animals resulting from contamination or adulteration of Animal Feeds. Three panelists presented approaches to preventing or reducing inadvertent residues.

Dr. Wm. B. Buck, University of Illinois explained a program for Animal Feed Specimen storage. This program would allow producers to sample their own ingredients on a routine basis and to submit them regularly for chemical analysis. Testing would be based on those agents with high potential for residue contamination. This program could be expanded to include small feed mills and suppliers lacking in-house facilities for quality assurance against chemical contamination.

The quality assurance program of the National Renderers Association was presented by Mr. Maurice Streigler, Bastrop, Texas. Their Association through its Task Force for Hazardous and Toxic Chemicals has recently passed a resolution suggesting the testing of all fat products going into the food chain and all animal proteins produced in conjunction with fat that is not shipped into the food chain. Testing would include PCB's and Chlorinated Hydrocarbons. They recommend that frequency and manner of testing be adapted to the volume and needs of individual plants. In addition, they provide a listing of laboratories available for doing the analyses. A workshop on Disaster prevention in Rendering Plants is to be offered in the near future. The National Renderers Association recommends that whenever a multiple death problem is encountered, members should verify through a veterinarian the cause of death and if the animals were contaminated. In addition, the state veterinarian and/or State Department of Agriculture should be contacted to ensure safety of rendering such products. The Association is working closely with USDA and FDA to initiate preventive programs and quality assurance for reducing contamination of rendered products in animal feeds.

Dr. Carl Graham, Kansas City, Missouri, outlined major areas of concern for contamination through the feed industry. Although relatively in-
frequent, feed contamination most often occurs due to ingredients contaminated before purchase or acquired from common carriers. This often involves fertilizers and Agricultural Chemicals. Major classes of Feed ingredients vary in their potential for contamination, with animal by products, Plant proteins and grain products being most at risk. In house practices to prevent inadvertent contamination include:

(a) Control of insecticide and fumigant use and storage  
(b) Use of lubricants approved for food contact  
(c) Use of uncontaminated paints and epoxys  
(d) Care in application of spray-on insulations

Major preventive steps recommended in the feed industry include (1) Knowledge of the supplier and (2) Maintaining a quality control and testing program. Quality control is made more difficult by the large volume and rapid turnover of feed ingredients. However, quality control can provide an indication of chemical trends in ingredients and serves as a quality control for suppliers of ingredients.

Panelists and the committee agreed that while problems and approaches of the various segments of the industry are complex and different, some common approaches can be made.

These include:

(1) Awareness of the potential for contamination must be enhanced through education and publicity aimed at all levels of the industry. The programs outlined by panelists in this committee could do much to improve awareness. Another example of awareness, being pursued through FDA, is a National Symbol for Feed Ingredient and Chemical Bags to indicate materials with potential for contamination.

(2) There is continued need for close cooperation among all phases of the feed ingredient industry and animal food chain. A problem in one segment often is passed on to other portions of the industry.

(3) Industry and Government should continue to work together in a reasonable and cooperative manner stressing the preventive approaches discussed here.

Additional reports were given.

Drs. L. Lomax and H. Gosser, University of Georgia, reported on studies with Cyclopiazonic acid mycotoxin in swine. Characteristics of poisoning by the toxin and recognition as a potential feed toxicant were discussed. Dr. T. M. Wilson, Pennsylvania State University, reported an update on the Fusarium Research Center at Pennsylvania State University. This serves as a repository for pure cultures of medically and botanically important Fusaria and is available for use of veterinarians and others.

Meeting adjourned at 4:15 p.m.

Report Submitted November 9, 1982
KINETICS OF INACTIVATION OF AFRICAN SWINE FEVER ANTIGEN WITH BINARY ETHYLENIMINE

G. M. Schloer, Microbiologist
United Stated Department of Agriculture
Agricultural Research Service
The Plum Island Animal Disease Center
P.O. Box 848
Greenport, New York 11944

ABSTRACT

African swine fever (ASF) soluble antigen, used for routine diagnostic tests, is produced from infected Vero cells. The residual infectivity of antigen after solubilization and ultracentrifugation ranges from $10^{6.5}$ to $10^{8.0}ID_50$/ml. Antigen shipped from Plum Island Animal Disease Center high containment laboratory has to be completely inactivated while still retaining antigenicity. Since binary ethylenimine (BEI) inactivates nucleic acid with minimal effect on protein, it was used to inactivate the residual virus present in soluble ASF antigen. The effect of time, temperature and concentration of BEI was studied. Inactivation with 10 mM BEI at 37°C for 3 hours completely inactivates virus, but some loss of antigenic activity occurred. Treatment with 10mM BEI at 17°C inactivated 5 logs of virus after 5 hours of treatment, while 5.0 mM BEI inactivated 5 logs of virus in 7 hours. Inactivation with 10mM BEI for 20 hours at 17°C was used to assure complete loss of infectivity while still retaining most of the antigenic activity. Inactivated antigen was found to contain no residual infections ASF virus as demonstrated by inoculation of swine and tissue cultures.

INTRODUCTION

Soluble African swine fever (ASF) antigen is currently used for both the immunoosmoelectrophoresis (IEOP) (4), and enzyme-linked immunoassay (2). Large scale production of antigen was initiated at the Plum Island Animal Disease Center (PIADC) as a result of the simultaneous report of outbreaks of ASF in Brazil and the Dominican Republic in 1978 (3). Over 10 liters of antigen have been prepared, inactivated, safety tested and stored at -70°C and are available for shipment. Antigen has been shipped both to the Dominican Republic and Haiti for laboratory use in order to monitor swine sera during the ASF eradication programs in those countries.

All biologicals shipped from PIADC must be free of infectious agents, thus, complete inactivation of antigen is mandatory before shipment. Acetylenimine (AEI), B-propiolactone, and glycidaldehyde have been shown to effectively inactivate ASFV (7), but the compounds are considered potentially carcinogenic for humans. Binary ethylenimine (BEI) may also be potentially carcinogenic, but is is generated from bromethylamine (BEA), a less toxic compound (1). Previous work had
shown that ASF antigen treated with 10 mM BE1 for three hours at 37°C completely inactivates the residual virus in the antigen, but does result in some loss of antigenicity (6). Moreover, storage of antigen at temperatures above \(-70°C\) also reduces antigen titer. The kinetics of inactivation of ASF antigen was studied in order to establish optimal conditions of antigen inactivation and improve the quality of the antigen.

**MATERIALS AND METHODS**

**Virus and cell culture.**

The twelfth passage of ASFV/Brazil/78 virus adapted to Vero cell cultures was received from I. C. Pan of the PIADC. Serial passages of virus were made as described previously (6). A microtiter assay in Vero cells was used to titrate the virus infectivity. Serial ten-fold dilutions of virus were employed using four replicates per dilution. Titers were expressed in ID\(_{50}\). Conditions of the assay will be described (manuscript in preparation).

**Immunoosmoelectrophoresis and immunodiffusion assays.**

Details of the assay were described previously (4, 6). All samples were tested with a hyperimmune serum obtained from a pig inoculated with ASFV/Dominican Republic/78 and ASFV/Lisbon 60. Serum was obtained from A. H. Dardiri of the PIADC. Samples for assay included the untreated virus, zero time and the final product after each treatment. Block titrations of antigen and antisera were done for the IEOP assay, while for the immunodiffusion (ID) assay serial two-fold dilutions of antigen were tested against a single concentration of antiserum.

**Soluble ASF antigen.**

The production of ASF antigen was described previously (4, 6). In brief, Vero cells grown in 110 x 285 mm glass roller bottles were inoculated with virus at a multiplicity of infection ranging from 0.5 to 1.0. After a 4 hour adsorption period at 37°C, infected cells were incubated at 33°C for 40 to 44 hours. ASF antigen was obtained from sonicated infected cells. After sonication, the supernatant fluid was clarified by low and high speed centrifugation. The final supernatant fluid was diluted with phosphate buffered saline (PBS) as pH 7.4, and the antigen was stored at -70°C until it was activated. The antigen before inactivation has a residual virus titer ranging from \(10^6.5\) to \(10^8.0\) ID\(_{50}\). The protein concentration was 5 mg/ml.

**Determination of the optimal temperature of virus inactivation**

The rate of inactivation of residual ASF virus present in antigen in the presence of 10 mM BE1 was determined at 37°C, 27°C, 17°C and 7°C. The production of BE1 from BEA was described previously (1). In brief, a solution of 100 mM BEA in 200 mM NaOH was heated at 37°C for 1 hour to produce 100 mM BE1. Two control tubes containing 200 mM NaOH and respectively, were also made. Three flasks of antigen and three tubes containing 100 mM BE1, 200 mM NaOH or PBS, respectively, were equilibrated for 30 minutes at the above temperatures. At zero time, 1.0
ml of BEI was added to 9 ml of antigen in a flask, followed by the addition of 1.0 ml of 200 mM NaOH or 1.0 ml of PBS to each of the respective flasks. Flasks were agitated by stirring. Inactivation of virus by BEI was stopped by the addition of an excess of Na$_2$S$_2$O$_3$. Accordingly, 0.9 ml samples from each flask were rapidly pipetted into 0.1 ml of 200 mM Na$_2$S$_2$O$_3$. Samples were taken at 2 minutes after initial inoculation, considered as zero time, and at appropriate intervals thereafter.

**Determination of the optimal concentration of BEI.**

The antigen was produced from the 23rd passage of ASFV/Brazil/78 in Vero cells. Four flasks containing 9 ml of antigen and 4 vials of BEI containing 100 mM, 50 mM, 25 mM and 10 mM BEI were equilibrated at 17°C. At 0 time, 1.0 ml of each concentration of BEI was added to a flask of antigen, which resulted in four flasks of antigen containing 10.0, 5.0, 2.5, and 1.0 mM BEI. Flasks were agitated by stirring. Samples of 0.9 ml were taken from each flask at 0, 2, 4, 5, and 24 hours post inoculation (p.i.) and diluted into 0.1 ml of 200 mM Na$_2$S$_2$O$_3$. Samples were stored at 5°C before assay.

**RESULTS**

**Optimal temperature of inactivation.**

The rate of inactivation of virus by 10 mM BEI is linear and decreases with the decrease in temperature at 37°C, 27°C and 17°C, with no viable virus detected at 1, 3 and approximately 10 hours, respectively. Reaction at 7°C was slower with some residual infectivity seen at 24 hours post inoculation. Antigen diluted in PBS showed no drop in titer when treated at 37°C for 2 hours, 27°C for 4 hours, 17°C and 7°C for 24 hours. In all cases, antigen treated with 200 mM NaOH at the above time and temperature was non-infectious.

IEOP assays on the original, zero time and final samples after treatment at different temperatures are shown in Figure 2. There was only a minimal effect on antigen treated with 10 mM BEI at 37°C and 27°C for 1.5 and 2 hours, respectively. Little or no reduction in antigenicity was seen in virus treated with 10 mM BEI at 17°C and 7°C. Figure 3 shows the results of titrations of antigen with 10 mM BEI at different temperatures. There appear to be at least 4 major precipitin lines detected in both unstained preparations of the IEOP and ID assays. The number of lines diminishes with antigen dilution. A principal antigen had a titer of 1/32 in the ID assay when virus was treated with BEI at 17°C and 7°C. However, the antigen had a titer of 1/8 when treated at 37°C for 1.5 hours with 10 mM BEI. The identity of these principal antigens is unknown at present.

Inactivation with 10 mM BEI at 37°C and 27°C is extremely rapid, whereas inactivation at 17°C was effective and readily controlled. This temperature was chosen as optimal for inactivation.

**Optimal concentration of BEI.**

The rate of inactivation at 17°C with varying concentrations of BEI is
seen in Figure 4. Inactivation with 1.0 mM BE1 was slow with no inactivation occurring at 5 hours p.i. Some inactivation not indicated in the figure was seen at 24 hours p.i. Inactivation of virus with 2.5 mM BE1 was first detected at 4 hours p.i. Treatment with 10 mM BE1 showed inactivation of 5 logs of virus after 5 hours of treatment, while 5.0 mM BE1 inactivated 5 logs of virus in 7 hours.

The residual virus titer in different lots of soluble ASF antigen can range from $10^{6.5}$ to $10^{8.0}$ ID$_{50}$/ml. Most lots of antigen prepared for inactivation represent a pool of approximately 360 ml. In order to assure complete inactivation of residual virus, antigen was inactivated with 10 mM BE1 for 20 hours at 17°C.

DISCUSSION

Complete inactivation of residual virus present in soluble ASF antigen is necessary because the antigen is shipped both to Latin American countries and to the National Veterinary Services Laboratory at Ames, Iowa. Over 15 lots of ASF/Brazil/78 antigen, 400 ml of antigen per lot, have been treated by the above method. All of the lots have shown a complete loss of infectivity as indicated by inoculation of both animals and tissue cultures (unpublished data). Although 5 mM BE1 treatment may also be effective, treatment with 10 mM BE1 for 20 hours at 17°C results in minimal effect on antigenicity and assures inactivation of virus. Moreover, repeated tests on antigen stored at -70°C for as long as 2 years show but a small decrease in antigenicity as measured by the IEOP assay (unpublished data).

African swine fever virus is an extremely stable virus, which can withstand wide extremes of temperature and pH (5, 8). Our results show that ASFV is rapidly inactivated by 20 mM NaOH at a final pH in antigen of 9.0. This differs from previous reports which indicate that ASFV remains infectious after treatment for 6 hours at pH 13.0. Both the total amount of protein present in the preparation and, perhaps, differences among virus strains may account for the discrepancy in results.

The number of precipitin lines seen both by the IEOP and ID assays is an indication of the multiplicity of antigens present in the ASF antigen preparation. It is interesting to note that one of the antigens appears to be sensitive to treatment at 37°C. The identification and isolation of the principle viral proteins that react in the IEOP assay will result in an improved and more specific test.

Inactivation of residual ASF virus in antigen with 10 mM BE1 at 17°C for 24 hours effectively inactivates infectivity of ASF virus with minimal effect on viral antigenicity.

ACKNOWLEDGEMENTS

The excellent technical assistance of David Perkins and Elizabeth King is gratefully acknowledged.
Figure 1. Comparison of the effect of different temperatures on the inactivation of ASF/Brazil/78 antigen treated with 10 mM BEI.
Figure 2. IEOP titration of ASF/Brazil/78 antigen using hyperimmune anti-ASFV swine serum. Antigen samples taken before treatment and after treatment with 10 mM BEI at different temperatures for 0, 1.5, 2.0, 5.0 and 24 hours. Numbers within the boxes refer to the number of precipitin lines found between each respective antigen and antiserum dilution.
Figure 3. Immunodiffusion titration of ASF antigen with hyperimmune anti-ASFV serum as described in Figure 2.

Figure 4. Comparison of the effect of different concentrations of BEI on the inactivation of ASFV/Brazil/78 at 17°C.
REFERENCES


RIFT VALLEY FEVER:  
GLOBAL SPREAD OR GLOBAL CONTROL?  

H. W. Lupton, DVM, PhD, C. J. Peters, MD, and  
G. A. Eddy, DVM, PhD  

From the Department of Viral Pathogenesis and Immunology, Virology Division, U.S. Army Medical Research Institute of Infectious Diseases, Fort Detrick, Frederick, MD 21701.  
The views of the authors do not purport to reflect the positions of the Department of the Army or the Department of Defense.  

SUMMARY  

Rift Valley Fever (RVF) has long had a major role as a domestic animal and human pathogen in sub-Saharan Africa. With the Egyptian epizootic of 1977-78 the virus demonstrated its ability to spread and cause extensive disease in a totally new ecological context, the irrigated region of the Nile valley. Conditions which might prove receptive to epizootic disease exist not only in the Middle East (e.g., Israel, the Tigris-Euphrates basin) but also in areas which could be reached by incubating or viremic air travelers (e.g., the western hemisphere, Australia, Southeast Asia). Precise predictions of the risk of introduction and spread of disease in these regions are hampered by our lack of knowledge of the ecology of the virus and its vectors in Africa. The principal method of effective control of RVF in Africa is the use of potent veterinary vaccines for sheep and cattle, and supply would limit their use in areas of recent disease extension. These problems can be overcome through the effective application of existing research and technology. While it seems unlikely on the basis of the historical record that RVF will be responsible for epizootic/endemic disease in the United States, increasing air communications augment the risk of return of viremic travelers to potentially receptive areas in the western hemisphere. In the eventuality that an epizootic of this mosquito-borne disease should occur in North or South America, the choice of control measures is limited, and the control methods used for diseases such as foot and mouth disease would have little applicability.  

The burgeoning field of veterinary arbovirology deals with important domestic and exotic veterinary diseases such as Rift Valley fever (RVF), Venezuelan encephalitis, African horse sickness, African swine fever, Nairobi sheep disease, and ephemeral fever. Both from the veterinary and medical standpoints, RVF virus (RVFV) is potentially one of the most dangerous of these and of the approximately 400 other registered arboviruses.1 The virus itself is a member of the Phlebovirus genus of the family Bunyaviridae. These viruses have a three-segmented, negative-sense RNA genome and mature into approximately 100-nm lipid-enveloped virus particles in the region of the cellular Golgi apparatus. In general, they are transmitted by mosquitoes or sandflies. They usually cause acute cytolytic infections in cell culture which are reflected in
vertebrate infections which resolve without the development of chronicity or persistence. The biology of RVFV was reviewed by Weiss in 1957, exhaustively cataloged by Easterday, and recently updated by Peters and Meegan.

Classical Epidemiology—RVF virus was first discovered in Kenya in 1931 as a consequence of a major epizootic in sheep with secondary human infections. Subsequent research in Uganda, Kenya, Zimbabwe, South Africa, Nigeria and elsewhere in sub-Saharan Africa suggests that the virus exists throughout most of the continent (Fig. 1) in a poorly-understood natural cycle which probably has no relation to man or domestic animals. However, periodically, climatic conditions (usually periods of high rainfall) lead to an increase in the density of biting arthropods in an area where susceptible domestic animals exist. The virus then may spread from its enzootic setting to infect the herds and initiate explosive epizootics. There is no evidence that there has been geographic spread of the virus within Africa with two exceptions: (a) the 1977 Egyptian epidemic-epizootic to be discussed below and (b) recognition of RVF in South Africa with the 1951 epizootic. It seems likely that the active South African veterinary profession would have identified the disease prior to 1951 if it had been a major problem on the veldt. It is possible that the virus spread from the eastern coastal forests of Natal province where it is probably enzootic, or alternately that some other factor such as changing husbandry practices led to its emergence as an epizootic disease.

The major targets during typical epizootics are European breeds of sheep and cattle. Ruminants indigenous to Africa are also susceptible to some degree although much less is known about their clinical response. Infected sheep and cattle develop high viremias and perhaps 10 to 30% die from hepatic necrosis. Pregnant animals virtually always abort. Young animals are more susceptible to the disease and mortality approaches 100% in lambs under a week of age. The high viremia which develops in susceptible species has several consequences: (a) it is easier to infect potential arthropod vectors; (b) blood and recently slaughtered carcasses may be important in transmission. This is compounded by the properties of the virus: it is highly infectious for man and animals by injection or by aerosol; and (c) virological diagnosis is usually easy, if attempted. There is some evidence that encephalitis may occur occasionally in domestic animals and it is regularly seen in experimental infection of rodents.

Transmission—As mentioned above, we have no real knowledge of the enzootic cycle. Mosquitoes infected with RVFV have been trapped deep in the forest and may be involved. Since the virus has been isolated from

---

*As this manuscript went to press, Dr. James Meegan has just identified an ungrouped arbovirus of West Africa. Zinga virus, as actually being RVFV. This extends the range of RVF through Central and Western Africa.*
several species of mosquitoes during epizootics, they are also thought to be important in that setting. Biological transmission has been established in the laboratory, but mosquitoes studied quantitatively have been relatively inefficient vectors of RVF. They require high concentrations of virus in their blood meal before they are infected and transmit the virus to only a fraction of susceptible hosts when they feed again. The poor vector capacity of the species tested and the low isolation rates from unengorged mosquitoes trapped during epizootics raise some doubts as to the role of mosquitoes as the sole vectors of RVF. It is likely that the high viremia of sheep and cattle and voracious biting by large numbers of the insects overcome these quantitative considerations, but other mosquitoes and other arthropods are under investigation as potential transmitters. RVFV belongs to a group of viruses often associated with sandflies, but no isolates have been made from the modest number of Phlebotomines tested. RVFV has been recovered from Culicoides by two workers, but the significance of the isolations still needs to be evaluated. Ticks have neither yielded field isolates nor transmitted infection in the laboratory.

Since laboratory studies show the virus to be highly infectious by parenteral inoculation and physically stable, the role of contaminated arthropod mouth parts in mechanical transmission must be considered. Disease might be transferred if an arthropod began feeding on a viremic animal and then moved to a susceptible host to finish its meal. This could be relevant to the isolation of RVF virus from black flies (Simulium) in South Africa.

One might go further and ask whether any arthropod is necessary to spread the disease. Although occasional ovine-to-ovine transmission has been demonstrated under experimental circumstances, the bulk of evidence suggests that arthropods are the major transmitters of RVFV to domestic animals. However, the situation with man is different. Most documented human infections in Kenya, South Africa or Zimbabwe occurred after necropsy or slaughter of RVF-infected livestock and were thought to be transmitted by contact or aerosols.

Lessons from the Egyptian Epidemic—The previous discussion has dealt predominantly with RVF as we understand it from sub-Saharan Africa. In 1977, RVF was recognized in Egypt for the first time and resulted in a massive epidemic. RVFV antibodies were reported in human sera from the adjacent Sudan as long ago as 1936, yet both historical and retrospective serological evidence suggested that RVFV had not been present in Egypt for decades prior to the epidemic. The limited oligonucleotide fingerprint data available support the concept of a single introduction of RVFV into Egypt. The exact determinants of the introduction will probably never be known, but there are several plausible hypotheses. One possible explanation is that the disease previously occurred in areas of the Sudan which had little traffic with Egypt. Perhaps as a consequence of changing ecology around Lake Nasser, the 1976 Sudan RVF epidemic, or other factors, camel
caravans entering Egypt either at Aswan or through clandestine desert routes to Sharquiya Governorate in the delta may have carried the virus with them. Because of the distances involved, one must invoke several cycles of acute infection, transport of infected vectors, or other mechanisms for the virus to reach the delta. Whatever the explanation for the introduction of RVF, the virus spread rapidly and caused an impressive epizootic/epidemic. The close ecological relation of man to his domestic animals or the proclivity of local vectors for human feeding may have led to the high human attack rates observed during the Egyptian epidemic. In 1977, 18,000 human cases with 598 deaths were officially reported.11 If the results of a 1978 serosurvey34 are extrapolated to the official population figures there may have been more than a million infections of man. The epidemic in the human population resulted in many patients with complications including retinal vascular disease,35 encephalitis, and hemorrhagic fever.36 Some have speculated that the Egyptian RVFV strains may be more virulent than classical isolates, but there are no quantitative field or laboratory data to support this assertion.10 As a matter of fact, the low incidence of severe or complicated human disease in Egypt (perhaps 1%) plus the reports of similar complications from South Africa28 and Zimbabwe37, 38 suggest that most RVFV strains have a similar spectrum of pathogenicity. The impact of RVF on the domestic animal population was only documented by anecdotes, but the death of breeding stock, loss of lamb crops, and post-abortion infertility must have impacted significantly on the availability and cost of animal protein in Egypt.39

After a very impressive epizootic/epidemic in the summer and fall of 1977, virus activity declined with winter. With the onset of warm weather in 1978, transmission of virus once again occurred and the disease clearly extended its geographic distribution within Egypt to involve several new governorates.11, 39 During 1979, there were occasional anecdotes of outbreaks and a few RVF virus isolates, but by 1980 there was even less evidence of RVF activity. The potential for RVF infection in Egypt apparently continues to exist (susceptible domestic animals, similar ecologic and climatic conditions), but for no documented reason disease activity has diminished and perhaps even disappeared. It is impossible to predict whether RVF will cease to be a problem in Egypt or whether it is temporarily dormant and will erupt to cause major disease.39

Prospects for Local Spread—During the Egyptian epidemic contiguous countries may have been at risk for spread of the disease. Israel, concerned with desert nomad traffic and increased commerce with Egypt following the Camp David agreements, instituted a broad campaign of surveillance and vaccinated domestic animals.40 Several nearby areas seemed to offer potentially receptive conditions for RVF extension, including the Tigris Euphrates river basin with a delta ecology resembling that of the lower Nile. In spite of these concerns and of clear evidence of spread of disease within Egypt, there was no sign of RVF activi-
ty in nearby countries. Early reports of serological evidence of RVF in United Nations troops and Bedouin residents of the Sinai peninsula were based on cross-reactive serological tests. Reexamination of these same sera as well as independent surveys with the more specific neutralization test have not indicated the presence of RVF in the Sinai.

Prospects for Distant Spread—Could RVF be introduced into a country such as the United States and establish itself as a major disease problem? Introduction through domestic animals or pets seems unlikely since disease activity is mainly rural and distant from shipping points, the incubation period is short, and duration of viremia is brief. Chronic RVF infection is unknown, although residual virus may persist in lymphoid organs for several weeks. Humans are more mobile and may circulate very high titers of virus in their serum, so that secondary spread might occur through man-biting arthropods or blood samples sent to the clinical laboratory. An example of this type of episode was reported recently after a Canadian tourist became infected with RVF during a game safari in Kenya. Fortunately her acute illness occurred before leaving the country so she was probably no longer viremic when she developed retinitis at her next destination. This lady is one of two recently reported cases of RVF retinitis occurring in foreigners in Africa. Since ocular disease is thought to be an uncommon sequela of RVF infection, RVF may occur in visitors to rural enzootic or epizootic areas more frequently than currently appreciated. However, only 2 of 400 missionaries who had spent from several months to several years in Africa had serological evidence of previous RVF.

If a viremic human returned to this hemisphere, he might well infect biting arthropods. Certainly mosquitoes from the United States can transmit RVF in the laboratory, but we have no way of knowing whether they achieve the densities and other conditions necessary to sustain an epizootic. Local sheep and cattle are known to be susceptible to infection. Thus, all the links in the chain are present, the probabilities are the only imponderables. Since there is no history of an introduction, future problems seem less likely, although this must be weighed against the increasing frequency of intercontinental air travel. The explosive Egyptian epidemic gives little comfort when considering the potential consequences.

Recognition of an introduction. The alert practitioner could be instrumental in providing rapid diagnosis of cattle or sheep disease before further spread could occur. The occurrence of death from liver necrosis in adult animals, more widespread fatalities in younger animals, and extensive abortion among cows and ewes would be the initial clinical presentation (reviewed in 4, 5, 73, 74). Many persons exposed to blood from sick animals during their destruction or necropsy would develop a febrile illness with muscle aches, photophobia, and prostration. An unfortunate few of these humans would develop blindness, encephalitis, or fatal hemorrhagic fever. Recognition of this constellation of events in the field should lead to the request for special diagnostic tests. In the United
States, Plum Island Animal Diagnostic Center has the responsibility for veterinary RVF virus isolation and the Centers for Disease Control, Center for Infectious Diseases, Special Pathogens Branch, for human diagnosis. The practitioner should remember that samples from acutely ill animals or abortuses may contain large quantities of virus and would be highly infectious. Responsible organizations should be contacted for details of specimen collection and transportation. If RVF is suspected, the samples should not be sent to a routine diagnostic laboratory because of the danger to the laboratory personnel. The importance of awareness of practitioners and farmers can be illustrated from previous epidemics. The 1951 South African epizootic/epidemic required 6 months for final diagnosis; however the Egyptian outbreak was identified within 3 weeks after recognition because of aggressive laboratory work and a high index of suspicion.5

*Veterinary Vaccines*—A major tool, either to deal with established disease in Africa or an introduction into a previously uninvolved area, would be vaccination of susceptible livestock. Available evidence suggests that control of disease in domestic animals will prevent amplification of virus transmission to arthropod vectors and terminate human disease as well. It should be borne in mind that this assertion has never been tested in areas of high human population density where man-arthropod-man cycles might occur. RVF vaccines should fulfill the same criteria as the veterinary immunogens, but certain qualities need additional emphasis.51 Virulent epizootic disease often occurs in areas where it is necessary to utilize an inexpensive vaccine which induces long-term immunity and can be produced from an unsophisticated technological base. The biologic characteristics of the virus make fetal or neonatal pathogenicity a major potential side effect and fetal or neonatal protection a major goal. Since the parent virus is a human pathogen that is infectious by aerosol, an attenuated strain would have to be safe for man or precautions against human infection would have to be incorporated into vaccine production and deployment programs. Geographic areas where RVF is a potential threat or where recent RVF introductions have caused disease but where an enzootic cycle is not clearly established, would have stringent requirements for total inactivation of killed immunogens. These regions would also require that attenuated vaccines not have the potential to revert to a virulent state or produce a viremia infectious for arthropods.

Two vaccines exist and are in common use in sub-Saharan Africa to control livestock disease (Table 1). An attenuated vaccine was derived by serial intracerebral passage of virulent RVF virus in adult mice to produce the so-called “Smithburn neurotropic strain.”18, 52, 53 This vaccine can be readily produced in infant mice, is stable on storage in the lyophilized state, and gives long-lasting protection to adult animals as well as passive immunity to their offspring. An inactivated vaccine was developed by formalin treatment of RVFV grown in tissue culture.54, 55 The resulting product was absorbed on aluminum hydroxide gel and
stored in the liquid state up to 6 months before use. This vaccine also provides solid protection, but there are no published studies to establish the duration of immunity and the need for booster injections. The production of the inactivated vaccine requires relatively expensive cell culture facilities and rigorous testing for residual live virus.

In spite of their usefulness in sub-Saharan Africa, the applicability of these two vaccines to the prevention or control of RVF in this country or other potential extension areas is limited. The currently used attenuated vaccine has never been cloned or genetically characterized. It may contain or develop virulent populations when inoculated into sheep or cattle. Although acceptable for use in enzootic areas, the vaccine is abortogenic and teratogenic for a small fraction of sheep. Many vaccinated sheep develop detectable viremias and the transmission of this viremia to insects is an unwelcome possibility which has never been explored experimentally. As an example of the genetic heterogeneity which may be present in RVFV populations, mouse virulence of the clones of an Egyptian isolate from a fatal human hemorrhagic fever case has been compared to that of the Smithburn neurotropic strain, IB8 (a tissue-culture adapted strain avirulent for sheep and cattle), and Lunyo (a naturally occurring mouse-attenuated RVFV variant). Several of the clones from the hemorrhagic fever isolate are as attenuated for mice as putative vaccines. Clearly it is impossible to predict but desirable to investigate whether clones of the attenuated virus or other RVFV isolates will be genetically stable, immunogenic, nonteratogenic, and useful as vaccines. Alternate approaches could be based on exploring the reassortant potential of the Bunyaviridae family to produce "hybrid" viruses or an attenuation by chemical mutagenesis under laboratory control.

The currently available inactivated veterinary vaccine is produced in the liquid state and may have a storage life of only months, which precludes stock-piling to deal with future emergencies. Data on onset and duration of protection are limited. Nevertheless, the development of an improved killed vaccine would reduce the need for costly, elaborate and perhaps unsuccessful development and testing of candidate attenuated vaccines. Furthermore, a killed vaccine would be more acceptable for use in countries bordering enzootic areas or to attempt to eradicate RVF (perhaps following an introduction) than any live vaccine. The major problem, as always, lies in the development of a nonliving immunogen with sufficient antigenic mass to develop a solidly protective immune response rapidly. Experience with the existing inactivated vaccine and with less potent prototype vaccines in domestic animals suggests that relatively small amounts of antigen can produce a protective response of rapid onset and that this response can be measured by sensitive tests for serum neutralizing antibodies. Several cell culture substrates may be applicable and techniques to augment viral antigen production employed so successfully with poliovirus could be useful for RVF vaccine. The resulting vaccine would presumably be
stable in freeze-dried form, since lyophilized human RVF vaccines have been successfully stored for more than 10 years. Alternately, gene-cloning technology or antigen capture by monoclonal antibodies might be applicable to prepare more potent immunogens.

Surveillance for RVF—Because of its aerosol infectivity and potential for introduction and spread in non-African countries, work with live RVFV requires precautions which limit the general application of diagnostic tests. Work with live virus (isolation from clinical specimens or neutralizing antibody tests) should be performed only by vaccinated personnel; otherwise, laboratory infections can be expected. High standards of laboratory containment have been recommended in nonendemic areas to prevent the possibility of an accidental introduction of RVFV. A beta-propiolactone-inactivated antigen for the hemagglutination-inhibition (HI) test has been developed and can be used safely in any laboratory. The specificity of the HI test is limited, so that isolated positive sera require confirmation by a more specific test, such as neutralization, to exclude the possibility of cross-reacting antibodies from other related Phleboviruses. Negative results with a properly performed HI test are useful in excluding RVF; multiple high-titered positive reactions in the presence of epidemic abortion and human disease would be virtually diagnostic of RVF. Recent interest by the U.S. Department of Agriculture, the Office International des Epizooties, the World Health Organization, and the Food and Agriculture Organization makes it likely that awareness of the threat of RVF to potentially receptive countries will increase and that an epizootic might be recognized more quickly to permit institution of control measures.

Control of RVF in Africa—Eradication of RVF from as-yet uncharacterized enzootic foci is obviously not feasible. Control of epizootic disease in domestic animals has been attained with the inactivated and live attenuated vaccines discussed previously. In sub-Saharan Africa this has presumably prevented disease in farmers and veterinarians. Residents of enzootic areas remain at risk. An investigational inactivated human vaccine has been used successfully in laboratory workers and may be of benefit to veterinarians, abattoir workers, and others at high risk of infection. As mentioned above, the RVF epidemic-epizootic in Egypt dissipated for unknown reasons. The detailed mechanisms of transmission were never adequately characterized and the impact of the major attempted control measure, aerial spraying, was never conclusively demonstrated.

Control of a RVF Introduction—Since there are multiple hosts, multiple vectors, and even multiple ways to infect (parenteral, aerosol, or biological arthropod transmission), control and eradication could be challenging. The paradigm in planning for an eventuality of this type should not be foot-and-mouth disease, but rather a disease such as Venezuelan encephalitis which shares with RVF the characteristics of multiple arthropod vectors, high viremias in domestic animals which lead to amplification of vector infection, absence of latent infection or carrier
states in mammalian hosts, low probability of contact infection between animals, and a high potential to cause human disease (reviewed in 70). In these circumstances the classical approach of destroying apparently infected herds would lead only to increased economic losses; brief viremia, infected vectors, asymptomatic infections, and explosive spread would result in control teams killing predominantly immune animals with the least potential for spreading disease. Vector control by aerial spraying is often invoked in arboviral epidemics but expenses are measured in millions of dollars and efficacy may be limited, particularly if control must be directed toward multiple species of vectors which live in diverse habitats and may possibly be resistant to insecticides.71 There are no veterinary RVF vaccines currently licensed for use in the United States. The current products do not have defined standards of efficacy and safety which have been reviewed in this country, and all are produced in countries where foot-and-mouth disease is endemic. The investigational human vaccine is very expensive, supplies are limited, and there are no present plans for expanding stocks. Thus, we are left with the conclusion that we have no effective means of combating an RVF epizootic in this country if the virus should be introduced and encounter favorable conditions for epizootic spread.

Immediate needs—Dissemination of information to veterinary and medical practitioners and laboratories about the potential of RVF for spread and about the characteristic clinical and epidemiological patterns would facilitate early diagnosis of an introduction. This would decrease extension of the virus, limit human and animal disease, and enhance attempts at control and eradication. In the short term, reserves of a lyophilized inactivated veterinary vaccine would be useful to deal with the appearance of RVF in the United States. Using the pattern of current human vaccine technology, a safe effective vaccine could be prepared and stored for at least 10 years. Ongoing research in several laboratories should succeed in producing an improved vaccine or vaccine-adjuvant combination within the decade.51-64

Future Research Needs—Economic realities dictate that much of the investigation described below will have to be carried out in developed countries. This can be safely achieved with modern techniques for microbiological containment. The natural history of the entire Phlebovirus genus, particularly RVF, needs further work. If we understood the enzootic cycle of RVF the quantitative determinants of epizootic disease (vector density, vector competence, vector biology, host factors), and the biology of RVF in Egypt or the South African veldt, we might be able to define areas at risk to develop epizootic disease or endemic foci after an introduction of the virus. We could combine this knowledge with information on local vectors to design effective control programs if an introduction occurred. The role of non-mosquito vectors, such as Culicoides sp. seems particularly important. Surveillance of RVF could be greatly improved if safe, rapid, and specific tools for diagnosis could be made field-ready and validated. Experimental enzyme-linked
solid-phase assays for antibody and antigen appear feasible. Additional data on the distribution of RVF in Africa and the risk of infection of travelers would be useful in assessing the probability of introduction of the virus by viremic humans and the potential value of vaccination of travelers to high risk areas of rural Africa. Finally, further vaccine development is desirable to develop improved immunogens. The experience with Venezuelan encephalitis in Texas suggests that a live attenuated vaccine may ultimately be necessary to efficiently control epizootic disease; however, experimental studies with inactivated RVF vaccines suggest that solid protection could be rapidly achieved with a potent non-living immunogen. If these and other points could be resolved, it would be possible to formulate rational recommendations for international and national vaccine policies.

TABLE 1 — Existing Rift Valley Fever Vaccines

<table>
<thead>
<tr>
<th>Feature</th>
<th>Attenuated</th>
<th>Inactivated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Protect against disease</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>2. Onset of protection</td>
<td>? 3-4 days</td>
<td>? 7 days or less</td>
</tr>
<tr>
<td>3. Suppress viremia</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>4. Protect fetus</td>
<td>Yes</td>
<td>Yes, if antibody titer high enough</td>
</tr>
<tr>
<td>5. Colostral protection</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>6. Long-lasting immunity</td>
<td>Yes</td>
<td>Probably requires booster</td>
</tr>
<tr>
<td>7. Teratogenicity, abortogenicity</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>8. Produce viremia</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>9. Reversion to virulence</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>10. Residual virulent virus</td>
<td>No</td>
<td>No, if properly inactivated and tested</td>
</tr>
<tr>
<td>11. Stability on storage</td>
<td>Yes, if lyophilized</td>
<td>Months in liquid state; protracted if lyophilized</td>
</tr>
<tr>
<td>12. Stability in field use</td>
<td>Good</td>
<td>Good</td>
</tr>
<tr>
<td>13. Technological outlay</td>
<td>Mouse colony or tissue culture facility</td>
<td>Tissue culture facility</td>
</tr>
<tr>
<td>14. Doses/ml</td>
<td>≥ 1,000</td>
<td>0.5 - 2</td>
</tr>
</tbody>
</table>
REFERENCES


VESICULAR STOMATITIS OUTBREAK—COLORADO
PRESENTED TO:
FOREIGN ANIMAL DISEASE COMMITTEE
EPIZOOTIC ATTACK PLANS COMMITTEE

Dr. P. R. Henry

VESICULAR STOMATITIS OUTBREAK, COLORADO 1982

In late June and early July, Emergency Programs alerted Regional Directors and Veterinary Services Area Veterinarians in Charge that New Mexico and Arizona were experiencing an outbreak of Vesicular Stomatitis. On July 22, Colorado reported its first case. This case was in horses in Grand Junction. Specimens from this case were submitted by a private practitioner. The first case in cattle was investigated on July 31 in Durango, Colorado. 75% of the animals in a 54-cow dairy were affected by the disease.

On the night of July 27, I received a call from Dr. Schiefer, the section Veterinary Medical Officer and Foreign Animal Disease Diagnostician on the Western Slope of Colorado. He had been investigating reports of vesicular conditions as far north as Craig in Moffat County, which is approximately 200 miles from his official duty station. Dr. Schiefer related that he was inundated with reports from veterinary practitioners who were observing vesicular conditions in horses, cattle, and in at least one case, a human. His estimate was that to investigate reported cases in bovines, we should open field stations in Durango and Grand Junction, Colorado. Further, he recommended that at least 3 FAD Diagnosticians should be detailed to Durango, and that 4-6 Diagnosticians should be detailed to Grand Junction. This report was immediately relayed to the Assistant Regional Director, who authorized the establishment of Field Offices at Durango and Grand Junction, as well as initiating action to detail Foreign Animal Disease Diagnosticians. Animal Health Technicians and other support personnel to these field offices for assignments.

The office in Durango was activated on July 28, and the office in Grand Junction was activated over the next few days. Personnel from nearby states began arriving on July 28, 29, 30 and 31, driving government-owned vehicles. Others arrived by air from distant states, and were conducting investigations by the early part of the first week of August. The Area Office was reinforced with Colorado personnel, and personnel detailed from other offices for maintenance of records and reports, and to coordinate operational activities.

At the peak of the outbreak, 41 personnel were committed full time. This included 40 Veterinary Services employees and one state FAD Diagnostician from Montana. In all, 14 states detailed personnel to the Colorado outbreak.

Initially, we received more reports of vesicular conditions in horses. These were immediately followed by reports of the disease in dairies and beef herds. It became necessary to screen reports to establish priorities.
VESICULAR STOMATITIS

on which cases were investigated; cattle, sheep, goats and swine were first priority. In cattle, those reported having any combination of lesions which included lesions of the feet were the highest priority. This was true in sheep, goats, and swine as well. Practitioners were our best source of reported cases. Most practitioners easily understood the reasons for our investigative priorities. Many practitioners utilized our diagnostic laboratory at National Veterinary Services Laboratories to verify a diagnosis of Vesicular Stomatitis in horses by submitting sera during both the acute and convalescent phases.

Concurrent with our gearing up the organization to conduct a large number of investigations, the media began to insist upon news releases. Current status information was provided almost daily to TV, radio, and press from the area office and both field offices. The media people were, for the most part, patient and cooperative. A side light on news and information of interest is that the Colorado State Department of Public Health made periodic releases of high-quality information. We (Veterinary Services) should have been doing likewise. A full time information specialist should have been on board to relieve busy managers of this time-consuming activity. Perhaps one reason we did not call for an information specialist to work on site was that the course of the disease was uncertain. Another thing that happened was that each time we gave out a news release, there was a subsequent increase in the number of cases reported.

With the buildup of personnel to handle the outbreak, the need for additional administrative support at the field offices became very apparent. Again, we did not request additional administrative support due to the uncertain course of the disease. In retrospect, we should have had this support on board from day one.

A full time Epidemiologist would have been of help in attempting to chart the course and movement of the disease in the field.

A Hewlett-Packard computer terminal and printer were sent to the Grand Junction Office along with an operator, so that the operator could gain experience on entering data from the VS Form 12-27 investigation reports. This data then went into the Emergency Programs computer located at National Veterinary Services Laboratories. Our computer equipment was later moved to the Area Office and operated there until the number of investigations dropped to one or two each day. I believe the experience gained by data entry operators under actual field conditions will be valuable to future outbreak reporting needs. We discovered that the system was not of as much value to us in the field as we had anticipated. This was due in part to our operator not having been trained in data retrieval, and in part to lack of adaptability in the programming.

A team of Agricultural Research Service Virologists and Entomologists conducted insect survey and trapping activities in areas where the disease was most active. Virus isolation results from most insect pools
collected have not yet been reported, though Agricultural Research Service reports that they do have a virus isolation from one pool of culicoides.

A team of Entomologists and Physicians from the Center for Disease Control Branch in Ft. Collins, Colorado, collected samples from people and from animals in those herds where we had done investigations. Veterinary Services furnished a Foreign Animal Disease Diagnostician to this team to collect samples from animals. We have not yet heard from the Center for Disease Control on the results of their investigations.

During the outbreak, Veterinary Services responded to over 400 reports of vesicular disease. Investigations were conducted and specimens submitted from 378 premises. Specimens submitted from 262 of the 378 premises were positive serologically to the New Jersey strain of Vesicular Stomatitis. 65 virus isolations were made from the 262 premises with positive serology. This was 69% with positive serology and 25% with virus isolation. All of the serology and virus isolation was of the New Jersey strain of Vesicular Stomatitis.

Disease outbreaks initially were concentrated in Southwestern Colorado, and in and around Grand Junction, Colorado. Investigations were conducted in all the counties north and south along the Utah State line, from new Mexico to Wyoming. 20 western slope counties reported outbreaks, and eventually investigations were conducted in 33 of the 63 counties in Colorado.

Veterinarians, Animal Health Technicians, Virologists, Entomologists, Epidemiologists and Physicians participated in working on this outbreak. All were alert for a common denominator which would track the movement and explain the spread of vesicular stomatitis.

Many thoughts were expressed about vectors, both plant and animal, the leafhopper was suspected in recently harvested forage, as were aphids. In this regard, we requested that a plant pathologist be detailed to the outbreak for the purpose of studying the relationship of plants, insects and/or mutation of a plant virus to an animal virus. Some people proposed altitude as a factor limiting the geographical distribution of the disease. Other theories were offered suggesting that older native animals did not contract the disease, as opposed to younger animals and animals recently shipped in from areas where Vesicular Stomatitis was not present. It is obvious to me that much information is unknown about Vesicular Stomatitis.

A veterinary economic impact study is underway to determine the losses suffered by dairies as a result of this disease. It is believed the losses are significant.

Surveillance studies made by Agricultural Research Service and Center for Disease Control indicated that all animal populations in the
area where cases existed were affected. This observation is supported by reports from livestock owners in the affected geographic area. We observed cases in horses, cattle, sheep, goats, dogs and humans, including several of our own people.

The few slides I have will portray somewhat graphically the incidence geographically, and some statistics associated with the outbreak.
THE 1982 DANISH FOOT-AND-MOUTH DISEASE (FMD) OUTBREAK

*Drs. William W. Buisch, Keith A. Hand, and Kay W. Wheeler

The impact of the diagnosis of foot-and-mouth disease (FMD) Type O in 66 dairy cattle in Brenderup, Denmark, on March 18, 1982, was felt worldwide. Over 37 percent of the pork shipped internationally is of Danish origin. Forty million dollars worth is exported per year to the United States, and $230 million dollars worth is exported to Japan. In addition, Denmark is a member of the European Economic Community (EEC) and presently markets approximately 80 percent of the pork distributed to the EEC member countries.

When FMD was diagnosed in this outbreak, Canada, Japan, the Scandinavian countries, and the United States immediately banned further imports of fresh, chilled, or frozen meat products from Denmark. This resulted in a prompt increase of 20 percent in the cost for pork products in Japan, and with an overabundance of pork products remaining in Denmark, a reduction of one Danish kroner (1 U.S. dollar = 8 kroner) per kilogram to the Danish farmer. The Danish authorities estimated that FMD cost their livestock industry, including export trade, approximately $20 million Danish kroner per week (U.S. dollars, $2.5 million). The total Ministry of Agriculture expenditures for this outbreak were approximately $22 million Danish kroner. Therefore, it is easily concluded that the benefits of an eradication program for FMD far outweigh the cost when one takes decisive action in slaughtering all known infected herds in order to protect the general livestock population.

In total, 22 cases were diagnosed positive with the last case identified on the Island of Zealand on May 4, 1982. Basically, the outbreak was divided into three segments. The first segment occurred from March 18th through April 14th in the eastern portion of the Island of Funen. The second segment occurred from April 16th through April 21st on the northern portion of the Island of Funen. The last segment of the outbreak occurred with one case diagnosed on May 4th on the Island of Zealand. Epidemiologically speaking, they were not able to tie the three segments together, other than to indicate that the wind, migratory birds, and tourists with contaminated meat products may have played a role in its spread.

In addition, it was noted by the Danish authorities that the occurrence of FMD in Denmark to some extent coincided with reports about FMD in the German Democratic Republic. Again, the prevailing winds, numerous migratory birds, and tourists during the period prior to the 18th of March suggest to the Danish authorities that the source of origin of the outbreak in the German Democratic Republic and in Denmark might have been the same. In our opinion, it appears that most of the 22 positive cases were the result of direct contacts. Veterinary practitioners were responsible for the spread to five of these premises. In addition, milk
trucks or milk-truck drivers were responsible for the spread to six additional premises. A slaughter truck was associated with two additional outbreaks.

Of the remaining nine cases where known contacts did not occur, three cases appear unique. Cases 1, 18, and 22 may have all been primary cases (separate introductions). Foreign tourists rented a cottage about 125 yards from the initial premises prior to the first animal demonstrating clinical signs. The owner of case 18 owned a number of fishing cottages and renters of these had to pass through his barnyard to get to the cottages. An energy plant located about 1.5 miles from premises 22 commonly receives coal from FMD countries.

The remaining six cases probably resulted from area spread due to undetermined direct contact from movements of personnel and equipment and movements of barn cats and dogs. A large number of flies were noted on several of the positive premises and may have accounted for some spread. Also, the Danish regulatory officials consider the wind as a source of spread, especially when the humidity was extremely high.

The last time Denmark had a case of FMD was in April 1970. In addition, vaccination for FMD had been prohibited since 1977. With a susceptible livestock population, it was essential that a strict “stamping-out policy” be initiated. The disease eradication program was based on an immediate notification by farmers when disease was suspect, the immediate and rapid depopulation of infected herds, “freeze” on movements of animals, etc. (area disease control measures).

The factors in establishing an early diagnosis included:

1. A prompt notification of practitioners by farmers.
2. Short transport time of diagnostic specimens from infected premises to laboratory.
3. The willingness of private practitioners, State Veterinary Service officials, and laboratory officials to work around the clock.

A final diagnosis was based on:

1. Typical clinical signs only.
2. Clinical signs and established contact to a previous case.
3. Clinical signs confirmed by a laboratory examination.

Laboratory confirmation included:

1. Isolation on bovine and porcine kidney cell cultures.
2. Complement-fixation test.
3. Enzyme-linked Immunosorbent Assay (ELISA) test.
4. Inoculation of 4-day old baby mice.

In addition to the depopulation and cleaning and disinfection of the infected premises, a control area was established for a radius of 10 kilometers.

Within this area:

1. Movement of cloven-hoofed animals was prohibited.
2. Visitors (except veterinarians) to farms with cloven-hoofed animals were prohibited.
3. Artificial insemination schedules were canceled.
4. Removal of feed and manure from a farm in the area was prohibited.
5. Public milk control, mastitis control programs, etc., were prohibited.
6. Entering barns by milk-truck drivers was prohibited.
7. Milk-truck pipes were disinfected on each farm.
8. Slaughtering was only allowed in local slaughter houses.
10. Voluntary cancelation of public events; example, sports.

In addition, within 250 meters of the infected premises a cordon sanitaire was established. This was enforced by the local police and in effect completely stopped the movement of animals, animal products, animal feed, waste, and utensils from the affected area. In addition, movement of people to and from the area was allowed only by authorization of the local police. People on the affected farm could not leave until cleaning and disinfection of the buildings and surroundings were complete. They were also to abide by the restrictions for the control area and all depopulated premises were restricted from restocking until authorized by the State veterinary authorities.

On June 4, 1982, Denmark declared themselves free of FMD based on article 2.1.1.2 of the International Zoosanitary Code of the Office of International des Epizooties. The article basically establishes that an FMD infected zone may be considered as being free from the disease when at least 30 days have elapsed (after "stamping-out policy") after disinfection and no new case of the disease has been found.

The total compensation for the animals destroyed was $16.5 million Danish kroner (U.S. dollars, $8.1 million). This included indemnity for 1,839 head of cattle, 2,352 pigs, and 3 sheep. The eradication efforts of the Danish FMD outbreaks were impressive. The ability to virtually stop total movement within the control zone kept area spread to a minimum. The complete cooperation of farmers (many of whom had previous knowledge due to past outbreaks of FMD) was a definite advantage in detecting new cases. Also, the ability to depopulate infected herds, prior to massive amounts of virus being produced further reduced the risks of area spread.

Seven of the 22 cases were diagnosed and depopulated with only one animal in each of these herds demonstrating clinical signs. This further substantiates the cooperation of the industry and the dedication of the Danish regulatory veterinarians.

The United States was immediately notified of the March 18, 1982, FMD outbreak in Denmark and was most fortunate in being allowed to
send observers. In addition, followup reports were received in a timely manner from the Danish authorities, the U.S. Embassy in Denmark, and the Office of International des Epizooties.

As a result of this outbreak, all fresh, frozen, or chilled meat from Denmark after March 6, 1982, was prohibited entry into the United States. Also, all other Danish animal products and byproducts were regulated. Present policy indicates that Denmark will be considered for FMD free status by the United States after 1 year has elapsed from the last diagnosed case of FMD.

The prompt, decisive action demonstrated by the Danish authorities in the control of FMD was most impressive. Hopefully, we will be able to respond as successfully when we are faced with similar circumstances.
The committee on Foreign Animal Diseases met on November 9 and 10 during the annual USAHA meeting held at the Radisson Hotel, Nashville, Tennessee. Seventeen of the committee members and approximately thirty guests attended the meetings.

The meetings were highlighted by several presentations which summarized the current states of exotic disease of concern to the U.S. livestock industry. Drs. Dardiri and Edwards, from the USDA Plum Island Animal Disease Center, addressed the subject of heartwater which was recently reported on the island of Guadaloupe in the Caribbean. Dr. Dardiri gave an overview of the disease which was followed by Dr. Edwards's report on his recent visit to several Caribbean Islands in search of further evidence of heartwater outbreaks. While the vector tick, *Amblyomma variegatum* is widely distributed in the islands, the disease does not appear to be widespread.

Dr. Floyd M. Jones, USDA FAD advisor, in Guatemala and El Salvador
presented a slide program (report) on FAD training programs in Central America. This included training programs, simulated exercises, and demonstrations presented to local groups, government veterinarians, quarantine inspectors, and international groups of technical personnel. Included in this presentation was a discussion of the suspected ASF outbreak in Guatemala which was later proved to be classical hog cholera and not ASF.

Dr. Hunt McCauley gave an update on his extensive owner survey of schistosomiasis mortality in Sudanese cattle, and Dr. Harold Lupton presented a paper on Rift Valley Fever (see proceedings manuscript) a disease of continuing importance in Africa. Dr. Gertrude Schloer, from Plum Island gave an interesting paper on the kinetics of inactivation of African Swine Fever virus antigen (see proceedings manuscript). Dr. Al Smith of Oregon State University reviewed calicivirus infections in various animal species.

EMERGENCY PROGRAMS PROGRESS REPORT

During fiscal year 1982 (October 1, 1981-September 30, 1982), there were 184 investigations in the United States for foreign animal diseases.

An investigation of a stomatitis outbreak in horses and cattle in Arizona, on June 2, 1982, resulted in the diagnosis of New Jersey vesicular stomatitis. In late June, vesicular stomatitis was diagnosed in horses in New Mexico, and a number of cases occurred in cattle during the first 2 weeks of July. By mid-July, numerous cases of the disease began occurring in western Colorado and eastern Utah. The largest number of cases were investigated in Colorado. In early August, vesicular stomatitis was found in Wyoming, and by late August, the disease was found in Idaho. During September, additional cases were found occurring in Montana, Nebraska, and South Dakota. In October, the State of Washington was also added to the list of those involved.

Cattle, horses, sheep, goats, buffalo, and dogs were found to be affected (positive serology). Human infection was also found in many of these States.

Between June 2 and November 3, 1982, numerous field investigations were conducted in states, where laboratory confirmation of the disease had been obtained from blood samples. A summary of these is presented in Chart I.

<table>
<thead>
<tr>
<th>State</th>
<th>Total Cases Investigated</th>
<th>Lab Results of Investigated Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pos</td>
</tr>
<tr>
<td>Colorado</td>
<td>406</td>
<td>301</td>
</tr>
<tr>
<td>Wyoming</td>
<td>75</td>
<td>44</td>
</tr>
<tr>
<td>Utah</td>
<td>25</td>
<td>23</td>
</tr>
<tr>
<td>Idaho</td>
<td>109</td>
<td>70</td>
</tr>
<tr>
<td>New Mexico</td>
<td>37</td>
<td>31</td>
</tr>
</tbody>
</table>
Investigations have been also conducted and samples submitted from States surrounding the outbreak: These summaries are presented in Chart 2.

<table>
<thead>
<tr>
<th>State</th>
<th>Total Cases Investigated</th>
<th>Lab Results of Investigated Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pos</td>
</tr>
<tr>
<td>California</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Iowa</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Illinois</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Kansas</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Louisiana</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Minnesota</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Missouri</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>North Dakota</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Oklahoma</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Oregon</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Texas</td>
<td>19</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>0</td>
</tr>
</tbody>
</table>

The last major epizootic of vesicular stomatitis in the U.S. occurred in 1964-66.

The initial velogenic viscerotropic Newcastle Disease (VVND) case for fiscal year 1982 was found on December 21, 1981. This case occurred in Colorado Springs, Colorado, and was eradicated by December 28, 1981. Twelve additional cases occurred during fiscal year 1982—nine cases in California, two cases in Texas, and a single case in Florida. Most of these resulted in dead-end cases as the positive birds were purchased from unknown individuals on the street or at “swap meets.” Two cases in California occurred in pet shops. Again, the investigation revealed that the infected birds were purchased “off the street.” Although the source of the disease was not disclosed, there was tracing of sales from these shops. The case in Florida occurred in a combination retail outlet—exhibitor—training facility. For a time, the owner refused to sign the indemnity claims, and since State regulations were not enforced, it took over 1 month to close the case. One case in Texas resulted in some anxiety as the owner purchased the bird and then proceeded to tour the zoo with the bird in a camera bag; however, additional exposure did not occur.

On March 29, 1982, Emergency Programs was notified that the University of Kentucky Livestock Diagnostic Laboratory had a complement-fixation (CF) titer for contagious equine metritis (CEM) in
several mares in Kentucky. These mares had been bred to "J. O. Tobin," a stallion located at Spendthrift Farms. On March 30, 1982, the mares "Finely" and "Valenciennes" were culture positive for CEM. The mare "Cautious Bidder" was culture positive for CEM on April 2, 1982. Approximately 16,500 CF tests and approximately 900 cultures were conducted on mares in Kentucky without additional infection being disclosed.

On July 27, 1982, and again September 2, 1982, a fly, *musca vitripennis*, was intercepted at McGuire Air Force Base, New Jersey. This is the first recorded introduction of this fly in the Western Hemisphere and is important in that it may carry the nematode, *Parafilaria bovicola*, the causative agent of hemorrhagic bovine parafilariasis. A survey of the area in early September suggested that the fly has not become established. Another survey is planned for this spring.

One foreign animal disease diagnosticians' course was conducted during this fiscal year. Therefore, with the addition of 13 newly trained diagnosticians, we now have available in the United States a total of 241 veterinarians trained in the differential diagnosis of foreign animal diseases.

Foreign animal diseases awareness seminars were held at veterinary colleges in the States of Illinois, Massachusetts, Minnesota, and Oklahoma. A foreign animal disease seminar for diagnosticians was held at the National Veterinary Services Laboratories, Ames, Iowa. A course and two seminars on military support for emergency animal disease programs were given. In addition, a wildlife seminar for foreign animal disease diagnosticians was held in Athens, Georgia, under the direction of the Southeastern Cooperative Wildlife Disease Study, and a seminar for teachers of foreign animal diseases was held in Hyattsville, Maryland.

The five Regional Emergency Animal Disease (READEO's) are fully staffed and maintained to respond rapidly to outbreaks of emergency diseases.

A test exercise was held to test our preparedness to respond to an emergency outbreak situation. The Southeastern and South Central Regions within Veterinary Services were involved. NADA (a code name for foot-and-mouth disease) was used for the exercise in question. The computerized Recorded Emergency Animal Disease Information system was used successfully in the test exercise and was also used during the recent outbreak of vesicular stomatitis.

During November 1981, we participated with Mexico in their test exercise simulating an outbreak of foot-and-mouth disease. In January 1982, Canada, Mexico, and the United States met to discuss the establishment of a North American Foot-and-Mouth Disease Vaccine Bank. Legal documentation relative to the establishment of the bank is currently in process.

With the repeated introductions of VVND into southern California and
the risk it was posing to the poultry industry, a regulation requiring the identification through banding of pet birds from California was implemented. This was short lived, however, due to the lack of support of the pet bird and poultry industries for the manner in which it was implemented. It is still, nevertheless, in the regulations and may be imposed; if necessary.

The Technical Support (TS) staff of Emergency Programs (EP) has continued to support Cooperative Agreements with the University of Wisconsin and the Southeastern Cooperative Wildlife Disease Study in Athens, Georgia. These projects are concerned with refinement of a fingerprinting technique in the diagnosis of VVND and defining the role that wildlife will play in an outbreak of relevant foreign animal diseases.

During the past year, our staff has been involved in several activities in the Caribbean. A tick survey was conducted in the Dominican Republic and Haiti to determine the prevalence of *Ornithodoros puertoricensis*, potential vectors of African swine fever. A mission was also completed in six islands of the Eastern Caribbean for the purpose of monitoring the movement of the tropical bont tick, *Amblyomma variegatum*, and beginning surveillance activities for heartwater disease in this part of the Caribbean Basin.

The TS staff has completed printing a new disease guide, the Rift Valley Fever Eradication Guide.

The Data Bank now has approximately 46,000 articles on foreign animal diseases covering 23 diseases and entomological items. Memorandums of Understanding were completed in which 18 of the 27 U.S. colleges of veterinary medicine have agreed to assist the Animal and Plant Health Inspection Service upon request, during officially declared animal disease emergencies.

Significant progress has also been made in the refinement of the Recorded Emergency Animal Disease Information (READI) computer system. The system is intended to quickly transmit pertinent, diagnostic, epidemiologic, and administrative data among the five READEO's, the National Veterinary Services Laboratories (NVSL), and Hyattsville. Training of data entry personnel and the use of graphics in output display have been accomplished.

The National Animal Disease Surveillance System will be discussed in other committees reports, and is on target in its development.

GLOBAL STATUS OF ANIMAL DISEASES EXOTIC TO THE UNITED STATES

*Foot and Mouth Disease (FMD)*

Denmark had 22 cases, of type A starting in March, 1982, with the last case occurring in May. The country had been considered free for a number of years and is now trying to regain that status. A recent survey conducted on blood samples from slaughter cattle and deer, revealed no
serological evidence of the disease. U.S. Veterinary Services personnel were able to observe the outbreak and eradication measures adopted by the Danes.

East Germany also had an outbreak of type O1 in the spring. Their eradication measures were not as drastic, resulting in further outbreaks in August on two previously infected premises.

Turkey is having numerous cases of FMD, a source of great concern to Europeans. Types O1 and A22 have been reported.

West Germany had a single case (type O1) in garbage-fed pigs in June, 1982. Spain reported one case (type A) in March.

The Soviet Union reported cases late in 1981 and early in 1982. Most were type O1, but a few were type A22.

In Africa, the disease was reported from Egypt and Libya (type O), Tunisia (type A), Nigeria and Zambia (SAT2), Burundi, Kenya, and Mozambique (SAT3), South Africa (SAT1), and Zimbabwe (SAT1 and SAT2).

From Asia, disease reports came from India (type O, A, C, and Asia), Kuwait (Type C), Iran, Sri Lanka, Thailand, Hong Kong, Iraq, Jordan, Uganda, Pakistan, and Saudi Arabia (all type O). There were no reports from the People's Republic of China; we assume the disease exists there.

In the Americas, FMD was reported from Argentina and Bolivia (types A, O); Brazil (types A, O, C); Colombia (types A, O); Ecuador (type A); Paraguay and Peru (types A, O, C); Uruguay (type A) and Venezuela (types A, O). In 1970, Chile started a systematic campaign to rid the country of FMD. This campaign appears to have been successful as no outbreaks have occurred since 1979. The country is now making efforts to gain U.S. recognition of FMD-free status.

Swine Vesicular Disease

Swine Vesicular Disease was again reported from Great Britain, Italy, and West Germany. While Great Britain appears to make the most strenuous efforts to eradicate the disease, it remains the country reporting the most outbreaks.

African Swine Fever

African Swine Fever until recently, remained stable in its distribution. It was reported from Italy (Sardinia), Spain, Portugal, Angola, Mozambique, and quite recently, Cameroon. Since few disease reports from Brazil are received, we cannot consider the disease to be eradicated there. The Dominican Republic remains free, as, apparently, does Cuba. Eradication in Haiti is underway, making good progress after initial program difficulties.

Hog Cholera

Hog Cholera is still causing problems in many parts of the world. It was reported from Korea, Taiwan, Japan, Hong Kong, Thailand, Chile, Brazil,
Colombia, Mexico, Paraguay, Peru, Guatemala, Greece, Italy, Portugal, France, Luxembourg, Belgium, and Holland. Holland had managed to stay free of the disease for some time; the reintroduction apparently occurred from Belgium and resulted in depopulation of over 20,000 pigs.

**Rinderpest**

Rinderpest is still reported from Upper Volta, Niger, Nigeria, Mali, Tanzania, Egypt, and Sudan in Africa. An international vaccination campaign, similar to the successful efforts mounted in the 1960's is being organized, but the present worldwide economic slump creates funding difficulties. The disease is also reported from the United Arab Emirates, Syria, Iran, Saudi Arabia, and India. It appears that live animal imports to OPEC countries permitted the disease to enter some of these areas.

**Contagious Bovine Pleuropneumonia (CBPP)**

Vaccination campaigns for the eradication of rinderpest can be combined with efforts to vaccinate for CBPP without great difficulty, and it is hoped that this will be considered in future campaigns as both diseases often occur in the same areas. CBPP was reported from Angola, Nigeria, Kuwait, Upper Volta, Mali, Uganda, Ghana, Cameroon, Kenya, and Ivory Coast. Very likely, the disease exists elsewhere in Africa. Of special interest are several foci of CBPP discovered early in the year in Southern France. Extensive surveillance and eradication measures are underway.

**Lumpy Skin Disease**

Lumpy Skin Disease was reported from South Africa, Angola, Burundi, Madagascar, Namibia, Uganda, and Kenya.

**African Horsesickness**

African Horsesickness is more or less consistently reported from South Africa and Namibia but the disease may exist in other countries not reporting it. For import purposes, the whole continent of Africa is considered infected.

**Dourine**

Dourine is occasionally being reported from South Africa and Namibia. Persistent reports also come from Italy.

**Glanders**

Glanders is occasionally seen in South Africa, Namibia, and Turkey.

**Rift Valley Fever**

No cases were reported.

**Teschen Disease**

Only reported in Madagascar, but we assume it is present elsewhere.

**Sheep and Goat Pox**

Sheep and Goat Pox were reported in Morocco, Kuwait, Turkey, Kenya, Libya, Nigeria, Iran, Mali, Tunisia, Israel, Jordan, and Algeria.
Contagious Equine Metritis

Contagious Equine Metritis was reported from the United Kingdom, Ireland, France, West Germany, Belgium, Italy, Austria, Denmark, Sweden, Australia, Japan, and the USA.

Venezuelan Equine Encephalomyelitis

No cases reported.

Research on Vesicular Diseases

This group of viral diseases consists of foot-and-mouth disease (FMD), swine vesicular disease (SVD), vesicular exanthema of swine (VES), and vesicular stomatitis (VS). This is a review of selected reports considered pertinent to the specific diseases.

Foot-and-Mouth Disease Virus (FMDV)

Following last year's report of the first production, through gene splicing of an effective vaccine against FMD (1), work has continued on the cloning of major vaccine strains. Stable expression was engineered for VP₃ of additional strains and immunogenicity demonstrated to date for at least two additional types (2). Another recent advancement in the future control of foot-and-mouth disease is the chemically synthesized peptides corresponding to several regions of VP₃ and the inducement of neutralizing and protective antibodies by these peptides (3). Although many problems remain to be solved, the results obtained show promise. The possibility of a practical multivalent Bio or organically synthesized vaccine raises hopes for the future (4).

Fweyemamu and Ouldridge (5) report on antigenic variation of FMDV as a factor in the control of the disease and recommend that for vaccine selection, tests based on the virus neutralization reaction are preferred to those based on the complement fixation reaction.

A report on the thermal stability of FMDV by Doel and Baccarini (6), and the resistance of heating to 49°C of inactivated and non-activated 146S particles of seven strains of FMDV is of interest. They found that external stabilities of the strains differed significantly and suggested a correlation between external stability of the 146S particle and vaccine potency.

The titration of FMD antibodies in cattle sera in a comparative study of the serum neutralization test in suckling mice and ELISA showed the results obtained by the two tests to be in good agreement (7). Lombard and Pirond (8) compared an enzyme-linked immunosorbent assay (ELISA) to a cell culture serum neutralization test (SNT) for the detection of FMDV antibodies in the sera of vaccinated cattle. At 21 days after either a primary or revaccination, both tests indicated similar levels of antibody. At 3 and 6 months after vaccination, the titers were slightly higher by ELISA, perhaps indicating the ELISA might be sensitive to non-neutralizing antibodies.

Because a number of outbreaks of foot-and-mouth disease have been
associated with failures of the inactivation process, one of the main themes of the meeting of the Research Groups of the Standing Technical Committee of Tubingen, 1981, dealt with vaccine safety testing. Six papers were presented on various aspects of safety testing vaccines. The use of elution or elution and concentration aided in the detection of infective particles when tests were conducted in either mice or tissue culture. The first order kinetics of the inactivation of virus infectivity by the azaridine compounds was also discussed. The use of a double dosing system of inactivant at 24 hours and 48 hours made it possible to arrive at probable confidence levels for innocuity that are many orders of magnitude below those being detected in any other form of innocuity test. There is still much interest in methods for the inactivation of FMD virus for vaccines and the optimum methods for testing to ensure that only safe products reach the field.

During and after the 1975 outbreak of type O FMD in Malta, cattle, sheep, goats and pigs were sampled to determine the extent of apparent infection and the effect of vaccination on the development of carriers. Oesophageal pharyngeal (OP) fluid samples were tested for virus and serum samples for virus infection-associated antibody. FMDV was detected in only three of 278 OP samples from ruminants on 23 premises (free of clinical disease) during course of the outbreak. No virus was recovered from 305 ruminant OP fluid samples taken two months after the last case of disease on premises close to those infected at the time of the outbreak. Antibody to VIA antigen was detected in 11 of 196 serum samples taken on premises at high risk during the outbreak and 12 of 379 samples taken on a second visit. All samples taken a year later were negative. Despite large amounts of virus excreted by pigs, the spread of virus during the outbreak appears to have been confined largely to clinical disease (10).

Scientists at Pirbright have shown by electrofocusing of virus-induced polypeptides from infected cells and fingerprinting of ribonuclease T hydrolsates of the virus RNA that viruses causing the outbreaks FMD in Jersey, the Isle of Wight and France belong to the same strain. It has also been shown that this strain is indistinguishable from the O Lausanne 1965 strain used in vaccine production. This outbreak may have been caused by escape from a laboratory, by the use of a vaccine contaminated with the virus or by an incompletely inactivated FMD vaccine (11).

The findings obtained by Glaster et al. (12) and Donaldson et al. (13) support the hypothesis that under certain conditions, the airborne transmission of FMD over a long sea passage is possible.

The outbreak of foot-and-mouth disease in Britain in 1981 and the outbreak in Denmark in 1982 have been attributed to airborne transmission. Both countries, prior to these outbreaks, had been free of FMD for approximately 12 years.

**Swine Vesicular Disease**

The first outbreak of SVD occurred in Great Britain in December 1972
FOREIGN ANIMAL DISEASE

and to date there have been 483 outbreaks. During the last four years, confirmed outbreaks have been mainly confined to one region, linked directly to outbreaks in that region or have occurred as isolated cases related to the feeding of swill. SVD legislation in Great Britain is based on that for FMD and all pigs are slaughtered when infection is confirmed with compensation at full market value. Slaughter policies are also used in West Germany, Austria and Japan (14).

An indirect enzyme-linked immunosorbent assay (ELISA) has been developed for the rapid serological confirmation of swine vesicular disease (SVD) and for the surveillance of antibody against this disease in pigs. The ELISA was less sensitive than the double immunodiffusion (DID) (15).

SVD virus is one of the most resistant animal viruses to physical changes and because of its size and single-stranded RNA genome, is also one of the most resistant to gamma radiation. Since it occurs in substantial titers in epithelial and other tissues, and is highly infectious for pigs, it is a useful virus with which to test any effluent treatment system (16).

Vesicular Exanthema of Swine Virus (VESV)

Feeding swine seal tissues from inoculation experiments resulted in seroconversion in swine which were fed tissues from seals infected with vesicular exanthema of swine virus but not in those which were fed tissues from seals infected with San Miguel sea lion virus. Two to three weeks post-ingestion the antibody titers were comparable to those of convalescent swine. This indicates that seroconversion did occur in these seal-fed swine and that even inapparently infected wildlife may pose a threat to domestic livestock (17).

Vesicular Stomatitis Virus (VSV)

A widespread outbreak of vesicular stomatitis, (New Jersey strain) has occurred in Colorado, New Mexico, Wyoming, Arizona, Utah, Idaho, Nebraska, Montana, South Dakota, Washington, and Tennessee. This outbreak, starting early June, has been the first reported outbreak in this area since 1966. Reported cases are investigated to ascertain that foot-and-mouth disease has not entered the country. Research reported this year involves mostly molecular biology and is too extensive to be included in this report.

REFERENCES


EXPORT—THE BOTTOM LINE ON ANIMAL HEALTH

Harold A. Waters, D.V.M.*

It is an honor for me to be invited to address the General Assembly of this historically significant organization. As I have reviewed the reports of the previous meetings going back to 1897, I have noted that exportation of animals has been one of the stated objectives for the control and eradication of a disease, beginning with bovine pleuropneumonia, and continuing through tick fever, tuberculosis, foot-and-mouth disease, fowl plague, brucellosis, hog cholera—the list is endless. The control and eradication of each of these diseases have opened markets both with our neighbors to the North and South, as well as abroad. Last year, 1981, livestock, poultry, and semen valued at more than $224 million (a summary is in Table 1) was exported from the United States.

Table 1

The following volume of animals, poultry, and cattle semen were exported from the United States during calendar year 1981 with dollar value as listed:

<table>
<thead>
<tr>
<th>HEAD</th>
<th>VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baby Chicks Breeding Stock</td>
<td>15,347,677</td>
</tr>
<tr>
<td>Baby Chicks Except Breeding</td>
<td>23,665,231</td>
</tr>
<tr>
<td>Stock</td>
<td>1,544,894</td>
</tr>
<tr>
<td>Turkey Poults</td>
<td>1,511,907</td>
</tr>
<tr>
<td>Baby Ducks, Geese, Guineas</td>
<td>5,894,905</td>
</tr>
<tr>
<td>Live Poultry, other than Adult</td>
<td>47,963,804</td>
</tr>
<tr>
<td>Sub Total</td>
<td>27,247,078</td>
</tr>
<tr>
<td>Eggs Hatching (doz.)</td>
<td>27,247,078</td>
</tr>
<tr>
<td>Sub Total</td>
<td>399,724</td>
</tr>
<tr>
<td>Cattle</td>
<td>87,818</td>
</tr>
<tr>
<td>Horses</td>
<td>66,900</td>
</tr>
<tr>
<td>Sheep and Goats</td>
<td>220,882</td>
</tr>
<tr>
<td>Swine</td>
<td></td>
</tr>
<tr>
<td>Sub Total</td>
<td>399,724</td>
</tr>
<tr>
<td>Cattle Semen</td>
<td>2,587,882 doses</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This information was furnished by the Foreign Agricultural Service, USDA, and is derived from U.S. Department of Commerce sources.)

*Formerly Chief Staff Veterinarian, Export Animals Health Program, Veterinary Services, APHIS, USDA. present address, Animal Health International, Inc. 5235 Yorktown Blvd., Arlington, VA 22207.
Please note, as an example, horse exporters enjoyed $120 million export market. This broad market was protected and enhanced by the eradication of Venezuelan equine encephalomyelitis in 1971 at a cost of only $19.5 million.

The exportation of this healthy genetic material generates an estimated additional 96 cents for each dollar in farm exports from this country. That means, for example, that the $224 million in exports last year generated $215 million in economic activity for a total direct and indirect benefit to our economy of approximately $439 million.

Now that we have looked at the benefits of export, let's look at the barriers. First, we must complete brucellosis eradication. Cattle from States not Class A, or Free, and swine from nonvalidated herds and areas are not acceptable, and cannot meet the import requirements of most countries. Tuberculosis-free States, and we now have 20 of them, are being recognized abroad as safe places to select cattle. We need the other 32 States in the cattle tuberculosis-free category. Pseudorabies in swine must be eliminated to gain entrance for pigs into countries which either embargo our pigs or insist upon a cumbersome testing regime and prolonged import quarantine. Tests and quarantine cost money which cannot be spent for improved genetics. Bluetongue continues to be a major barrier. Many countries insist upon animals originating in bluetongue-free herds or areas. Perhaps a surveillance system and a vector abatement program would provide the safeguards requested, if developed. Enzootic bovine leukosis is an emerging disease of economic significance, primarily in export. The Uniform Methods and Rules for the Establishment and Maintenance of designated Bovine Leukosis Virus-Free Herds, proposed in committee of this organization in 1980, if implemented, would be a start toward opening markets now closed to our cattle. Further research on this disease is also needed. These are but just few examples of where we have an answer to animal health import restrictions on our livestock if they were implemented.

This does not mean we can rest on our accomplishments. The cost of the recent outbreak of foot-and-mouth disease in Denmark is an example of the impact of a disease upon the economy of a country. All Danish pork and swine imports were stopped by countries free of this disease. Effective import quarantine procedures in our country are vital to preserving and developing our animal, poultry, semen, and embryo export potential.

While I am mentioning animal embryos, I should note that this is a growing segment of our cattle and swine export market. Properly done, embryo transfer is an effective means of providing disease-free genetic material. In developed countries there will be a growing demand for an embryo health certification system. This organization is in a good position to develop recommended health standards for this purpose for adoption by State and National governments.

The United States Animal Health Association has had an Import-
Export Committee for a number of years. This year an Export Subcommittee has been established to give special attention to the health problems associated with export. I have been asked by our President, Glenn Rea, to chair this group. The Import-Export Committee Chairman, Clint Booth, and I have tried to develop a good representation from all aspects of the export industry. We will endeavor to serve the interests of this organization as we work within the committee structure.
REPORT OF THE COMMITTEE ON IMPORT-EXPORT

Chairman: Clint Booth, Texas

J. N. Armstrong, NV; Ken Baumgartner, IL; Duane Bohrer, ND; R. B. Caffey, MD; Dan Childs, FL; S. J. Cougar, TX; Jack Dahl, ND; J. R. Day, TX; R. L. Evinger, ID; A. E. George, MD; D. J. Gilhooley, HI; R. C. Goulding, CA; John Gray, TX; W. B. Grene, FL; A. E. Hall, IL; Frank Harding, IL; B. W. Hawkins, OR; D. E. Herrick, MD; James House, NY; R. C. Knowles, MD; Bob Mathis, AZ; Robert Nicholas, CA; E. G. Ongert, NV; Brent Perry, TX; D. A. Price, CO; William Prichard, OR; G. B. Rea, OR; Charles Reid, FL; James Roswurm, CA; R. H. Rumler, VT; R. M. Scott, MI; H. M. Steinmetz, DC; J. E. Thomas, NV; J. S. Walker, NY; Basil Ward, NY; Harold Waters, VA; Maurice Mix, VT.

The Committee on Import-Export met on November 10, 1982, during the annual meeting of the USAHA, held at the Radisson Plaza and Hyatt Regency Hotels, Nashville, Tennessee. The meeting was called to order with 25 members and a total attendance of over 50 people.

The chairman asked the committee for comments on last year's committee report.

Dr. D. E. Herrick, assisted by Drs. Sam Richardson and Alan George, reviewed the past year's activity of the Import-Export staff.

Review of 1982 Activities of Import-Export Animals and Products Staff

IMPORT ANIMALS

There have been several changes regarding the importation of horses from countries where contagious equine metritis (CEM) exists. Stallions from CEM countries may be imported under permit provided they have been treated and cultured negative in the country of origin. Imported stallions must also undergo scrubbing, treatment, and culture negative while in U.S. quarantine. In addition, the stallion must test breed mares and then must also culture negative for CEM. The number of States approved to receive stallions for the required treatment and quarantine is nine. These approved States are California, Colorado, Kentucky, Maryland, New York, North Carolina, Ohio, South Carolina, and Virginia.

Mares from CEM countries may be imported under permit provided they are treated for CEM overseas and culture negative. Part of the required treatment includes surgical removal of the clitoral sinuses which have been incriminated as harboring CEM organisms. Approximately 300 mares have been imported under the new CEM regulations during fiscal year 1982. Approximately 5 percent of these mares had incomplete clitoral sinusectomies.

Initially, mares with incomplete surgery were refused entry. Later, the regulations were changed to allow the importer the option of having the mare sent to New York State College of Veterinary Medicine, Cornell University, Ithaca, New York, to receive additional surgery. Imported
mares must also be treated and culture negative while in U.S. quarantine. There are six States approved to receive mares for the required treatment and negative cultures. These approved States are California, Colorado, Kentucky, New York, South Carolina, and Virginia.

The regulations were changed to allow the importation of horses from Canada with negative test results for equine infectious anemia (EIA) provided the samples for such tests were drawn within 180 days of the date the horse was offered for importation. Previously, a negative Coggins test for EIA was valid for 60 days prior to export on Canadian horses and 90 days for U.S. horses returning from recognized exhibits or events in Canada.

A new regulation became effective concerning the importation of thoroughbred racehorses from West Germany and standardbred racehorses from Australia. Under this new regulation, such horses from these two countries can be imported under less stringent CEM restrictions provided they are certified to have been in a continuous racing and training status in the country of origin. The Federal veterinarian in the country of origin must also certify that they have never been bred, have not been on any breeding premises, and that the horses are negative for CEM on three sets of swabs collected at 7-day intervals. This regulation also applied to thoroughbred racehorses from the United Kingdom, Ireland, and France.

A new Veterinary Services (VS) policy has been adopted relating to the importation of animals which are affected with dermatomycosis (ringworm) and papillomatosis (warts). Animals previously offered for importation with either ringworm or warts were refused entry. Under the new policy, affected horses or livestock presented at the Canadian or Mexican borders will be refused entry and returned to the country of origin for treatment. Horses or livestock from all other countries will also be refused entry if they have extensive ringworm or wart lesions. Horses or livestock presented for entry at animal quarantine stations with minor ringworm or wart lesions may be treated by a private veterinary practitioner and released from quarantine.

VS has developed a proposed protocol for the importation of swine semen from the People's Republic of China (PRC). This protocol has been circulated to specialists in exotic diseases at Plum Island Animal Disease Center and the Emergency Diseases Program Staff, as well as personnel at the National Veterinary Services Laboratories (NVSL). There has been tentative agreement on the substance of the proposal, and it has now been sent to PRC to see if they will concur in a protocol which involves VS direct supervision of semen collection and processing in their country.

Earlier this fiscal year, VS approved importation of brucellosis-affected, untested heifers to Clint Feedyards at Clint, Texas, on a trial basis. The animals were fed in a quarantine feedlot and had to meet specified conditions for handling by Texas Animal Health Commission
employees. At the conclusion of the first importation and before an assessment had been made of the feasibility of the program, six additional feedyards were approved as an extension of the experiment. Only approximately 800 heifers were brought on the second shipment, part of which went to Clint Feedyards and others went to Seven Rivers Farm and Cattle Company in Caldwell, New Mexico. All of the animals have been returned to Mexico for both trials. In October, there was a request for an additional 15,000 head to come in under similar guidelines. The Deputy Administrator, VS, approved the latter shipment only if the animals were negative to brucellosis test, since he has let it be known that no more shipments of untested heifers would be approved on an experimental basis. The animals now coming from Mexico will be imported more nearly in conformity with Title 9, Code of Federal Regulations, Part 92.35(d). While on-the-farm brucellosis testing is waived, all animals must be negative to brucellosis tests at the U.S. border to qualify for entry. However, as per 92.35(d), only negative animals in the same lot where suspects were found will be permitted entry to Seven Rivers quarantine feedyards. They also must meet the same conditions for handling as the untested heifers did previously.

### Animals Imported:

<table>
<thead>
<tr>
<th></th>
<th>FY 1981</th>
<th>FY 1982 Estimate*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cattle</td>
<td>653,059</td>
<td>703,200</td>
</tr>
<tr>
<td>Swine</td>
<td>145,141</td>
<td>155,500</td>
</tr>
<tr>
<td>Horses</td>
<td>36,533</td>
<td>38,983</td>
</tr>
<tr>
<td>Sheep</td>
<td>10,865</td>
<td>12,300</td>
</tr>
<tr>
<td>Others</td>
<td>35,727</td>
<td>29,845</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>881,325</td>
<td>939,828</td>
</tr>
</tbody>
</table>

### Animals Imported:

<table>
<thead>
<tr>
<th></th>
<th>FY 1981</th>
<th>FY 1982 Estimate*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cattle (Canadian border ports)</td>
<td>339,234</td>
<td>402,000</td>
</tr>
<tr>
<td>Cattle (Air &amp; Ocean ports)</td>
<td>369</td>
<td>369</td>
</tr>
<tr>
<td>Cattle (Mexican border ports)</td>
<td>313,456</td>
<td>320,200</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>653,059</td>
<td>722,569</td>
</tr>
<tr>
<td>Swine (Canadian border ports)</td>
<td>144,852</td>
<td>152,300</td>
</tr>
<tr>
<td>Swine (Air &amp; Ocean ports)</td>
<td>289</td>
<td>302</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>145,141</td>
<td>152,602</td>
</tr>
<tr>
<td>Horses (Canadian border ports)</td>
<td>24,068</td>
<td>26,200</td>
</tr>
<tr>
<td>Horses (Air &amp; Ocean border ports)</td>
<td>10,012</td>
<td>12,000</td>
</tr>
<tr>
<td>Horses (Mexican border ports)</td>
<td>2,453</td>
<td>2,500</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>36,533</td>
<td>40,700</td>
</tr>
</tbody>
</table>

Estimated pending final reports.

* Presented by Dr. Walker
1. An amendment was published on May 19, 1982, allowing a single individual exclusive use of Harry S. Truman Animal Import Center (HSTAIC) if, after a lottery is announced, there are no participants for that lottery or if a lottery is held and the total number of animals retested is less than 50.

2. The General Services Administration is presently negotiating for a site to build a new port facility in Detroit, Michigan.

3. A revised cooperative agreement was published on May 28, 1982, which made it more economical for individuals to import animals through HSTAIC from Europe and Brazil.

4. A final rulemaking, making the preembarkation requirements for cattle from Ireland more rigid, was published on November 1, 1982. This regulation becomes effective December 1, 1982.

5. A lottery for importation of cattle from Brazil and France was published on March 24, 1982. No applications were received; therefore, exclusive use was granted to import 50 head of Limousin cattle from France. The cattle are scheduled to begin quarantine at HSTAIC in mid-December 1982.

ANIMAL PRODUCTS

Personnel of the Import-Export Animals and Products Staff are continuing efforts to draft a revision of Part 95. Initial studies indicated that the task would be more difficult than previously thought. The Office of the General Counsel has assisted with suggestions for changes in format.

Italy is regarded by the U.S. Department of Agriculture as a country affected by foot-and-mouth disease (FMD), African swine fever (ASF), hog cholera (HC), and swine vesicular disease (SVD). None of these diseases occur in the United States.

In 1978, the Animal and Plant Health Inspection Service and the Italian veterinary service entered into an agreement to conduct research on the survival of ASF and SVD viruses during the curing process of Parma hams. It is known that the curing process of these hams will destroy FMD and HC viruses.

In the agreement with the Italians, they were to furnish a special chamber which would control both air temperature and humidity for the experiment. The Italians had agreed to ship the chamber soon after the agreements was entered into. Instead, they procrastinated and the chamber did not arrive in the United States until about November 1, 1981.

Our researchers at Plum Island Animal Disease Center (PIADC), with the assistance of an Italian technician, assembled the chamber and began the research on SVD virus survival about April 1, 1982. This delay in starting the experiment was caused by remodeling work on the facility at PIADC. At the end of the first 40 days of the experiment, there was no...
appreciable reduction in the number of virus particles. The second sampling (90 days) for virus in the hams was made the first week in August. There was some decline in the number of viruses, especially in the fat tissue. The decline of virus numbers in the muscle was less pronounced. The third sampling was taken November 2, 1982. Results will be available before December 1, 1982.

AVIAN IMPORT ACTIVITIES

A. Commercial Birds

1. Two import commercial quarantine facilities in Louisiana and three in Florida have had their approvals removed by VS due to violation of standards. Action to remove the approval of two facilities in California, one in New York, and one in Illinois has been initiated.

2. A proposal has been initiated to increase the space reservation fee from $40 to $80 for poultry and birds imported through USDA operated quarantine stations.

3. As a result of meetings between USDA and representatives from the commercial bird industry during 1982, a number of significant changes occurred in the program. These changes are as follows:

   a. Birds from specific countries will no longer be embargoed for 90 days following disclosure of viscerotropic velogenic Newcastle disease (VVND) infection in birds imported from that country. USDA will continue to notify the country of origin, but the 90-day ban will not be reinstated unless the disease appears to be pandemic.

   b. USDA accounting procedures will be changed, resulting in importers being billed for each quarantine period. Previously, importers were billed monthly which resulted in much confusion.

   c. Psittacosis preventative treatment was broadened and now bird station operators may feed medicated feed pellets in addition to the cooked ration recommended by U.S. Public Health Service for psittacine birds in quarantine.

   d. Commercial stations may move within the same port servicing area if the Area Veterinarian in Charge approved the site and the standards. No new stations can be approved because USDA does not have enough supervisory personnel and plans to further reduce personnel.

   e. USDA now requires banding of imported psittacine birds with stainless steel, coded bands. Small psittacines (budgerigar, cockatiel, and smaller) may use approved aluminum or plastic bands because 1/4” stainless steel bands are too large. The attempt to band psittacine birds moving interstate from California was rescinded for lack of support. Part 60 (banding of psittacine birds moving in commerce) is written but is on hold for possible emergency use.

   f. A total of 57.87 man-years of work for FY 1982 was less than
IMPORT—EXPORT

utilized by USDA in FY 1981. Despite the decrease in man-years, a greater number of birds were imported in FY 1982.

B. Pet Bird Program

1. Action has been taken to increase rates from $80 to $100 for a single pet bird and $125 for two pet birds. The increased rates will take effect on January 1, 1983, and should help reduce the deficit in the program.

2. A regulation is now being promulgated to remove Brownsville, Texas, as a pet bird quarantine station. The quarantine trailer used at Brownsville is now being used in San Ysidro, California, for smuggled birds.

3. USDA has been informed that the Mexican Government will ban the export of birds through land ports. Certain species may be exported by permit only.

C. Smuggled Bird Program

1. A proposal for a permanent facility in Mission, Texas, to replace an obsolete building has gone out for bids and a permanent facility at Otay Mesa, near San Ysidro, California, may be built to replace temporary trailers now in use at that site.

2. A Memorandum of Understanding has been signed by USDA, U.S. Department of the Interior, U.S. Customs Service, and the U.S. Department of Justice to seize and quarantine illegal entries of wildlife animals and birds. USDA responsibility will be to quarantine and test birds for poultry diseases (such as VVND). Quarantine costs, along with seizure and transportation expenses, will be pro-rated among the various Agencies involved in the operation.

D. Poultry and Hatching Eggs

1. New hatching egg requirements will reduce the amount of time USDA has to devote to supervising importations. This will be accomplished through a reduced number of inspections.

2. USDA will continue to require foreign origin poultry flocks to be tested for adenovirus 127.

3. A regulation to require that flocks of origin be Mycoplasma gallisepticum and M. synoviae-free will be promulgated.

4. VVND-free status was denied the United Kingdom because of deficiencies in their pet and commercial bird programs.

5. Hatching eggs (but not live poultry) from VVND-free countries may enter the United States without having to undergo quarantine as long as no other significant poultry diseases exist in those countries.
### COMMERCIAL BIRD QUARANTINE IN PRIVATE STATIONS

<table>
<thead>
<tr>
<th>Number of Quarantines</th>
<th>Birds</th>
<th>DOA</th>
<th>Died in Quarantine</th>
<th>Total Released</th>
<th>WND Refused Entry</th>
<th>Other Refused Entry</th>
</tr>
</thead>
<tbody>
<tr>
<td>FL</td>
<td>70</td>
<td>179951</td>
<td>8241</td>
<td>20720</td>
<td>1</td>
<td>11916</td>
</tr>
<tr>
<td>CA</td>
<td>105</td>
<td>260212</td>
<td>15074</td>
<td>41480</td>
<td>5</td>
<td>4344</td>
</tr>
<tr>
<td>HI</td>
<td>4</td>
<td>16567</td>
<td>40</td>
<td>1832</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>IL</td>
<td>25</td>
<td>213208</td>
<td>29236</td>
<td>69808</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>LA</td>
<td>16</td>
<td>7830</td>
<td>203</td>
<td>1914</td>
<td>1</td>
<td>132</td>
</tr>
<tr>
<td>MI</td>
<td>5</td>
<td>13082</td>
<td>93</td>
<td>2911</td>
<td>0</td>
<td>2033</td>
</tr>
<tr>
<td>NY</td>
<td>22</td>
<td>74792</td>
<td>2825</td>
<td>8557</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>TX</td>
<td>4</td>
<td>4994</td>
<td>54</td>
<td>149</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>WA</td>
<td>4</td>
<td>1162</td>
<td>1</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOTALS</td>
<td>255</td>
<td>771798</td>
<td>55676</td>
<td>147381</td>
<td>8</td>
<td>18425</td>
</tr>
</tbody>
</table>

*Vesicant Newcastle Disease (WND)*
### PET BIRDS

<table>
<thead>
<tr>
<th>Number of Quarantines</th>
<th>Total Received</th>
<th>WND Refused Entry</th>
<th>Other Refused Entry</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Birds</td>
<td>DOA</td>
<td>Died in Quarantine</td>
</tr>
<tr>
<td>SAN YSIDRO</td>
<td>89</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>LOS ANGELES</td>
<td>628</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>NEW YORK</td>
<td>574</td>
<td>7</td>
<td>30</td>
</tr>
<tr>
<td>MIAMI</td>
<td>800</td>
<td>4</td>
<td>42**</td>
</tr>
<tr>
<td>HONOLULU</td>
<td>63</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>BROWNSVILLE</td>
<td>41</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>EL PASO</td>
<td>46</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>LAREDO</td>
<td>37</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>NOGALES</td>
<td>12</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>2290</td>
<td>11</td>
<td>91</td>
</tr>
</tbody>
</table>

* Velogenic Newcastle Disease (WND)

**28 finches
## FY 1982

### USDA Facilities

<table>
<thead>
<tr>
<th>Number of Quarantines</th>
<th>Total Received</th>
<th>VND Refused Entry</th>
<th>Other Refused Entry</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Birds</td>
<td>DOA</td>
<td>Died in Quarantine</td>
</tr>
<tr>
<td>NY</td>
<td>99</td>
<td>4661</td>
<td>66</td>
</tr>
<tr>
<td>HI</td>
<td>17</td>
<td>1012</td>
<td>14</td>
</tr>
<tr>
<td>FL</td>
<td>33</td>
<td>619</td>
<td>1</td>
</tr>
<tr>
<td>Totals</td>
<td>149</td>
<td>6282</td>
<td>81</td>
</tr>
</tbody>
</table>

*Venezigenic Newcastle Disease* (VND)
<table>
<thead>
<tr>
<th>COUNTRY OF ORIGIN</th>
<th>VISCOGENIC VELOCENIC NEWCASTLE DISEASE (VND) - INFECTED LOTS IN FY 1982</th>
</tr>
</thead>
<tbody>
<tr>
<td>ARGENTINA</td>
<td>1</td>
</tr>
<tr>
<td>BELGIUM</td>
<td>1</td>
</tr>
<tr>
<td>HONDURAS</td>
<td>3</td>
</tr>
<tr>
<td>INDONESIA</td>
<td>1</td>
</tr>
<tr>
<td>MALAYSIA</td>
<td>2</td>
</tr>
<tr>
<td>MEXICO</td>
<td>2</td>
</tr>
<tr>
<td>EL SALVADOR</td>
<td>1*</td>
</tr>
<tr>
<td>INDONESIA</td>
<td>1*</td>
</tr>
<tr>
<td>MEXICO</td>
<td>1*</td>
</tr>
</tbody>
</table>

*Velegenic Newcastle Disease (VND)
### Imported Birds (Commercial)

#### Summary

<table>
<thead>
<tr>
<th>FY Period</th>
<th>Lots (Birds) Released</th>
<th>Lots (Birds) Refused Entry</th>
<th>Program Cost Per Bird ($)</th>
<th>Program Cost Total ($)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1974</td>
<td>13 (27,696)</td>
<td>6 (18,969)</td>
<td>9.23</td>
<td>255,627</td>
</tr>
<tr>
<td>1975</td>
<td>71 (124,597)</td>
<td>19 (30,446)</td>
<td>7.27</td>
<td>906,313</td>
</tr>
<tr>
<td>1976</td>
<td>179 (222,922)</td>
<td>24 (47,943)</td>
<td>6.27</td>
<td>1,399,307</td>
</tr>
<tr>
<td>1977</td>
<td>276 (313,537)</td>
<td>16 (35,197)</td>
<td>6.02</td>
<td>2,000,000</td>
</tr>
<tr>
<td>1978</td>
<td>409 (520,725)</td>
<td>12 (28,770)</td>
<td>4.41</td>
<td>2,300,000</td>
</tr>
<tr>
<td>1979</td>
<td>406 (341,174)</td>
<td>36 (32,296)</td>
<td>2.03</td>
<td>693,108</td>
</tr>
<tr>
<td>1980</td>
<td>428 (591,375)</td>
<td>15 (6,810)</td>
<td>1.53</td>
<td>907,786</td>
</tr>
<tr>
<td>1981</td>
<td>471 (518,472)</td>
<td>12 (21,182)</td>
<td>1.90</td>
<td>988,846</td>
</tr>
<tr>
<td>1982</td>
<td>394 (554,321)</td>
<td>10 (19,145)</td>
<td>2.50</td>
<td></td>
</tr>
</tbody>
</table>
IMPORT—EXPORT

EXPORT ANIMALS

Two shipments of swine totaling 829 head were exported to PRC. A delegation of Chinese officials visited the United States to negotiate on a one-shipment basis the health conditions of these animals. Further discussions are to follow to negotiate future health agreements for swine, horses, and cattle.

The budget for Export Animals for FY 1983 totaling $1.4 million was deleted from the Import-Export budget. User fees were to be instituted by legislation to replace this appropriation. This legislation has yet to be enacted by Congress.

A new export facility has been approved at Minneapolis, Minnesota. Station reviews for the Export Animal Health Program were held in Missouri, New York, and Wisconsin.

Swine exports are continuing to increase and are becoming a major segment of our livestock export industry.

VS has communicated and is negotiating with the European Economic Community (EEC) on health requirements for the exportation of cattle to member countries. Bluetongue (BT) and enzootic bovine leukosis are the diseases of concern. VS has proposed States in the Northeast to be declared free of BT, and animals be shipped during the nonvector season. Discussions are continuing on the health requirements.

The outbreak of vesicular stomatitis in the United States has caused an impact on exports of livestock. Taiwan has imposed a 6-month ban on the importation from the United States of cattle, horses, swine, deer, sheep, goats, and semen. Other countries have requested that the animals be tested serologically negative and originate from States where no cases of vesicular stomatitis have been reported.

Embryos for both cattle and swine are being exported in significant numbers and are being incorporated for the first time in the statistics for animals exported.

<table>
<thead>
<tr>
<th>Animals Exported</th>
<th>FY 1981</th>
<th>FY 1982</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cattle</td>
<td>134,847</td>
<td>170,325</td>
</tr>
<tr>
<td>Porcine</td>
<td>24,887</td>
<td>187,788</td>
</tr>
<tr>
<td>Sheep</td>
<td>182,102</td>
<td>288,951</td>
</tr>
<tr>
<td>Goats</td>
<td>70,236</td>
<td>82,859</td>
</tr>
<tr>
<td>Horses</td>
<td>28,688</td>
<td>82,587</td>
</tr>
<tr>
<td>Total</td>
<td>440,760</td>
<td>812,510</td>
</tr>
<tr>
<td>Bovine embryos</td>
<td></td>
<td>533</td>
</tr>
<tr>
<td>Porcine embryos</td>
<td></td>
<td>160</td>
</tr>
</tbody>
</table>

Dr. Ronald B. Caffey gave the following report on the activities of Plant Protection and Quarantine.
UNDERCOOKED PERISHABLE CANNED PORK PRODUCTS

During Fiscal Year 1982, port of entry sampling of shipments from six different establishments in three countries: Denmark, Holland, and Poland, indicated undercooked product. Laboratory tests were conducted by Food Safety and Inspection Service Laboratories.

IMPROPER HANDLING OF FOREIGN ORIGIN GARBAGE

*Airports*—Approval (Compliance Agreements) was withdrawn from one airport catering establishment due to improper handle of foreign garbage: As of September 30, 1982, approval withdrawal actions are pending for two additional firms.

*Maritime*—Improper handling of foreign origin garbage cases aboard *four* different vessels at San Juan, Puerto Rico, were successfully prosecuted.

PREPARATION FOR 1984 OLYMPICS

Both the Los Angeles and International Olympic Committees have been furnished copies of “Travelers Tips” and additional specific information relative to APHIS animal, animal product, and plant materials import requirements. These committees will include this information in material which they are providing to travel agencies, participants, etc.

QUARTERLY INSPECTION CHECKLIST

A form has been developed with input from the catering industry to facilitate inspection of catering establishments which handle foreign garbage at airports. Incorporation of the use of this form into existing inspection activities will begin early in fiscal year 1983 and is expected to increase uniformity of inspection procedures, as well as improve communication with catering establishment corporate headquarters.

MODIFIED PASSENGER INSPECTION SYSTEMS

Modified passenger inspection programs are in effect at Miami, Boston, and Los Angeles airports which are considered to be a variation of the original Agricultural Primary Screening Inspection System (APSIS) concept. In San Juan and Chicago, the APSIS system is in operation. At present, discussions are being held with airline officials at New Orleans and the British Airways Terminal at John F. Kennedy International Airport regarding accelerated passenger inspection systems. The Animal and Plant Health Inspection Service (APHIS) is committed to expanding modified systems when requested by airport management, and proposed modifications will ensure that such systems not reduce agricultural enforcement. In these cases the cost of modification is paid for by the airport management. Under the Accelerated Specialized Inspection System Test (ASIST), a Customs or Immigration inspector performs all inspection functions at the first or *primary* station. If the primary inspector believes a closer inspection or additional processing is warranted, he directs the passenger to the *secondary* inspection station.
after the passenger has collected his luggage. Secondary Agricultural inspection is completed and performed by Plant Protection and Quarantine (PPQ) employees.

Under APSIS, the PPQ officer works in the passenger lines in front of the U.S. Customs Service' primary booths. Passengers are assessed and questioned by the PPQ officer using an agricultural risk profile. Passengers with such forms coded "no risk" are not queried for PPQ interest at primary, and 100 percent hand baggage inspection is not required. Declarations of high risk passengers are so coded and these passengers are directed to secondary agricultural inspection.

Fiscal Year 1982 Report of Animal Products Imported/Exported

STATISTICS

Vessel and aircraft arrival

- 39,079 vessels were boarded and inspected
- 1,879 lots consisting of 1,503,871 kg of garbage were removed from these vessels
- 5,551 garbage handling discrepancies were corrected
- 206,877 aircraft arrived from foreign locations
- 7,829,065 kg of garbage was removed from these aircraft

Meat and animal products refused entry/confiscated

- Ship passenger baggage
  - 197 lots
  - 1,098 kg
- Aircraft passenger baggage
  - 66,500 lots
  - 78,600 kg
- Border crossing
  - 20,421 lots
  - 21,756 kg
- Post office
  - 6,781 lots
  - 14,088 kg

Commercial Meat Shipments

- Beef
  - 696 lots
  - 156,047 kg
- Pork
  - 6,098 lots
  - 467,300 kg
- Poultry
  - 10 lots
  - 3,031 kg

Meat and meat products checked

- 33,552 lots
- 392,337,773 kg

Shoe Cleaning and Disinfection

- 57,151 were inspected
- 8,740 were cleaned and disinfected

Export Certification

- 17,284 lots
- 701,753,689 kg

Animal Products Imported

- 2,584 lots
  - 36,415,871 kg
  - Restricted entry
- 83,655 lots
  - 286,007,688 kg
  - Unrestricted entry
- 270 lots
  - 570,807 kg
  - Refused entry

CIVIL PENALTIES BILL (H.R. 6679)

Legislation to impose civil penalties for violation of APHIS plant and animal quarantine regulations has passed the House and was referred to
the Agriculture Sub-Committee Agricultural Research and General Legislation, chaired by Senator Richard G. Lugar of Indiana. This is a sub-committee of the Senate Agriculture, Nutrition and Forestry Committee, which is chaired by Senator Jesse A. Helms. The Bill authorizes the Secretary of Agriculture to assess civil penalties after notice and opportunity for an agency hearing for an amount up to $1,000. The maximum criminal fine for knowingly violating APHIS regulations is increased to $5,000.

The fate of this legislation will be decided during final sessions of the 97th Congress which begin November 29.

Dr. Harold A. Waters gave the report of the Export Subcommittee which is included as Appendix I.

Dr. Jerry S. Walker presented a report on research by ARS in support of import and export of animals and animal products which is included as Appendix II.

Dr. Walker also presented a paper (AAVLD) on the weak gamma sterilization for both import and export items, the abstract is included as Appendix III.

The following are recommendations of the committee:
1. APHIS continue negotiations with Peoples Republic of China on health agreements for swine, horses and cattle.
2. APHIS continue negotiating with the European Economic Community (EEC) on health requirements for the exportation of cattle to member countries.
3. Urge passage of Civil Penalties Bill (H.R. 6679) on violations of APHIS plant and animal quarantine regulations.
4. Establish a subcommittee on embryo transfer health certification and disease research, or provide adequate time in Import-Export Committee schedule.
5. APHIS confer with livestock and poultry associations in order to revise US Origin Health Certificates for Uniformity.

The committee considered the following resolutions:
1. Require negative BTV test of all ruminants entering the US from Mexico. Moved by Mr. Maurice Mix. Died for lack of a second.
2. Support of APHIS in their stand to protect the domestic livestock industry and this nation's wildlife resources from disease both domestic and foreign as their No. 1 priority. Moved by Mr. Bert Hawkins. Seconded by Mr. Jack Dahl. Passed unanimously.
3. Increase federal funding for embryo research. Moved by Dr. Brent Perry. Seconded by Mr. Frank Harding. Passed unanimously.

These resolutions have been presented to the Resolutions Committee. There being no further business, the meeting was adjourned.
IMPORT—EXPORT

APPENDIX I

COMMITTEE ON IMPORT-EXPORT
EXPORT SUBCOMMITTEE

The first meeting of the Export Subcommittee met at 1:30 pm November 9, 1982. Twenty-three USAHA members were present, nine of which were subcommittee members. Twelve persons were from industry, eleven represented government.

Dr. Allen E. George, Veterinary Services, APHIS, reported on the Export Animal Health Program. His report is enclosed. Dr. Jerry S. Walker, ARS, USDA, reported on research in USDA affecting export animal health certification. Dr. Brent Perry, American Embryo Transfer Association, reported on embryo transfer technology. Dr. Douglas Hare, Canadian Animal Disease Research Institute reported on embryo transfer disease research.

It is recommended that an embryo transfer subcommittee be established within the committee on Import-Export to deal with embryo transfer health certification and disease research.

The subcommittee reviewed origin health certificates. They recommended that Veterinary Services, APHIS, develop uniform health certificates for animals and poultry exported from the United States. Veterinary Services has agreed to confer with livestock and poultry associations in order to revise US Origin Health Certificates for uniformity. Veterinary Services is preparing import animal health requirements of other countries on their computers to maintain current listing of requirements for VS and exporter use.

Interpretation of import health requirements of other countries by US and Canada animal health officials was discussed. The Subcommittee asked VS for regular consultation by VS with Canadian health officials to maintain uniformity of health certification. Discussion was also held by the Export Subcommittee on retest procedures on animals exported. Uniform testing procedures are essential to avoid retest errors.

Brucellosis vaccination of cattle with Strain 19 was discussed. Official calfhood vaccination is restricting exportation of U.S. cattle. Both Eastern and Western European countries prohibit importation of cattle from herds in which Strain 19 vaccine has been used within the past two years. Korea also discriminates against brucellosis vaccinates. VS was requested to provide background information on Strain 19 vaccine usage in the United States to veterinary officials of these countries to aid in removing these trade barriers.

Infectious Bovine Rhinotracheitis (IBR) vaccine, live virus origin, also, has caused similar trade barriers. Use of killed virus IBR vaccine is encouraged for immunizing cattle for export.

The Export Subcommittee reviewed the Export Meat Certification Program. The impact of Salmonella testing of meat and poultry in im-
reporting countries was recognized. Elimination of the source of Salmonella in meat in the U.S. is needed. Most Salmonella contamination originates from the meat protein supplements in animal feed. The Subcommittee encourages U.S. Government funding of research on Salmonella toward eliminating this contaminant in animal feed.

Dr. John Atwell reported to the Subcommittee on the O.I.E. Zoosanitary Commission meeting in Paris in October 1982. The recommended certification standards for bluetongue, leukosis, and tuberculosis of cattle have been modified. These modifications will allow U.S. exports under these standards. Frozen embryo health recommendations have been drafted and will be distributed for comment. Veterinary Services will seek Export Subcommittee comments on this draft.

Diagnostic laboratory service for export animal health certification was discussed. Development of full service veterinary diagnostic laboratories in exporting states is needed to replace reduced service at National Veterinary Services Laboratories at Ames, Iowa. Local diagnostic testing service to facilitate exports is desired by exporters.

The meeting was adjourned at 4:45 pm.

Respectfully submitted,

Harold A. Waters, Chairman

APPENDIX II

RESEARCH BY THE AGRICULTURAL RESEARCH SERVICE IN SUPPORT OF EXPORT AND IMPORT OF ANIMALS AND ANIMAL PRODUCTS

H. G. Purchase, J. S. Walker,* and R. H. Alsmeyer

U.S. Department of Agriculture, Agriculture Research Service, National Program Staff, Beltsville, Maryland, and Plum Island Animal Disease Center, Greenport, New York

INTRODUCTION

Agricultural exports contribute greatly to the balance of trade between the United States and other countries. Exports of animals and animal products from the United States have risen from just over $1 billion in 1972 to over $4 billion in 1981. Of this, $2 million is for exports of live animals and $2 billion is exports of meats and meat products. These exports could be jeopardized by the outbreak of particular infectious animal diseases in the United States. For example, the $13 million poultry export trade to the United Kingdom was recently halted because authorities in the United Kingdom declared that it is free of Newcastle disease and thus would not accept poultry or poultry products from countries with Newcastle disease or from countries that vaccinate against this disease. Other diseases, for example, vesicular stomatitis (which broke out recently in the Rocky Mountain states) and the oc-
currence of *Salmonella* in our meats (particularly poultry meat) and meat products jeopardize these exports. In addition, the exports could be expanded if the United States could meet import requirements of certain foreign countries for diseases such as bluetongue and bovine leukemia.

Imports of animals and animal products into the United States amounted to $1.6 billion in 1972 and $3.7 billion in 1981. Like the countries to which we export animals and animal products, the United States has import restrictions to keep out diseases that could infect humans and animals. Many of these diseases, such as foot-and-mouth disease, African swine fever, or fowl plague, could devastate animal agriculture in the United States if they entered the animal population.

The primary mission of the Agricultural Research Service (ARS) is research. ARS scientists study all aspects of agriculture—soil, water, and air, crop pests, crop production, animal science, human nutrition, and post-harvest technology. ARS is also responsible for the diagnosis of foreign animal diseases in samples taken from animals and animal products imported into the United States and from suspected cases in the United States. In this paper, we describe the research conducted by ARS scientists in support of export and import of animals and animal products. We have also made a rough estimate of the proportion of each project (and thus the funds expended on each project) that directly supports the importation or exportation of animals and animal products.

**RESEARCH IN SUPPORT OF EXPORTS**

*Research to allow U.S. animals and products to meet import requirements of foreign countries*

Many countries ban imports from the U.S. because diseases that occur in the U.S. do not occur or are not known to occur in those countries. Diseases that are major barriers to export of our animals and animal products are bluetongue, epizootic hemorrhagic disease, bovine leukemia, salmonellosis, and Newcastle disease. Research in ARS is directed towards methods for certifying that animals or animal products are free of these diseases.

Research on bluetongue includes development of new methods of primary isolation and detection of the virus, and elucidation of the mechanism of latent infection and persistence in animals and of transmission in sperm and across the placenta in cattle and sheep. Studies on the ability of the *Culicoides* vectors to transmit the virus and on integrated management procedures to obtain area-wide control of *Culicoides* are also ongoing. Some comparative research is also being undertaken on epizootic hemorrhagic disease. These two diseases are of increasing importance to our export trade, as the recent exports of cattle to Australia demonstrated. The Office International des Epizooties (OIE) recommendations are very specific in regard to bluetongue and epizootic hemorrhagic diseases and are being widely adopted by developing countries.
Research on Newcastle disease emphasizes new methods to detect the virus in carrier birds and better methods to immunize against the disease. New vaccines are being developed against avian influenza.

Research on bovine leukemia includes comparison of methods for identification of bovine leukemia virus-infected cattle and development of a radioimmunoassay test for the virus. Studies continue on the importance of vertical transmission and on clarifying factors affecting horizontal transmission of the virus. The influence of genetic factors on the interaction of bovine leukemia virus with the host are also being studied and the effectiveness of an inactivated vaccine is being evaluated under field conditions. The studies are directed towards identification and elimination of bovine leukemia viruses from domestic cattle and certification of herds free of infection. Animals from such herds could be exported to countries that claim to be free of the disease.

Certain countries claim to be free of salmonella infections and may ban the importation of salmonella-contaminated meat. Research to control salmonella on meat and poultry products attacks the problem at the production, processing, and product levels. Ways to prevent the spread and transmission of salmonella infections in birds—fumigation of the eggs and heating poultry feed—and ways to reduce the level of salmonella infection by exposure of newly hatched chicks to the normal bacterial flora of the gut are being developed and evaluated. Better assay and sampling techniques are being developed and changes in chilling procedures, including the use of various sanitizers, are being examined. Similar research on Campylobacter is increasing, with major emphasis on determining the survival of the organisms in meat and developing techniques to control the pathogen.

Research to improve productivity of exported livestock

Research on understanding the immunity and developing vaccines to Babesia species and some of the research on detection, vaccination, chemotherapy, and prophylaxis of anaplasmosis is to improve the survival and productivity of exported breeding stock. Similarly, work conducted on immunity and vaccination against East Coast fever falls in this category. One might also consider a very small portion of the research on foot-and-mouth disease vaccines, for development of such a vaccine would improve productivity of exported U.S. livestock.

Research on automated testing methods and development of reference biologicals could assist in the diagnosis and prevention of disease in exported livestock.

Research to maintain quality of exported animal products

Study of export meat products, particularly beef and pork hearts, tongues, and livers is concentrated on proper chilling, packaging, and handling to avoid microbial deterioration and physical damage. Test shipments of these meat offal products are being made to Europe, particularly to France, The Netherlands, and the United Kingdom. These
tests have provided information that has been incorporated into several brochures now being used by the meat export industry to improve procedures and packaging to insure that meat products delivered to importing countries are sound and of high quality.

Emergency Preparedness for Far East Market Problems

A USDA study requested by Congress recommended a two-phase plan of action for increasing research responsiveness to problems associated with all agricultural exports to the Far East. Under the leadership of ARS, six agencies (ARS, the Foreign Agricultural Service, the Animal and Plant Health Inspection Service, the Office of Transportation, the Federal Grain Inspection Service, and the Food Safety and Inspection Service) are participating in the newly formed interagency response management group to carry out recommendations contained in the study. The group will develop and implement an interagency plan for early warning and response to problems as they begin to emerge, and they will develop and implement a research plan to avoid problems.

RESEARCH IN SUPPORT OF IMPORTS

Research to assure that foreign animals and products meet import requirements of the United States

The major research and diagnostic efforts on foreign animal diseases are to prevent the introduction of the foreign animal disease agents into this country. Diagnostic tests have been developed and diagnostic competence is maintained for 44 different foreign animal diseases, including foot-and-mouth disease, African swine fever, heartwater, trypanosomiasis, and exotic serotypes of bluetongue. Reagents have been produced and stockpiled in the event of an emergency. Methods of destroying the virus in the environment and in biologic substances have been identified. Large numbers of samples from domestic animals and animals imported for zoologic parks are tested for the presence of a wide spectrum of foreign animal agents.

The Parma ham project, a collaborative effort of the United States and Italy, is another important research endeavor. The persistence of two viruses, swine vesicular disease (SVD) and African swine fever (ASF), is being determined in hams cured by the parma process. To date, SVD virus has been isolated through 90 days. The experiment is to run for 15 months.

Research to insure safety of imported food products from animals

Research is ongoing to develop techniques to identify the highest temperature attained in the inside of beef roasts and processed meat for soup stocks to that we can be assured that foreign animal disease agents and food poisoning organisms have been inactivated.

Training

Considerable efforts at training domestic and foreign scientists at the Plum Island Animal Disease Center are undertaken each year. Because
there is no way to be absolutely sure that imported animals and animal products never contain exotic animal disease agents, scientists in the United States must be prepared to identify disease agents that are inadvertently introduced into the country. Similarly, training in the diagnosis of foreign animal diseases is important to scientists in countries near the United States from which diseases could be introduced into the United States.

OTHER RELATED RESEARCH

Work on the identification of animals carrying contagious equine metritis and equine infectious anemia, which relates to both the export and import of animals, has been recently terminated. Work done primarily for domestic purposes, including research on brucellosis, tuberculosis, Johne's disease, and leptospirosis, is also important to export and import of animals.

In an ongoing program and a collaborative effort with Canada the transmission of foreign and domestic animal diseases (including foot-and-mouth disease, bovine viral diarrhea, bluetongue, bovine leukemia, and the porcine parvovirus infection by embryos) is being examined. Embryo transplantation will likely be the major route of exchange of germplasm between countries in years to come. The findings of these embryo transplantation studies will be used by the United States and other countries to formulate import regulations.

DISCUSSION AND CONCLUSIONS

The Agricultural Research Service is very much aware of the needs of U.S. animal industries to export animals and animal products to foreign countries and to import germplasm from certain foreign countries, and of the wishes of many foreign countries to export their animals and animal products to the United States. At the Agricultural Research Service, we have an ongoing program of research which I have described, that supports import and export of animals and animal products and we have developed a plan to respond with research to any researchable problems that might occur.
<table>
<thead>
<tr>
<th>Projects</th>
<th>Research support FY 1982 est. ($000)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Import</td>
</tr>
<tr>
<td>Domestic Diseases</td>
<td></td>
</tr>
<tr>
<td>Anaplasmosis</td>
<td>468</td>
</tr>
<tr>
<td>Bluetongue</td>
<td>28</td>
</tr>
<tr>
<td>Bovine leukemia</td>
<td>116</td>
</tr>
<tr>
<td>Salmonellosis</td>
<td>8</td>
</tr>
<tr>
<td>Foreign Animal Diseases</td>
<td></td>
</tr>
<tr>
<td>Foot-and-mouth disease</td>
<td>220</td>
</tr>
<tr>
<td>African swine fever</td>
<td>278</td>
</tr>
<tr>
<td>Newcastle disease and avian influenza</td>
<td>116</td>
</tr>
<tr>
<td>Rotavirus infections</td>
<td>8</td>
</tr>
<tr>
<td>Equine encephalomyelitis</td>
<td>142</td>
</tr>
<tr>
<td>Babesiosis</td>
<td>10</td>
</tr>
<tr>
<td>East Coast fever</td>
<td>1,939</td>
</tr>
<tr>
<td>Trypanosomiasis</td>
<td>15</td>
</tr>
<tr>
<td>Decontamination</td>
<td>478</td>
</tr>
<tr>
<td>Diagnosis in General</td>
<td></td>
</tr>
<tr>
<td>Food &amp; Quality Safety of Meat and Poultry Products</td>
<td></td>
</tr>
<tr>
<td>Salmonella processing</td>
<td>705</td>
</tr>
<tr>
<td>Campylobacter</td>
<td>107</td>
</tr>
<tr>
<td>Microbial and physical deterioration and packaging of meat offal exports</td>
<td>73</td>
</tr>
<tr>
<td>Temperature to which meat was cooked</td>
<td>221</td>
</tr>
<tr>
<td>TOTAL</td>
<td>4,024</td>
</tr>
</tbody>
</table>
Biological diagnostic reagents that have been produced in the high containment laboratories at the PIADC have traditionally been “safety-tested” before the reagents are released to other laboratories. The safety test typically consists of extensive sampling and inoculation of susceptible tissue cultures, small laboratory animals and large domestic animals. Serum neutralization studies, clinical observations and other appropriate tests are conducted over a 21-day period. This safety test is therefore time-consuming, expensive and cumbersome.

Studies conducted at the PIADC during the past few years have demonstrated the efficacy of exposing diagnostic reagents to gamma radiation. The inactivation of all possible microorganisms is readily achieved, thereby assuring a sterile product. Extensive exposure determinations were done to establish appropriate dose levels and conditions for radiation.

Routine treatments are now given at 6 mega rad exposure, keeping the materials frozen at <56C or lower. This dose gives at least a two log or 100-fold margin of safety. We include known quantities of SVDV as a biological indicator (similar to spore strips for autoclave) and do not release the irradiated materials until after the exposed virus sample is shown to be inactivated.

INTRODUCTION

There have been many definitions of surveillance, varying from simple to complex, but each has been incomplete in some respects. With the assistance of colleagues and students of epidemiology, the author has developed a new definition which offers a broader concept of surveillance. This definition also attempts to be more complete by identifying components that should be used as criteria in evaluating and designing any surveillance effort, whether simple or complex.

The proposed definition:

"SURVEILLANCE IS A DYNAMIC PROCESS TO PROVIDE DATA FOR ANALYZING AND INTERPRETING THE OCCURRENCE OF HEALTH RELATED EVENTS IN A DEFINED POPULATION AS INFLUENCED BY HOST-AGENT-ENVIRONMENTAL FACTORS. THE PROCESS MAY UTILIZE SEVERAL ALTERNATIVES, REQUIRING ONE OR A COMBINATION OF METHODS AND TECHNOLOGIES, BASED ON THE PURPOSES, THE DEGREE OF CONFIDENCE DESIRED FOR THE RESULTS, THE PROBLEMS AND THE AVAILABLE RESOURCES. HEALTH RELATED EVENTS INCLUDE NOT ONLY CLINICAL DISEASE BUT ALSO SUBCLINICAL CONDITIONS AND SEROLOGIC, GENETIC, MICROBIOLOGIC, PATHOLOGIC AND BIOCHEMICAL DATA AS WELL AS DATA TO AID IN DETERMINING OUTCOMES SUCH AS DEATH, ILLNESS, DISABILITY, PRODUCTIVITY AND BENEFIT/COST RATIOS."

This definition attempts to provide a framework within which one can identify and characterize surveillance efforts. It also provides criteria which should be of assistance in evaluating and designing many types of surveillance to accomplish specific purposes within specific situations for agent-host-environment. This definition promotes the concept of specific surveillance plans for specific purposes. It discourages the concept that surveillance consists of one large monolithic system that uses the same methods, the same technology and the same population to fulfill all purposes and all needs. One monolithic national system of surveillance is not a panacea; is not sensitive to differences in the epidemiology of health related events; and certainly, is not sensitive to the differences in benefit/cost ratios as affected by the need for, and the barriers to, obtaining many different kinds of surveillance data.
EPIDEMIOLOGIC FACTORS

In designing and evaluating surveillance programs for one or several health related events, planners and administrators must continually utilize at least 8 epidemiologic factors as criteria for evaluation. These epidemiologic factors are listed in Table 1.

TABLE 1
EPIDEMIOLOGIC FACTORS AFFECTING DESIGN AND EVALUATION OF SURVEILLANCE PROGRAMS
1. PURPOSES-OBJECTIVES
2. STATISTICAL
3. BIOLOGIC (HOST-AGENT-ENVIRONMENT)
4. ECONOMIC (BENEFIT-COST)
5. SOCIAL-CULTURAL
6. EDUCATION-MOTIVATION
7. POLITICAL-LEGAL
8. TECHNOLOGIC

PURPOSES—OBJECTIVES

Purposes and objectives should not be global, should be as specific as possible and should serve the defined and prioritized needs of a group that will actually use the results of the surveillance. Too often we collect data just because it seems like a good idea, not because it will be used. And even if it will be used, the degree of need for these results should be prioritized by evaluating all factors including benefit/cost for collection as well as use.

STATISTICAL FACTORS

Statistical factors that must be considered relate to sources of data, methods of collection, tabulation, analysis and the ability to develop statistically valid results. This includes appropriate characterization of a numerator (the health related event) and a denominator (a defined population at risk of reporting).

BIOLOGIC FACTORS

Biologic factors include the interactions of the host, the agents, and the environmental factors influencing the health related event. For example, surveillance of Western encephalitis should be sequential. It should start with surveillance for measuring favorable weather patterns, followed by monitoring for significant increases in mosquito populations, followed by sampling mosquitos for WEE virus, followed by sampling nestling birds for virus and/or seeking reports of clinical cases in people. Clinical cases in horses serve as an excellent sentinel since equine cases usually begin to occur about 2 weeks before the first cases occur in humans. In contrast to sequential surveillance, effective surveillance of foot and mouth disease should be designed to emphasize early and accurate detection of
the first clinical cases in susceptible animals. Therefore, FMD surveillance depends primarily on early reporting of any vesicular disease by the practicing DVM.

A third example is surveillance for brucellosis which is a chronic infection that depends primarily on appropriate serologic testing of animals to detect this largely subclinical infection. A system for detecting only the clinical cases is not sufficient nor effective.

A fourth example is bovine tuberculosis. Although bovine tuberculosis is a chronic infection, there are pathologic lesions in the tissues without clinical disease until several months prior to the death of the animal. For this infection, a national on-farm surveillance system is not appropriate since (1) the prevalence of the disease is very low and (2) owners tend to send infected animals to slaughter, prior to or at first signs of any clinical signs of the infection. Surveillance by on-farm tuberculin testing is no longer appropriate because of the low prevalence of the infection and inadequate specificity of the test. However, a program of surveillance for lesions during slaughter followed by microbiologic confirmation has been very successful in detecting infection and has an excellent benefit/cost ratio. This is accompanied by traceback to the farm of origin with tracing and examination of all cattle that may have been exposed. Infection with Mycobacterium bovis is one example of the conditions which would be inappropriate for inclusion in a national, on-farm surveillance system because detection at the slaughter plant is more specific with microbiologic confirmation, detects lesions of infection earlier and costs less than on-farm systems.

Hopefully, these examples demonstrate the need to design surveillance efforts in accord with the epidemiologic knowledge of differences in host-agent-environmental factors for each of several hundred health related conditions. Consideration of biologic differences as well as the other 7 epidemiologic factors makes it essential to design and evaluate flexible, individualized plans for optimum surveillance of a given health related event. After appropriate evaluation it may be possible to combine surveillance for a similar group of events. However, the system must not be monolithic. Design must reflect the purpose of surveillance, the factors influencing each disease and provide for individual differences. Too often these individual differences have not been considered in designs for surveillance in previous years.

ECONOMIC FACTORS

Economic factors that must be considered relate to three types of costs: The costs of the disease (health event), the feasibility and costs of preventing or ameliorating the problem, and the costs of the surveillance effort. The place of the costs (on-site or off-site) must also be considered in designing surveillance efforts, since herd owners and sellers do not want to know about or report diseases when the costs can be passed "off-site" to buyers. This definitely influences detection and reporting. In addition,
benefit/cost studies of prevention, control, and eradication or treatment, require specialized data from the surveillance system in order to provide the epidemiologic coefficients and the economic loss data that are essential components of any benefit/cost analysis. Unfortunately, most surveillance efforts have not even considered how to obtain these specialized data when proposing or designing a surveillance program.

An economic constraint on surveillance, which is also perceived as an economic cost by the livestock producer, is the "identification" (ID) of animals to the farm or herd of origin. The owner believes it would cost to apply the ID, but more importantly it also would allow a buyer to return off-site costs to the seller when infected animals or animals with chemical residues were sold.

Many sellers, and some dealers, do not want effective identification because they operate under the principle of "buyer beware". Effective identification would permit the buyer to hold these sellers or dealers accountable and put the costs back "on-site" where they belong. With appropriate identification, sellers and dealers would have economic incentive to stop spreading infection, off-site, to the purchaser. Owners also believe it would increase their taxes if people knew the origin and flow of all cattle sold and purchased.

Removing these constraints and establishing identification of cattle, swine and sheep would greatly improve surveillance and substantially reduce costs. Acceptance of a permanent identification for animals would have a high benefit/cost ratio for surveillance as well as other objectives.

SOCIAL-CULTURAL FACTORS

Social-cultural factors that must be considered include knowing the influence of any social stigma associated with the event. For example, sexually transmitted diseases of people are poorly reported because of the social and cultural stigma associated with syphilis, herpes, gonorrhea. The same is true for some health related events in animals: for example, abortions from brucellosis and leptospirosis; cattle scabies; anaplasmosis; Johne's disease. Cultural and social pressures often discourage reporting as do the reward systems of our society.

EDUCATION-MOTIVATION FACTORS

Education-motivation are two very important and intertwined factors influencing surveillance. Many people have said that all we need is education to obtain high rates of detection and reporting. However, knowledge or understanding is not enough to motivate people to act or change their behavior. Educational efforts must be combined with motivation (a change in the reward system) if surveillance efforts are to succeed. Any design for surveillance must consider and plan to provide some positive rewards (rewards that people will desire) for the people involved in surveillance. These rewards must influence the livestock owner, dealers, bankers, the DVM, government employees and all other involved people.
Only through positive motivation, of the involved people, will it be possible to obtain a high probability of detection, accurate classification, timely reporting and appropriate collection of data on health related events.

Rewards should vary to fit the desires and perceptions of the different types of individuals and groups involved. For example, some people could be rewarded by timely return of results with their interpretation. Others want more in terms of helping their business or even direct payment of money for cooperating. For example, surveillance of bovine tuberculosis improved when state and federal meat inspectors were offered several hundred dollars for each animal they detected, with lesions of M. bovis, during slaughter inspection. It should be noted that the reward is paid only after the diagnosis is confirmed by culture of M. bovis from the tissues submitted by the inspector. Motivation through appropriate rewards is essential to maintain adequate surveillance. Many programs have deteriorated because rewards were not maintained.

POLITICAL-LEGAL FACTORS

Political-legal factors are important in several ways: (1) in determining the governmental resources that will be allocated to surveillance systems and the political group support for any surveillance; and (2), in mandating the identification of cattle, sheep and swine to the herd of origin along with appropriate records of all succeeding movements.

Mandatory identification is needed to make better use of sources of data, such as data from slaughter plants, diagnostic labs, DVM's, rendering plants and auction markets. Political and legal support for identification will be necessary to make optimum use of numerators and denominators. In terms of governmental resources, political support is needed to allocate funds to improve the numerator (the disease, case, etc.) and the denominator (population at risk of reporting). As I will explain in later paragraphs, it is essential to know the demographic characteristics of the animal populations at risk of reporting. Without accurate demographic information for the denominator, it is not possible to analyze and interpret the numerator (number of cases-events) in terms of rates, trends, problems or progress. In fact, without good denominator data, it is almost useless to collect data for the numerator. In designing surveillance programs, one should plan for and obtain governmental and private financial resources to characterize the population(s) of animals at risk for reporting of specified health events.

Political-legal factors also affect enactment and enforcement of laws and regulations which directly encourage or discourage the detection and reporting of health related events.

TECHNOLOGIC FACTORS

Technologic factors involve advances in identification of animals, detection and classification of health related events, communications and
ANDERSON

analysis of data. As noted earlier, identification of animals by herd of origin and from seller to buyer would offer a high benefit/cost ratio for any surveillance effort. In addition, electronic systems of identification would (1) improve accuracy for all concerned, (2) reduce labor and time now involved in reading the ID, and (3) would be more permanent. Electronic ID would also assist and complement computerization of financial and transactions records for buying and selling cattle as well as in collecting, tabulating and storing health related data for analysis and interpretation. For animals and farms with ID, the collection, storage, retrieval and analysis of data could be greatly improved with the use of the new advances in computer technology.

Advances in basic and applied science and technology continually increase our ability to detect health related events. These advances also improve the accuracy of our classification (diagnosis) of these events with the aid of a whole new array of instrumentation and equipment for our laboratory and clinical use. These technologic improvements have increased the rapidity as well as the accuracy of diagnosis and have aided our efforts to maintain freedom from specific diseases. Automation and new tests are aiding serologic detection of exposure experiences in large populations of animals. Recombinant DNA technology is contributing to development of more stable, more effective and more available vaccines. They will offer better and more widespread protection against FMD and other infections.

Improvements in communications technology will provide not only for person to person communication but communication among computer terminals at farms, auction markets, universities and government offices.

It must be noted forcefully and clearly that computers do not improve poor, incomplete, inaccurate or biased data. The saying—garbage input results in garbage output—is important for all of us to remember. Computers can aid us, but the first and most important task is to obtain accurate numerators (health events) and accurate denominators (characterization of a population at risk). Only then, can these data be appropriately analyzed and evaluated to provide a sound basis for interpretation and use of the results. Remember, computers are only another technologic aid similar to the telephone, the airplane, and the laboratory. The quality of the numerators and the denominators should be the first and foremost priority in allocating resources for surveillance programs.

In determining needs for computer hardware and software, one must again consider purposes, as well as many of the same factors that were used to individualize surveillance programs. One should plan to use the most flexible combination of hardware and software and give as much control as feasible to the people who are at the field level. This helps to maintain interest and promote responsibility and accountability in collecting and entering data into the system.

The following quote regarding surveillance of human health and
disease, I believe, illustrates the importance of knowing the quality of the data that have been collected:

"THE GOVERNMENT (STATISTICIANS) ARE VERY KEEN ON AMASSING STATISTICS—THEY COLLECT THEM, ADD THEM, RAISE THEM TO THE NTH POWER, TAKE THE CUBE ROOT AND PREPARE WONDERFUL DIAGRAMS. BUT WHAT YOU MUST NEVER FORGET IS THAT EVERY ONE OF THESE FIGURES COMES IN THE FIRST INSTANCE FROM THE VILLAGE WATCHMAN, WHO PUTS DOWN WHAT HE DAMN PLEASES."

Sir Josiah Stamp — 1929

CRITERIA FOR EVALUATING DATA NEEDS

In designing surveillance systems one must carefully evaluate the types of data that are needed for any given disease and method of surveillance as shown in TABLE 2.

TABLE 2
CRITERIA FOR EVALUATION OF DATA NEEDS
—QUANTITY AND QUALITY—

A. PURPOSES AND OBJECTIVES.
B. NEED FOR CHARACTERIZING THE DENOMINATOR
   1. POPULATION AT RISK OF REPORTING
   2. DEGREE OF CHARACTERIZATION
C. NEED FOR CHARACTERIZING THE NUMERATOR
   1. UNIT AT RISK OF REPORTING
   2. ACCURACY OF CLASSIFICATION (DIAGNOSIS)
   3. DEGREE OF DETECTION (PROBABILITY)
D. NEED FOR ANALYSIS AND INTERPRETATION
   1. TIMELINESS
   2. IMPACT—CONSEQUENCES
   3. BENEFIT-COST RATIO

DENOMINATORS

Table 2 again reminds us to define specific objectives to evaluate quality and quantity of data needed for denominators. Not only does one need to know the characteristics of the denominator, but the amount one needs to know depends on the degree of characterization needed for any given purpose.

For some purposes it may be enough to classify cattle as breeding or feeder cattle. However, for some disease and benefit/cost models it is necessary to have surveillance data which provide information on characteristics of the population at risk of the event, such as: sex, breed, age, use of veterinary medical services, type of operation and management patterns, patterns of movement of animals, environmental factors, and other data on the population which may be needed for analysis and inter-
interpretation of the numerator. The need for denominator data for specific characteristics, such as age or breed is illustrated by the following examples:

(1) in a study of 776 reported cases of bovine leukemia in Minnesota, 686 of these cases (88%) occurred in the Holstein breed of dairy cattle. Some people immediately analyzed these data impropely to say, since 88% of the reported cases were Holsteins, that Holstein cattle were more susceptible to leukemia and had a greater risk of leukemia than cattle of the Jersey breed which had less than 5% of the reported cases. This was not a correct interpretation. To correctly interpret these data, one needs a denominator to provide the characteristics of the dairy population at risk of reporting. Because the population at risk of reporting in this Minnesota study was characterized for each breed, the data demonstrated that 87% of the dairy cattle at risk of reporting (denominator) were Holstein. Having this information about the denominator quite clearly established that the 88% reported cases in Holsteins could be expected when 87% of the dairy cattle at risk were of the Holstein breed. Unfortunately, people who report and interpret numerators (cases of animal disease) often fail to know essential facts about the denominator (population at risk).

(2) another example is based on the need to characterize the population at risk (denominator) according to basic demographic factors such as age. In a study of bovine leukemia in Minnesota, investigators found that reported cases occurred most frequently in the group of dairy cattle 6-9 years of age. Some people, improperly, wanted to say the incidence rate and risk of disease was highest for 6-9 year old dairy cattle without properly using a denominator (population at risk) to determine age specific incidence rates in the population at risk. When age specific incidence rates were calculated, using the age distribution in the denominator, (population at risk) it was clearly demonstrated that the incidence rates and risk of disease increased with increasing age. The greatest risk of being a case was in dairy cattle greater than 9 years of age. People need to recognize that although there were fewer reported cases in dairy cattle more than 9 years of age (numerator) there were also fewer dairy cattle, more than 9 years of age (denominator), the population at risk of becoming a case. Appropriate characterization of the numerator and denominator were needed for accurate interpretation.

NUMERATORS

Table 2 item C lists several concerns that must be considered in evaluating the accuracy of reported cases being analyzed and interpreted as part of the numerator. First, we must be concerned with the unit at risk of reporting—individual animal, a relatively stable herd, or a "lot" (group) of cattle assembled for sale, purchase or some other special purpose. During past years, all three of these units have been used as numerators in reporting disease without knowing the characteristics of any of these numerators.
The unit (herd, lot, animal) being used as a numerator should be defined and characterized in all surveillance efforts. The unit may vary with the disease or condition being reported. The herd unit may be the most appropriate in some instances and individual animals may be the appropriate unit in other situations.

ACCURACY OF CLASSIFICATION

Accuracy of classification of cases is another major problem of surveillance. This involves accuracy of diagnosis and collection of the data. The accuracy of diagnosis is related to the competence and motivation of the reporting individual, degree of difficulty in making an accurate diagnosis, and the available resources, such as laboratories, to aid in diagnosis. Accuracy in data collection, including recording and tabulating also are problems that must be continually evaluated.

DEGREE OF DETECTION

Degree of detection involves the probability that a case (event) will be detected. This varies greatly with the type of disease and the importance. For example, a case of bovine rabies has a greater probability of being detected than a mild and transient inflammation of the udder (mastitis). Another important factor influencing the probability of detection and reporting of an event is the availability of a DVM to detect, diagnose and report the event (case). Lack of veterinary medical supervision of health and productivity in animal populations has a great influence on accurate reporting of most numerators (health-related events).

ANALYSIS AND INTERPRETATION

In designing and evaluating surveillance efforts, the type and amount of analysis and interpretation must be based upon the needs of the users and the importance of the data. Timeliness of analysis and feedback to users is an essential factor to be considered in any program. Timeliness will vary with the purpose and objectives, with type of user, and with type of disease. It may be appropriate to have preliminary, secondary and then final reports in sequence, as analysis and interpretation progresses.

Certainly one must consider the impact and consequences of reporting data with varying degrees of analysis and interpretation, but one must also consider the impact and consequences of delaying reporting until further analysis and interpretation. The benefit/cost ratio may be very helpful in determining the type and amount of analysis and interpretation needed before providing feedback to the interested groups and people. This benefit/cost ratio will vary among situations and among different diseases; e.g., daily feedback and interpretation of epidemic FMD data vs. 3 year feedback and interpretation of data for displaced abomasum of cattle. The need for reporting results of analysis and interpretation must continually be evaluated by factors listed previously in item D, Table 2.
TYPES OF SURVEILLANCE ACTIVITIES

ACTIVE SURVEILLANCE

Active surveillance is concerned with the efforts of investigators or an agency to actively seek reports of all cases by stimulating detection and reporting by others or by special studies of an appropriate sample of the population which can be statistically and biologically evaluated. Examples of active surveillance include:

1. Stimulation of detection and reporting by others such as:
   a. frequent contact and feedback in person, by phone, by personal letter, etc.
   b. providing assistance and consultation in diagnosis for rare, emergency or very difficult to diagnose diseases.
   c. providing a reward which is desired by those detecting and reporting the event(s).

2. Special studies of occurrence (time, place, animal/herd) of health-related events such as:
   a. outbreaks (epidemics) of selected health events.
   b. incidence/prevalence of clinical and subclinical conditions,
   c. mortality,
   d. case fatality,
   e. productivity,
   f. coefficients for benefit/cost studies,
   g. analytic and experimental intervention studies.

3. Note: all examples of active surveillance to report events (numerators) should include appropriate characterization of denominators (the population at risk of event reporting).

PASSIVE SURVEILLANCE

Passive surveillance is concerned with passive collection of reports of health-related events. These events are usually reported more or less selectively by professionals, by people affected and by agencies; each primarily concerned with only a particular portion of the numerator which comes to their attention and with little or no knowledge of the denominator. Examples of passive surveillance include reports from:

1. laboratories,
2. slaughter plants (except for targeted lesions, such as bovine tuberculosis),
3. DVM practitioners (except for special studies),
4. veterinary teaching hospitals (except for special studies),
5. research institutes (not special studies),
6. rendering plants,
7. human health agencies (zoonoses: not special studies),
8. people affected by the event,
9. pharmaceutic and biologic firms (not special studies).
10. Note: Some of these numerator data are flawed with inaccuracies in
diagnosis and there is very little knowledge, and no attempt to
characterize the denominator (population at risk of reporting).

COMBINATIONS

Combinations of passive and active methods of reporting often add to
the difficulties of evaluating numerators and denominators. It has been
well shown that passive methods of surveillance may vary greatly in re-
porting even though there is little change in the ongoing frequency of ac-
tual occurrence of the disease.\(^8\)

HUMAN HEALTH SURVEILLANCE EFFORTS

Professional colleagues often point, incorrectly, to the Morbidity and
Mortality Weekly Reports (MMWR) of the U.S. Centers for Disease Con-
trol as being an example of perfection in collecting reports of disease in
the human population. Unfortunately, CDC utilizes passive methods for
collecting much of the reported data, and unfortunately, these reports
have many of the same deficiencies noted in animal disease reporting sys-
tems over the last 30 years. Reports, on the occurrence of human lepto-
spirosis, assign cases to states that have a special interest in detecting
the infection in people without any differentiation for states that do not
care to report leptospirosis in humans.\(^9\) Thus, the geographic
distribution of human leptospirosis and many other diseases are a re-
fection of the motivation and probability of detecting and reporting
these diseases rather than a geographical representation of their actual
occurrence. As with animal diseases, the motivation for reporting some
human diseases is greater than for others, e.g., a very high proportion of
human rabies cases are reported, while a very low proportion of human
salmonellosis cases are reported (1 of 10,000). Epidemiologists are well
aware of these deficiencies and continually seek better data through
special studies with active surveillance, other types of surveillance
programs adapted to specific diseases and new forms of physician
oriented disease reporting.\(^11\)

A dramatic example of the problems encountered by the Centers for
Disease Control in collecting reports of human disease is described in rec-
t articles on the occurrence of Toxic Shock Syndrome (TSS) between
motivation is a source of bias in reporting with the following statements,
“The disproportionate geographic distribution of (reported) toxic shock
syndrome cases is undoubtedly due in part to differing levels of interest
among state health departments and local clinical investigators.”\(^12\)

Osterholm et al\(^13\) and Davis et al\(^14\) have published studies indicating fur-
ther bias in CDC data when one examines the temporal distribution of
TSS cases reported to CDC between October 1978 and September 1981.
CDC data (Figure 3, Reingold et al) show a gradual increase through the summer of 1980 with a peak of about 128 cases reported in August 1980. This was followed by a sharp decline through August 1981 to about 30 reported cases.12 However, using an active surveillance system, Minnesota did not experience the decline in reported cases that affected the passive surveillance system used by CDC from September 1980 to September 1981.16 Using an active surveillance system, Minnesota reported 197 cases of TSS up to June 30, 1981, equal to 15% of all cases reported by 50 states, although Minnesota has only 2% of the population at risk of reporting TSS in the U.S.15 Contrary to CDC data for the U.S., Minnesota did not show any significant changes in the reported occurrence of TSS between January 1980 and June 1981. Minnesota reported an average of 19 cases for the 3 quarters before removal of Rely tampons from the market and an average of 19 cases for the 3 quarters after removal of Rely tampons from the market.15

These data have been evaluated by many people because of the differences in reporting by the passive surveillance of CDC and the active surveillance of Minnesota. Alexander Langmuir, former Chief Epidemiologist, CDC, in a review for the Journal of Infectious Diseases stated, “it (the surveillance study in Minnesota by Osterholm and Forfang) describes a practical but thorough technique of both active and passive surveillance that sets a ‘gold standard’ for all states to emulate.”16

Studies in Wisconsin by Davis further demonstrated that passive systems like Wisconsin and CDC were greatly affected by publicity surrounding withdrawal of Rely tampons when reported cases reached their peak.17 Following this withdrawal, the reported cases sharply declined in both CDC and Wisconsin reports, but the Minnesota system was relatively unaffected by these external events and continued to report cases at a relatively constant rate.15

Peterson, from Washington, also criticized the bias in passive reporting systems as used by the Centers for Disease Control. He states, “As with foodborne disease outbreaks, we obviously have selective reporting in the nationwide reporting system for toxic shock syndrome that will bias the overall frequency in the direction of under-reporting.” He further commented, “The rates of occurrence of Toxic Shock Syndrome that have been reported are relatively meaningless numbers unless they can be put in perspective.”18

It should be noted that reporting of cases to obtain incidence rates is only one epidemiologic method to analyze for risk factors and develop etiologic hypotheses. In fact, Osterholm et al,19 Davis et al,20 and Shads et al21 designed and conducted analytic case-control studies during 1980 and 1981 which provided important data regarding occurrence in the population, risk factors and etiologic hypotheses.

The experience with TSS emphasizes:

1. the hazards of relying on a biased monolithic national system of surveillance;
2. the need for several types of surveillance activities as conducted by individual states and university investigators;
3. the value of denominator data characterizing the population at risk;
4. the value of using a number of epidemiologic methods including case-control studies as part of surveillance efforts;
5. the value of active vs. passive surveillance.

ANIMAL NUMERATORS AND DENOMINATORS

The review and constructive critique of the state-federal CDC system, using TSS reporting as an example of the problems with the numerator (reported cases), should not obscure the fact that surveillance systems for human health related events do have census data available as a denominator (population at risk). The information regarding denominators in human populations, as furnished by census data, use small units (census tracts) to provide considerable demographic information. Denominator data for the TSS studies were supplemented by marketing and production data from the tampon manufacturers and distributors. These facts again emphasize the need to develop improved methods of obtaining information regarding animal populations (denominator) at risk of health related events. In summary, to design surveillance efforts for animal health and productivity, we need to allocate priority resources to obtain valid data for a denominator (population at risk) as well as a numerator (data on the health related event).

PROBLEMS OF ANIMAL HEALTH RELATED SURVEILLANCE

For many years people have complained about the lack of quality and quantity of surveillance efforts for animal health related events. Veterinarians and veterinary medical organizations have participated in many efforts to design and implement state, national, international and local systems of surveillance designed to collect data on a broad range of health related events in several countries of the world. In spite of these studies and efforts, over the past 30 years, to design surveillance systems, most have been disappointing to their adherents and/or users by failing to provide the degree of validity and reliability desired in reporting the incidence and/or prevalence of selected health related events in animal populations.

Such failures were often predictable because key issues of implementation and maintenance were not considered as recommended in Tables 1, 2, and 3 of this paper.

Some excellent examples of problems and failures, when essential criteria were not followed, are provided in the papers of McCallon and Beal and Beal.

Example 1: PASSIVE vs. ACTIVE SURVEILLANCE: "Though only 1 case of chorioptic cattle scabies had been reported in Vermont in the preceding 8 months, an intensive inspection program for cattle scabies was
started in Vermont in February of 1968. In less than 3 months, 1,130 laboratory confirmed infected premises (herds) were found. U.S.D.A. data indicate that the small state of Vermont reported 5 scabies herds in 1967, 1,133 in 1968 and only 6 scabies herds in 1969. Was this a true epidemic in 1968? No, this was the result of improved detection of infected herds by changing from a passive system of reporting to an active surveillance system. When employees were instructed to look for chorioptic cattle scabies, they found it and reported the infected herds resulting in this dramatic increase. This example again illustrates the inadequacies of passive systems of surveillance wherein reported cases vary, not with the amount of infection, but with the amount of motivation and effort applied to seeking, detecting and reporting health related events. This example is similar to the large increase in reported leptospirosis in Iowa when investigators instituted active surveillance and the lack of any increase in reporting in surrounding states of Minnesota, Wisconsin and Missouri which continued to use passive surveillance.  

Example 2: Fallacies of using LABORATORY TEST DATA: In 1972 the laboratory test results for anaplasmosis indicated that of samples submitted for testing, Arkansas had 36.4% reactors and California had only 0.7% serologic reactors for anaplasmosis. However, a national probability sample survey indicated 5.3% reactor samples for Arkansas and 10.4% for California. The discrepancy was explained by different selections of numerators (samples) being submitted to the laboratories. In Arkansas, the Lab was receiving samples from cattle suspected of being infected while in California the Labs were receiving samples from cattle thought to be free of infection and selected for sale to Hawaii as anaplasmosis free. Selection bias and lack of knowledge of selection of numerators and denominators contribute greatly to unreliability of usual laboratory data for purposes of determining incidence or prevalence.  

Other examples from McCallon and Beals show inappropriate interpretation of data on trichinosis. They show failure to recognize the need for data on herd size (numerators and denominators) to provide stratification by herd size to avoid gross errors in extrapolation of data on pseudorabies from sample surveys to all herds in the U.S. or to compare data between 2 sample surveys. Failure to adequately characterize herds involved either as numerators or denominators, results in major errors in analysis, interpretation and actions that may be recommended. This can result in wasting millions of dollars for both industry and government.  

Recently, the U.S.D.A. meat inspection program has been publicizing plans to contribute data to a surveillance system for many diseases observed in animals at the time of slaughter. Unfortunately, such publicity has been misleading to many DVM's and to the industry. Data from meat inspection, if confirmed by laboratory examination can be helpful in detecting a few selected diseases such as tuberculosis. However, many diseases do not show significant gross lesions in animals at time of slaughter. These diseases would not be detected at slaughter. In addition,
slaughter plant inspection is not set up to provide definitive diagnoses and classification of disease in animals. Therefore, it seems wholly unjustified to plan to acquire valid and reliable data on incidence or prevalence of health related events in animals from meat inspection programs until (1) they are dramatically changed to provide accurate diagnoses of infectious and non-infectious conditions and, (2) until an adequate identification system can provide appropriate characterization of the animals being slaughtered (numerator) and characterization of the herd or flock of origin (denominator).

The more we analyze the needs for surveillance, the more obvious it becomes that animal identification as recommended by the National Brucellosis Technical Commission is essential.2 We must be able to trace movements of animals and to relate them to herds of origin as well as to herds of purchase. The movement of animals between and among states is very important in the transmission of infectious diseases such as brucellosis. Without an appropriate system of permanent identification there have been failures in the prevention of transmission of brucellosis from state to state and herd to herd.63

Although the Federal-State brucellosis eradication program has more data than for any other disease, Dr. Ray has discussed the many problems of collecting valid and reliable data. These include many of the issues and criteria discussed in Tables 1, 2, and 3 of this paper, such as motivation of people, education and training, political and legal problems, failures to appropriately characterize numerators and denominators. In fact, one of the big problems in conducting benefit/cost studies on brucellosis is the lack of adequate data characterizing the cattle population at risk of the disease in each of the 50 states.63, 64, 65

Table 3 provides a list of the methods that should be included in every surveillance system:

**TABLE 3**

SURVEILLANCE SYSTEMS SHOULD INCLUDE
APPROPRIATE METHODS FOR:

1. CHARACTERIZING THE DENOMINATOR-POPULATION AT RISK FOR REPORTING;
2. DETECTING AND CLASSIFYING THE NUMERATOR-HEALTH RELATED EVENTS;
3. COLLECTING AND TABULATING REPORTED EVENTS;
4. EVALUATING AND VALIDATING DENOMINATORS AND NUMERATORS;
5. APPROPRIATE ANALYSIS AND INTERPRETATION OF DATA;
6. TIMELY REPORTING OF OUTCOMES TO APPROPRIATE GROUPS;
7. EVALUATING OUTCOMES AND METHODS IN ACCORD WITH PURPOSES OF THE SPECIFIC SURVEILLANCE PLAN;

8. RECOMMENDING AND INITIATING APPROPRIATE ACTIONS TO ACHIEVE PURPOSES.

Timely reporting to appropriate groups is essential and methods should be designed to accomplish this effectively. Feedback requires that someone analyze and interpret surveillance data. Van Ness emphasized the importance of feedback by saying, "Without this meaning, the mere collecting together of statistics may represent, as observed by Theobald Smith in 1887, a stupendous and probably useless task."

RECOMMENDATIONS AND THE FUTURE

Fortunately, for the livestock industry, animal health officials and veterinarians progress is being made. The Morbidity and Mortality Committee of the U.S.A.H.A. has been providing leadership in making recommendations to APHIS for several types of improved plans for surveillance. Studies have been conducted in cooperation between the Committee, APHIS, the University of Minnesota and others to evaluate methods of surveillance for incidence and prevalence on appropriately selected farms. APHIS has adopted a plan and is working with the Morbidity and Mortality Committee to further develop designs and plans for a nationally coordinated plan for several types of surveillance for health related events in animals including productivity and benefit/cost data. With the interest and cooperation being furnished by veterinary epidemiologists throughout the U.S., the leadership of the U.S.A.H.A. Committee on Morbidity and Mortality, and the efforts of statisticians, epidemiologists and administrators at APHIS, it appears that planning, pilot testing of plans and implementation for several types of surveillance will proceed with appropriate review and evaluation during the next 5 years.

As part of its leadership in the field of surveillance, it is recommended that the Committee on Morbidity and Mortality of the U.S. Animal Health Association:

1. Continue to provide a forum for presentation and evaluation of new ideas and methods to improve surveillance;
2. Continue to provide leadership in the design and development of surveillance systems;
3. Continue to encourage design and development of systems with specific purposes and specific methodology to fit specific needs of individual or closely similar health related events (not one monolithic system);
4. Continue to review and evaluate plans, design and performance (outcomes) of surveillance systems using the concepts, criteria, and methods presented in this paper; and particularly to evaluate surveillance using the criteria and recommendations presented in:
SURVEILLANCE

(a) the definition of surveillance; (b) Table 1, Epidemiologic Factors; (c) Table 2, Criteria for Data Needs; (d) Table 3, Appropriate Methods.

5. Continue to review and revise the definitions, concepts and Tables presented in this paper as it seems appropriate.

REFERENCES


4. Osterholm, Michael; personal communication.


33. Davies, G.: Animal Disease Surveillance in Great Britain. Head Epidemiology Unit, Ministry of Agricultural Fisheries and Food, Central Veterinary Laboratory, New Haw, Weybridge, Surrey, United Kingdom, 1976.


HISTORICAL ASPECTS OF ANIMAL DISEASE REPORTING AND THE DEVELOPMENT OF A NATIONAL ANIMAL DISEASE SURVEILLANCE SYSTEM

William R. McCallon (DVM) and Victor C. Beal, Jr. (PhD)

Principal Staff Officer (Veterinary Economist), Emergency Programs, and Biometrician, National Program Planning Staffs; VS, APHIS, USDA, 6505 Belcrest Road, Hyattsville, MD 20782.

Veterinary Services (VS) of the Animal and Plant Health Inspection Service (APHIS), U. S. Department of Agriculture (USDA), its predecessor organizations, and similar organizations in many other countries have been involved in disease reporting and surveillance for many years. In general, this has been a frustrating experience for all concerned, as the need for valid information concerning disease prevalence, incidence, trends, and economic losses has long been recognized. However, no one, as yet, has developed a system sufficient to produce this information on a large scale.

Researchers and Regulatory veterinarians need accurate information concerning disease prevalence, incidence, trends, and economic losses in order to plan their research activities and regulatory programs. The Office of Management and Budget (OMB) of the U. S. Government and the U. S. Congress have insisted on economic analyses in their allocation of funds. Such analyses demand accurate information in the areas listed above.

Meat and milk producers need information concerning the economically significant diseases in their area, how various management practices affect these diseases, and the most cost effective means to prevent or control them. Most of this needed information is not now available.

Indeed, the livestock industry, those associated with it, as well as those attempting to serve its needs, are becoming increasingly aware of the need for valid information concerning disease prevalence and the economic importance of diseases. There is little, if any, disagreement about what information is needed, but there are many schools of thought as to how best to secure this necessary information. Consequently it would be beneficial to briefly review the history in this country with regard to disease reporting and surveillance, determine what the situation is now, and what the future should hold.

HISTORY

Traditional Animal Disease Reporting Systems in the United States

The National Report of Animal Diseases: For many years, VS coordinated a national disease reporting system. This was the National Report on Animal Diseases (NRAD). The system consisted of a small and varying number of practicing veterinarians in most states reporting the diseases they thought they had seen in the course of their practice the
preceding month. In general, this information was obtained through the filling out of questionnaires which were mailed out by the State Veterinarian each month. In some states, this was augmented or replaced by the records from veterinary school clinics and/or state diagnostic laboratories.

The National Animal Morbidity Report: The NRAD was supplemented by the National Animal Morbidity Report (NAMR). In general, the NAMR covered control program diseases such as brucellosis and diseases ancillary to these activities such as paratuberculosis and chorioptic scabies. However, it included some other diseases, also covered in the NRAD, such as rabies and anthrax. All program diseases were also included in the NRAD. This included official tests for VS program diseases.

Two Reports Make Up Traditional System: It was the responsibility of the VS Veterinarian in Charge in each state to compile the NRAD information and send it to national headquarters where the data was compiled on a state-by-state basis. After this was done, a summary of the NRAD giving national totals was distributed. In addition, the NAMR was compiled and distributed on a state-by-state basis. Such a system is the traditional reporting system used by nearly every country in the world.

The Traditional System in Free Enterprise and Socialistic Countries

It would appear that the U.S. and other private enterprise countries are at a disadvantage in implementing a traditional reporting system, as virtually none of their practicing veterinarians are employed by the Government, while in most socialistic countries, many or all of the veterinarians are employed by the Government. The seeming advantage, in this respect for socialized veterinary medicine is that Government employed veterinarians can be made to report while private enterprise veterinarians cannot and, in fact, a very small minority of them ever reported in any given month.

However, it should be pointed out that this apparent advantage for socialistic countries is not as great as might be expected, especially for developing countries. When one has the opportunity to observe how their reporting system works, the first thing noticed is that most Government-employed veterinarians are in large cities with no transportation so they have little opportunity to see sick animals. Additionally, there is little laboratory support for a diagnosis, so under these circumstances, there is some doubt about the source and validity of what is being reported.

Reviews of the Traditional System in the United States

National Academy of Science Review: There have been various reviews of the traditional system of disease reporting in the United States. The first critical review was under the auspices of the National Academy of Science (NAS) by Schroeder, et al. (1966). This review was quite thorough in terms of reporting what had been done and what the then current status was in the United States.
Veterinary Services Review: It was as a result of a second critical review inside of VS prior to the start of FY-73 that the NRAD was discontinued. Various facts were revealed as a result of that review.

The NRAD was more costly than supposed since most of the regulatory field veterinarians spent some time in calling on practitioners encouraging them to report. Additionally, considerable clerical and managerial time was spent at the State level soliciting and reviewing information. Also, about $50,000 was being spent at the national level in compiling and distributing the report.

It was pointed out that the traditional system revealed nothing concerning disease prevalence or economic significance for any area of the country. Additionally, it was pointed out that even disease trends could not be estimated with any degree of confidence.

Many individuals and firms requested information concerning how many cases of a certain disease were being reported. It was very misleading to tell them that only a few cases had been reported when we knew that this was probably a minute fraction of what had really occurred.

In view of the above factors, it was decided that the NRAD reporting system was providing little, if any, useful information, and it was, in fact, misleading. As it was fairly costly, the decision was made to discontinue it.

Studies of Ways to Improve Information

National Academy of Science Study: As a result of the NAS review and concurrently with the VS review, many thought that the traditional system should be improved or replaced so as to have a system which would provide valid data concerning disease prevalence and economic losses. Consequently, a study group was formed in 1969 under the auspices of the National Academy of Science (Hutton and Halvorson 1974). This group seemed to be divided into four schools of thought.

Traditionalist School of Thought: The "Traditionalists" thought that VS should continue the old reporting system and try to improve it by getting more veterinarians to report (Hutton, 1971). By doing this, perhaps some insight could be gained concerning disease losses and trends and it would at least be known whether or not a disease existed in the country. Also, the United States would have a system which was similar to those of other countries.

Their critics pointed out that no matter how many veterinarians reported, such a system would not reveal any valid information concerning prevalence of diseases and economic losses. (Harper et al. 1977). Additionally, it was pointed out that diagnosis was in doubt, as it was usually not based on laboratory backup and was often based on memory. Also, it was not uncommon for exotic diseases to be reported which on investigation proved to be something else. In view of this, such a system could not even reveal whether or not a disease existed in the country. If a
disease was not reported, there was no assurance it did not occur, and if reported, there was doubt about the diagnosis.

**Laboratory School of Thought:** The "Laboratory School" felt that only laboratory test results should be collected (Hutton, ibid). By doing this, there would be more certainty of the diagnosis, so the validity of the data would be much greater.

Their critics pointed out that there would still be nothing known about disease prevalence, economic losses, or whether a disease existed in the country if no positive laboratory test had been disclosed (Harper, ibid). In addition, there have been reports of a relationship between distance from a laboratory and frequency of submission. However, it was conceded that in those cases where positive tests were disclosed, we could be more confident that the disease existed than when a practitioner reported he had encountered the disease.

**Lumpers School of Thought:** The "Lumpers" wanted to continue the traditional reporting system, collect laboratory test results, inspection records, and data from many other sources (Hutton, ibid). By doing this, one could amass an impressive amount of data and offend no one, as all data would be included.

Their critics pointed out that such a system would provide no valid information concerning disease prevalence or economic losses and offer no significant improvement over the laboratory system concerning whether or not a disease existed (Harper, ibid). Moreover, it would be much more costly.

**Statisticians School of Thought:** The "Statisticians" felt that the only way we could determine prevalence, trends, losses, or even know whether or not a disease existed was to conduct a statistically sound survey using a diagnostic method of high accuracy (Beal and Cox 1970, Cox and Huddleston 1971).

Their critics pointed out that it would be much too costly to do this for a great number of diseases so, in effect, they were offering no feasible proposal for a reporting and surveillance system.

**Problems of Invalid Data:** Problems arising from invalid data are reflected by the old NRAD system which has been mentioned earlier. As mentioned above, the data was compiled from reports which were submitted from individual states. This data had an unknown denominator. Arkansas would report anywhere between 18 and 45 percent of the cases reported in the nation for anaplasmosis and California would report under one percent of the cases. Yet a probability sample survey of brucellosis market samples indicated that Arkansas had approximately five percent and California had approximately ten percent of the infected animals in the nation. Table 1 (Beal 1980) provides a picture of the difference between the old NRAD and the probability sample survey in regard to the anaplasmosis picture for seven States.

This table gives the relative distribution of anaplasmosis in seven
selected States with the average number of cases per year for fiscal years 1968 through 1972 from the old NRAD and with the estimated number of reactor animals and reactor rate from that probability sample survey of market cattle samples.

As can be seen from this table, there was little relationship between the true incidence of anaplasmosis as revealed by a probability sample survey and the biased picture as revealed by the old NRAD. As mentioned above, the NRAD was discontinued in 1972 because it was felt to be inadequate.

Table 1. Real and Apparent Distribution of Anaplasmosis.

<table>
<thead>
<tr>
<th>Location</th>
<th>NRAD Ave. No. Cases Per Fiscal Yr.</th>
<th>Reactors in 1,000's</th>
<th>Percent Reactors</th>
<th>Percent of Infection in United States</th>
</tr>
</thead>
<tbody>
<tr>
<td>United States</td>
<td>7,136</td>
<td>5,640</td>
<td>10</td>
<td>100</td>
</tr>
<tr>
<td>Arkansas</td>
<td>2,597</td>
<td>297</td>
<td>22</td>
<td>36.4</td>
</tr>
<tr>
<td>California</td>
<td>48</td>
<td>588</td>
<td>31</td>
<td>0.7</td>
</tr>
<tr>
<td>Louisiana</td>
<td>1,011</td>
<td>240</td>
<td>23</td>
<td>14.2</td>
</tr>
<tr>
<td>Mississippi</td>
<td>139</td>
<td>364</td>
<td>23</td>
<td>1.9</td>
</tr>
<tr>
<td>Missouri</td>
<td>155</td>
<td>368</td>
<td>12</td>
<td>2.1</td>
</tr>
<tr>
<td>Oregon</td>
<td>136</td>
<td>92</td>
<td>13</td>
<td>1.9</td>
</tr>
<tr>
<td>Texas</td>
<td>809</td>
<td>1,086</td>
<td>15</td>
<td>11.3</td>
</tr>
</tbody>
</table>

Veterinary Services Study of 1976-1977: As a result of the United States entry into the Organization of International Epizootics and of the failure of the 1974 NAS study to specify alternatives, VS embarked on yet another study of possible ways to improve animal disease information in 1976 (Harper ibid). Where the NAS study failed to examine the various sources of information in terms of validity, this study proceeded to do so. This study also provided estimates of the costs of various alternatives. The validity of information has also been examined by Leech and Sellers (1979).

As can be seen, over the past 10 years, a number of groups have studied the problem and that in every case, there has been little agreement between these four schools of thought. When one school of thought had the majority membership of the committee, their report tended to reflect the majority viewpoint.
Research in Statistically Valid Methods

Over the years, there has been research by various groups into methods of obtaining valid information. These activities which extend back for about 35 years appear to be limited in number.

Iowa and British Studies: The earliest known study was in the State of Iowa in 1946-1947 (Anon 1947, Snedecor 1947, and Newton 1947). Research also took place in Great Britain in the late 1950's (Leech 1971a and 1971b).

Minnesota Study: Beginning in the early 1970's, a group at the University of Minnesota began work on a reporting and surveillance system for the State of Minnesota (Diesch and Martin 1979, Martin and Diesch 1980, and Diesch et al. 1981). The big difference in their system and the traditional system was that a statistically determined number of herds from various areas of the State were randomly selected and surveyed by private veterinarians on a regular basis so that for the first time one could draw inferences concerning disease prevalence and trends over a period of years.

Their system was operational long enough to reveal its strengths and weaknesses. The great advantage it offered was being statistically based so the observation sample was near enough random that it disclosed some valid information concerning prevalence and disease trends. However, several problems were encountered.

Their system provided little information concerning economic losses and this is vital information as we are not particularly concerned about a disease of high prevalence if the resulting losses are very low.

The diagnosis was suspect as, like the traditional reporting system, most of the diagnosis was memory based on an educated guess by the herd owner or veterinarian with only occasional laboratory backup.

Surveillance and reporting was done for the most part by private veterinarians. This posed a problem, as in many cases work was not done on schedule or not at all. It seems unlikely that such a system will ever work well as practicing veterinarians tend to lose interest or get busy with other jobs and let the reporting system fall behind.

WHERE TO GO—RECENT DEVELOPMENTS—FUTURE PLANS

Where to Go

After over 10 years of numerous review and study groups, the problem still exists of where to go from here to get the needed information. Perhaps the best start would be to define what information the industry and other users need and then determine what system would be necessary to provide this information.

Basic Information Needed: As mentioned earlier, there is certain basic information needed. This includes; 1) knowledge of prevalence of a number of different disease, 2) knowledge of the economic losses
associated with these diseases, and 3) knowledge of whether these diseases are increasing, decreasing, or stable in prevalence.

Other Useful Information: Other useful information includes; 1) knowledge of the effect of various types of operations on disease prevalence, 2) knowledge of the effectiveness and cost of various control strategies on disease prevalence, 3) knowledge of the effect of the environment on disease prevalence, and 4) ability to make retrospective studies. This would require storing some samples in a serum bank.

To Secure This Information, it Would be Necessary To: It will be necessary to design a statistically based system similar in this respect to the Minnesota system. The field work would have to be done by specially trained, Government employed veterinarians who could be depended on to conduct surveillance in a timely, uniform manner. Diagnosis would have to be supported by laboratory tests in selected cases. This would include some serum which would also be stored in the serum bank. Production records would have to be noted and correlated with disease prevalence, types of operations, control strategies, and environmental factors.

Recent Developments and Future Plans

In October 1981, the Animal Plant and Health Inspection Service (APHIS) presented to the Committee on Morbidity and Mortality Reporting of the United States Animal Health Association (USAHA) a proposal for the development of a statistically based Animal Disease Surveillance System. “The USAHA Committee on Morbidity and Mortality voted unanimously to support the APHIS position on animal disease surveillance. This committee will review the progress of the phases in the development of this system on a continuing basis.”

Since that time, a working group from Veterinary Services (VS) composed of the authors and others have been developing the plan.

Course of Action: One or two States will be selected for a pilot project beginning next year. The project will be enlarged to include one State in each of our five regions the following year. The project may be enlarged somewhat over the next several years if necessary. At any rate, the pilot project will be continued for about 5 years to correct any problems which may develop. At this time, information should be available as to the cost and benefits of the system so a decision can be made as to whether or not to implement the system on a national basis.

The Sample: Initially, the system will cover beef cattle, dairy cattle, and swine. Additional species will probably be added later. The sample will be drawn from the United States Department of Agriculture's Statistical Reporting Services' sampling frame. Their sampling frame includes over 95 percent of our livestock. We intend to stratify the sample by types of operation and by size. For example, beef cattle will be divided into cow-calf operations, grazers, and feeders. These groups will be further divided into small, medium, and large sizes. We have been
working with University and government statisticians and animal husbandry experts in determining those groupings.

The Diseases: We will not be able to survey for all diseases. About 20 diseases or disease conditions per species will be our limit. Contacts are being made with Veterinary Schools, State diagnostic laboratories, and livestock organizations for their advice. Every year or two, diseases which do not seem to be of significant economic importance will be dropped from the list and replaced with diseases which may be.

The Data Collectors: Data will be collected by Federal, State, and university veterinarians. These veterinarians will receive specialized training in this area. Most will have only a few assigned herds—6 or less.

Sampling frequency and Duration: Plans are to start with monthly sampling. If this can be reduced to bi-monthly or quarterly intervals, costs would be greatly reduced. It is anticipated that each herd will be surveyed at least 1 year and perhaps as long as 3 years.

Diagnosis: The basic diagnosis will be made by the data collectors and the herd owners. Private practitioners will be consulted when they have been involved. Some samples will be subjected to laboratory confirmation to check the accuracy for the field diagnosis.

Training: Training courses are being developed for data collecting veterinarians. These courses should insure uniformity of collection. We anticipate the most difficult part will be the collection of economic data. These courses will be of about 1 weeks’ duration.

Information: The system will provide only limited benefit if it is not used by livestock owners in making their herd health plans. In view of this, we intend to work very closely with the Extension Service and the trade press.

CLOSING REMARKS

Most of those concerned have long recognized the need for more precise information concerning animal disease prevalence, incidence, trends, and economic significance. Unfortunately, this information is generally not now available and would be fairly costly to obtain. So, we have tried to make do with data from slaughterhouses, diagnostic laboratories, veterinary and owner reports, rendering plants, and any other source that was free or could be obtained at little cost. The result has been that we could never come up with frequency rates, as our data always contained either no numerator or denominator or the sample was badly biased. This has long been a very frustrating experience for those working in this area.

The time has come for us to develop a statistically sound system which will provide the needed information. We should recognize that we are pioneering and will make some mistakes, so we should proceed with caution through a lengthy pilot project stage. However, we now have the expertise to develop and implement a good surveillance given the resources and time to do so.
REFERENCES


THE FALLACY OF DRAWING INFERENCES FROM BIASED DATA:
SOME CASE EXAMPLES

William R. McCallon (DVM)
and
Victor C. Beal, Jr. (PhD)
Principal Staff Officer (Veterinary Economist),
Emergency Programs, and Biometrician,
National Program Planning Staffs;
VS, APHIS, USDA, 6505 Belcrest Road
Hyattsville, MD 20782

There are various needs for disease data that pertain to domestic farm animals. These needs include estimates of prevalence, incidence, and economic loss. These needs have generally been more of a problem for non-program diseases than for program diseases. Animal disease data drawn from various sources has been used in an attempt to meet these needs.

SOURCES OF DATA

In general, this needed data has come from three general sources. These sources are the records of on-going control or eradication programs, special surveys, and traditional animal disease reporting sources. Traditional animal disease sources include data from practicing veterinarians, diagnostic laboratories, and data that does not pertain directly to funded animal disease control programs, but is collected as an ancillary to these programs. The data from veterinarians and diagnostic laboratories used to be compiled in a report called the National Report of Animal Diseases (NRAD) while the ancillary data from disease control programs continues to be compiled in the National Morbidity Report (NMR), (Schroeder, et al. 1966). The NRAD was discontinued in 1972 due to biases which were inherent in the data (Beal, 1980).

EXAMPLES EXAMINED

The purpose of this paper is to examine the quality of information that may be obtained from the traditional sources and from certain non-representative random sample surveys. Many years of experience in attempting to work with biased data has led to the conclusion that attempting to draw inferences from such data is fraught with danger.

Four examples will be examined. They deal with chorioptic scabies in cattle, anaplasmosis, trichinae in swine, and pseudorabies in swine. The first two examples deal with the problem of biases from traditional types of disease data. The third example deals with a mixture of incorrect premises drawn from traditional sources and faulty surveys while the last example deals with biases arising from the incorrect examination of surveys of special populations. In essence, these examples compare information from complete censuses, random sample surveys, and traditional hit-and-miss reporting. Various lessons were learned from these examples.
FALLACY OF DRAWING INFERENCES

FAILURE OF TRADITIONAL SYSTEMS TO PROVIDE PREVALENCE ESTIMATES

The first lesson was to learn that one cannot estimate prevalence from a traditional reporting system. In emphasizing this lesson, the first example deals with chorioptic cattle scabies. In late 1967, APHIS and the State of Vermont decided to embark on a small chorioptic cattle scabies program. In doing this, a complete census of the herds was done for the mite. The disease seemed to be on the increase in the state, as only 2 cases had been reported in FY 1966, and 5 cases were reported in FY 1967. Nationally, the disease also seemed to be on the increase from 213 cases in FY 1966 to 287 cases in FY 1967 (U.S. Dept. Agric.) This is shown in Table 1.

Table 1. Distribution of Chorioptic Cattle Scabies in Vermont and the United States for 1966-1969.

<table>
<thead>
<tr>
<th>Month</th>
<th>Vermont</th>
<th>United States</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>0 0 0 3</td>
<td>50 67 55 74</td>
</tr>
<tr>
<td>February</td>
<td>0 0 345 2</td>
<td>23 32 367 49</td>
</tr>
<tr>
<td>March</td>
<td>0 1 321 3</td>
<td>23 29 358 17</td>
</tr>
<tr>
<td>April</td>
<td>0 2 464 6</td>
<td>3 18 477 22</td>
</tr>
<tr>
<td>May</td>
<td>0 2 1 7</td>
<td>12 14 17 7</td>
</tr>
<tr>
<td>June</td>
<td>0 0 1 0</td>
<td>1 1 3 6</td>
</tr>
<tr>
<td>July</td>
<td>0 0 0 1</td>
<td>6 7 3 4</td>
</tr>
<tr>
<td>August</td>
<td>0 0 0 0</td>
<td>7 19 3 4</td>
</tr>
<tr>
<td>September</td>
<td>0 0 0 0</td>
<td>57 56 3 4</td>
</tr>
<tr>
<td>October</td>
<td>0 0 0 1</td>
<td>18 5 8 2</td>
</tr>
<tr>
<td>November</td>
<td>0 1 0 0</td>
<td>29 18 11 7</td>
</tr>
<tr>
<td>December</td>
<td>0 0 1 1</td>
<td>8 7 22 6</td>
</tr>
<tr>
<td>Fiscal Year</td>
<td>2 5 1,133 6</td>
<td>213 287 1,389 225</td>
</tr>
<tr>
<td>Calendar Year</td>
<td>0 6 1,133 8</td>
<td>237 274 1,327 202</td>
</tr>
</tbody>
</table>

Data from NMR, United States Dept. Agric.

Though only 1 case had been reported in the preceding 8 months, an intensive inspection program was started in Vermont in February of 1968. In less than 3 months, 1,130 laboratory confirmed infected premises were found. This represented about 17% of their cattle herds. The program was not completed, as funding requirements for that much infection were not available. If one had no other information in examining Table 1, it would be easy to infer that chorioptic cattle scabies had dramatically increased in prevalence in the United States by observing the increase from 287 reported cases in FY 1967 to 1,389 reported cases in FY 1968.

However, when one observed what happened in Vermont, it was ap-
parent that the increase in prevalence was mainly in Vermont. The small state of Vermont disclosed four times as much disease as the other 49 states. It would seem that Vermont really had an outbreak and the other states did not. The fact that there were trained people looking for the disease accounted for the increase. This could not be ascertained from the routine NMR.

FAILURE OF TRADITIONAL SYSTEMS TO PROVIDE RELATIVE PREVALENCE

Thus, it has been learned that one could not determine prevalence from a traditional reporting system, but there was still some hope that relative prevalence could be estimated. The second example deals with anaplasmosis. A national probability sample survey was completed for this disease in 1972. This survey used brucellosis market cattle blood samples. In correlating these results with the traditional United States reporting system and laboratory tests, it was learned that relative prevalence between areas or states could not be determined with data from a traditional reporting system or laboratory test summaries.

Table 2 (Beal, 1980) shows data from the old NRAD and from the national survey. It can be seen that Arkansas had been reporting over 36% of the anaplasmosis in the U.S. while California was reporting less than 1% in the old NRAD. However, the survey revealed 5.3% for Arkansas and 10.4% for California. Similar discrepancies were found for the other high prevalence states. It is interesting to note that laboratory test results for the states of California and Arkansas seemed to substantiate the traditional reporting system. The majority of those tested from Arkansas were positive, while only a small percent of those from California were.

<table>
<thead>
<tr>
<th>Location</th>
<th>NRAD Ave. No. Cases Per Fiscal Yr.</th>
<th>Probability Survey Estimate of Reactors in 1,000's</th>
<th>Percent Reactors</th>
<th>Percent of Infection in United States</th>
<th>NRAD Survey</th>
</tr>
</thead>
<tbody>
<tr>
<td>United States</td>
<td>7,136</td>
<td>5,640</td>
<td>10</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Arkansas</td>
<td>2,597</td>
<td>297</td>
<td>22</td>
<td>36.4</td>
<td>5.3</td>
</tr>
<tr>
<td>California</td>
<td>48</td>
<td>588</td>
<td>31</td>
<td>0.7</td>
<td>10.4</td>
</tr>
<tr>
<td>Louisiana</td>
<td>1,011</td>
<td>240</td>
<td>23</td>
<td>14.2</td>
<td>4.3</td>
</tr>
<tr>
<td>Mississippi</td>
<td>139</td>
<td>364</td>
<td>23</td>
<td>1.9</td>
<td>6.5</td>
</tr>
<tr>
<td>Missouri</td>
<td>155</td>
<td>368</td>
<td>12</td>
<td>2.1</td>
<td>6.5</td>
</tr>
<tr>
<td>Oregon</td>
<td>136</td>
<td>92</td>
<td>13</td>
<td>1.9</td>
<td>1.6</td>
</tr>
<tr>
<td>Texas</td>
<td>809</td>
<td>1,086</td>
<td>15</td>
<td>11.3</td>
<td>19.3</td>
</tr>
</tbody>
</table>
However, it was later learned that the samples submitted to the laboratory from Arkansas were mainly from cattle suspected of having the disease. The samples submitted to the laboratory from California were mainly from dairy heifers destined to be exported to Hawaii. The disease had been eradicated there, and they required a negative test. Apparently the brokers in the business of gathering dairy heifers for export to Hawaii knew where to find herds with no history of the disease.

FALLACY OF PRECONCEIVED BELIEFS

The third example deals with trichinae in swine. The lesson learned here is that one should be a little skeptical about traditional beliefs concerning disease prevalence. Tables 3 through 5 show the pertinent data. This information is derived from Jefferies, et al. (1966), Zimmerman (1971), and Zimmerman and Zinter (1971).

The United States is one of the few developed countries in the world with no control or eradication program for trichinae in swine. It was generally believed that the disease was, for the most part, centered in garbage-fed swine and, for this reason, would be fairly easy to control or eradicate. Earlier non-random surveys seemed to substantiate this belief. However, it was felt to be necessary to obtain better information.

In 1965-66, APHIS conducted a random probability sample survey in garbage fed swine for the disease (Jefferies, et al. 1966). Additionally, two random sample surveys of slaughter swine were conducted by meat inspection (MPI) from 1966-70 (Zimmerman, 1971 and Zimmerman and Zinter, 1971). Basic prevalence data from these surveys is shown in Table 3. It can be seen that the rate for garbage fed swine was higher in the 1961-1966 MPI survey than in the 1965-1966 APHIS survey or the 1966-1970 MPI survey. There was doubt about the true randomness of the "garbage fed" swine in the first MPI survey.

Table 3. Results from Various Slaughter Surveys for Trichinae in Swine.

<table>
<thead>
<tr>
<th>Number Tested</th>
<th>Percent Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>Positive</td>
</tr>
<tr>
<td>1961-1966 MPI Survey</td>
<td></td>
</tr>
<tr>
<td>Butcher Swine</td>
<td>9,625</td>
</tr>
<tr>
<td>Breeder Swine</td>
<td>7,394</td>
</tr>
<tr>
<td>&quot;Garbage Fed&quot; Non Random</td>
<td>5,598</td>
</tr>
<tr>
<td>1965-1966 National APHIS Garbage Survey; Random</td>
<td>955</td>
</tr>
<tr>
<td>1966-1970 MPI Survey</td>
<td></td>
</tr>
<tr>
<td>Butcher Swine; Random</td>
<td>20,003</td>
</tr>
<tr>
<td>Breeder Swine; &quot;</td>
<td>1,858</td>
</tr>
<tr>
<td>All Grain Fed; &quot;</td>
<td>21,861</td>
</tr>
<tr>
<td>Garbage Fed; &quot;</td>
<td>590</td>
</tr>
</tbody>
</table>

*APHIS National Garbage Survey value is a weighted population estimate.
Table 4. Results from Various Slaughter Surveys for Trichinae in Swine.

<table>
<thead>
<tr>
<th>Number of Trichinae Per Gram of Diaphragm</th>
<th>Less Than 1</th>
<th>1 to 10</th>
<th>10 to 50</th>
<th>50 to 500</th>
<th>Over 500</th>
</tr>
</thead>
<tbody>
<tr>
<td>1961-1966 MPI Survey – Butcher</td>
<td>11</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>– Breeder</td>
<td>12</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>– “Garbage Fed”</td>
<td>81</td>
<td>30</td>
<td>10</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>1965-1966 National APHIS Garbage Survey</td>
<td>15</td>
<td>6</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1966-1970 MPI Survey – Butcher</td>
<td>17</td>
<td>1</td>
<td>2</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>– Breeder</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>– All Grain Fed</td>
<td>17</td>
<td>1</td>
<td>2</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>– Garbage Fed</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 5. Estimated Number of Slaughter Hogs with Trichinae Based Upon Prevalence Rates from National Garbage Survey and 1966-1970 MPI Survey in Grain Fed Swine.

<table>
<thead>
<tr>
<th>Estimated Trichinae Infested Swine</th>
<th>Estimated Trichinae Infested Swine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swine Slaughtered in 1981</td>
<td>Total Number</td>
</tr>
<tr>
<td>Total Swine</td>
<td>91,546,600</td>
</tr>
<tr>
<td>Grain Fed Swine</td>
<td>90,846,600</td>
</tr>
<tr>
<td>Garbage Fed Swine</td>
<td>700,000</td>
</tr>
</tbody>
</table>

aAbove data is based upon 50 gram samples from the diaphragm. Number of swine with infective doses is based upon the assumption that one trichinae per gram is infectious. (page 944, Zimmerman and Zinter, 1971).

As can be seen from Table 5, these surveys revealed an estimated 3,500 garbage fed infected swine slaughtered but 103,891 infected grain-fed swine. Only a little over 3% of the trichinae-infected swine were garbage fed. Many still thought that the garbage-fed swine would be more heavily infected and thus more likely to transmit the disease to humans, but the garbage-fed swine constituted only 4% of the heavily infected carcasses.

FALLACY OF IGNORING HERD SIZE DIFFERENCES

The final example concerns the important role of herd size in the prevalence of animal diseases. Most domestic diseases involve animal-to-animal transmission. Herds with the greatest number of herd additions have a greater likelihood of becoming infected. Larger herds tend to have more herd additions. In view of this, one cannot extrapolate prevalence...
disclosed from herd surveys to the national herd unless herd size is taken into consideration.

Beginning about 10 years ago, Pseudorabies seemed to be increasing in prevalence and economic importance. A mail survey was conducted of the National Pork Producers Council members in an attempt to gain information concerning prevalence and losses (Gustafson and Sherba, 1978). Their response was good, and the survey did provide useful information. However, in attempting to estimate the prevalence, it was overestimated by almost 100%. Tables 6 through 10 show the results of the survey and the extrapolation to the United States population of swine sold. Table 6 shows a basic summary of the data.

Table 6. Pseudorabies Survey of National Pork Producers Council Members, Number of Herds and Number of Animals Sold by Herds in the Survey.

<table>
<thead>
<tr>
<th>Type of Animals Sold</th>
<th>Number of Herds</th>
<th>Number of Animals Sold From</th>
<th>Number of Animals Sold</th>
<th>Infected</th>
<th>Uninfected</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>herds</td>
<td>Herds</td>
<td>Herds</td>
</tr>
<tr>
<td>Commercial</td>
<td>74</td>
<td>1,983</td>
<td>2,057</td>
<td>105,890</td>
<td>1,899,740</td>
<td>2,005,630</td>
</tr>
<tr>
<td>Feeder</td>
<td>13</td>
<td>529</td>
<td>542</td>
<td>23,860</td>
<td>403,861</td>
<td>427,721</td>
</tr>
<tr>
<td>Seed-Stock</td>
<td>8</td>
<td>243</td>
<td>251</td>
<td>4,800</td>
<td>75,908</td>
<td>80,708</td>
</tr>
<tr>
<td>Total</td>
<td>81</td>
<td>2,340</td>
<td>2,421</td>
<td>134,550</td>
<td>2,379,509</td>
<td>2,514,059</td>
</tr>
</tbody>
</table>

-Number of herds does not add up to total because a number of herds sold two or more types of animals.

As expected and as can be seen from Tables 7 and 8, the survey revealed that larger herds were more likely to be infected. It can be seen that the average size of an infected herd is 1,661 animals sold while the average size of an uninfected herd is 1,017 animal sold.

Table 7. Pseudorabies Survey of National Pork Producers Council Members, Average Number Sold by Various Classes of Herds.

<table>
<thead>
<tr>
<th>Classification of Herds</th>
<th>Number of Herds</th>
<th>Number of Animals Sold</th>
<th>Average Sold Per Herd</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Herds</td>
<td>2,421</td>
<td>2,514,059</td>
<td>1,038</td>
</tr>
<tr>
<td>Uninfected Herds</td>
<td>2,340</td>
<td>2,379,509</td>
<td>1,017</td>
</tr>
<tr>
<td>Infected Herds</td>
<td>81</td>
<td>134,550</td>
<td>1,661</td>
</tr>
<tr>
<td>Infected Herds Reporting Losses</td>
<td>52</td>
<td>97,910</td>
<td>1,883</td>
</tr>
<tr>
<td>Inf. Herds Not Reporting Losses</td>
<td>29</td>
<td>36,640</td>
<td>1,263</td>
</tr>
</tbody>
</table>

Table 7 shows data on animals sold per herd while Table 8 shows data both on animals sold per herd and animals sold per sales class. As can be
seen from Table 6, there were three classes of animals sold, namely commercial swine, feeder swine, and seed-stock swine.

Table 8. Pseudorabies Survey of National Pork Producers Council Members, Average Number of Animals Sold Per Type of Animal Sale Group and Per Total Herd.

<table>
<thead>
<tr>
<th>Infection Status</th>
<th>Number of Animals Sold</th>
<th>Number of Type of Sale Groups</th>
<th>Number of Herds</th>
<th>Average Per Sale Type</th>
<th>Average Per Herd</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Herds</td>
<td>2,514,059</td>
<td>2,850</td>
<td>2,421</td>
<td>882</td>
<td>1,038</td>
</tr>
<tr>
<td>No Inf. Reported</td>
<td>2,379,509</td>
<td>2,755</td>
<td>2,340</td>
<td>864</td>
<td>1,017</td>
</tr>
<tr>
<td>Infection Reported</td>
<td>134,550</td>
<td>95</td>
<td>81</td>
<td>1,416</td>
<td>1,661</td>
</tr>
<tr>
<td>Loss Information Reported</td>
<td>97,910</td>
<td>61</td>
<td>52</td>
<td>1,605</td>
<td>1,883</td>
</tr>
</tbody>
</table>

Data for the infected herds with loss information reported was available on the actual herds. However, information on all herds was available only for class of animal sold. Consequently, extrapolations to the United States population of animals sold as shown in Table 10 is on the basis of class of animal sold in the survey. Data on animals sold in the United States is from the 1974 Census of Agriculture and is shown in Table 9.

When herd size is not accounted for, it is estimated that 3,111,604 swine are infected. When herd size is considered, a lesser number of 1,558,212 swine are estimated to be infected. We are fortunate in the U.S. in that reliable data is available concerning herd size and marketing patterns. This information should be used, when applicable, in estimating prevalence.

Table 9. Data on Swine Sold From 1974 Agricultural Census.

<table>
<thead>
<tr>
<th>Number Swine Sold Per Farm</th>
<th>Total Number of Swine Sold</th>
<th>Percent Sold</th>
<th>Number of Herds With Sales</th>
<th>Average Number Sold Per Herd</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 to 4</td>
<td>65,638</td>
<td>0.08</td>
<td>27,205</td>
<td>2.41</td>
</tr>
<tr>
<td>5 to 9</td>
<td>193,447</td>
<td>0.24</td>
<td>28,181</td>
<td>6.86</td>
</tr>
<tr>
<td>10 to 24</td>
<td>1,077,186</td>
<td>1.35</td>
<td>66,544</td>
<td>16.19</td>
</tr>
<tr>
<td>25 to 49</td>
<td>2,254,390</td>
<td>2.82</td>
<td>64,038</td>
<td>35.20</td>
</tr>
<tr>
<td>50 to 99</td>
<td>5,213,079</td>
<td>6.52</td>
<td>74,339</td>
<td>70.13</td>
</tr>
<tr>
<td>100 to 199</td>
<td>10,447,765</td>
<td>13.08</td>
<td>75,548</td>
<td>138.29</td>
</tr>
<tr>
<td>1 to 199</td>
<td>19,251,505</td>
<td>24.10</td>
<td>335,855</td>
<td>57.32</td>
</tr>
<tr>
<td>200 to 499</td>
<td>23,222,844</td>
<td>29.07</td>
<td>77,077</td>
<td>301.29</td>
</tr>
<tr>
<td>500 to 999</td>
<td>17,348,768</td>
<td>21.71</td>
<td>26,111</td>
<td>664.24</td>
</tr>
<tr>
<td>1,000 to 1,999</td>
<td>10,537,645</td>
<td>13.19</td>
<td>8,194</td>
<td>1,286.02</td>
</tr>
<tr>
<td>2,000 to 4,999</td>
<td>6,133,135</td>
<td>7.68</td>
<td>2,230</td>
<td>2,750.28</td>
</tr>
</tbody>
</table>
FALLACY OF DRAWING INFERENCES

Table 10.
Expansion of Estimate of Swine Sold from Herds Reporting Loss Information from Pseudorabies Survey to Entire United States Swine Population of Animals Sold.

<table>
<thead>
<tr>
<th>Number of Swine Sold Per Herd</th>
<th>Number of Swine (Actual Loss in Loss Info Total From Loss)</th>
<th>Percent in Loss</th>
<th>Number of Swine (Total Sold From Loss Sold Type Herds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Per Herd</td>
<td>Survey Grouped according to Herd Groups</td>
<td>Sales Class Data Herds</td>
<td>Percent in Loss Info Herds</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----------------------------------------------------------</td>
<td>----------------</td>
<td>-----------------------------------------------------</td>
</tr>
<tr>
<td>1 to 199</td>
<td>47,394</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>200 to 499</td>
<td>242,291</td>
<td>2,710</td>
<td>1.118</td>
</tr>
<tr>
<td>500 to 1,000</td>
<td>457,813</td>
<td>10,300</td>
<td>2.250</td>
</tr>
<tr>
<td>1,000 to 1,999</td>
<td>627,080</td>
<td>15,600</td>
<td>2.488</td>
</tr>
<tr>
<td>2,000 to 2,999</td>
<td>301,880</td>
<td>12,500</td>
<td>4.141</td>
</tr>
<tr>
<td>3,000 to 4,999</td>
<td>227,200</td>
<td>28,000</td>
<td>12.324</td>
</tr>
<tr>
<td>5,000 and Over</td>
<td>610,400</td>
<td>28,800</td>
<td>4.718</td>
</tr>
<tr>
<td>Total</td>
<td>2,514,058</td>
<td>97,910</td>
<td>4.718</td>
</tr>
</tbody>
</table>

*Straight Line Estimate = (97,910 / 2,514,058) × 79,897,397 = 3,111,604. The Straight Line Estimate is in Error by 99.69 Percent.

SUMMARY

Some fallacies of drawing inferences from various types of biased data have been discussed. Several examples have been examined. It has been shown that animal disease data of the traditional nature contains various inherent biases. In addition, it has been shown that herd size distribution must be considered when surveys are conducted that originate from populations that are not representative of the population of interest.

REFERENCES

garbage fed swine. Proc. of 70th annual meeting of the United States Livestock Sanitary Association: pages 349-357


General agreement on the utility of probability sampling in a national annual disease and production economics surveillance system has become apparent in the community of interested parties and individuals. APHIS, Veterinary Services has presented a time table for implementing such a plan and proposes to conduct a pilot project forthwith in several states. There follows a brief outline on the issues which a statistician might raise and resolve during the planning of such a project. They are divided into four segments by a logic of chronological occurrence.

SAMPLING UNITS

The statistical thought process proceeds after there is agreement on the definition of the specific entity which can be drawn into the sample. Sampling units must be locatable to possibly be drawn. In order to conduct the random drawing of a sample, it must be possible to describe the total population of all such units. This list is often referred to as the frame. It must be possible to easily and accurately observe the desired data in any located sampling unit. Most sampling plans make use of stratification of the population of sampling units. It must be possible to accurately assign any sampling unit to only one specific strata and the number of sampling units in each strata must be known in the frame. It must be possible to exactly locate the relative position of any defined sampling unit in a hierarchical classification scheme for the population.

STATISTICAL SPECIFICATION

It might be said that a sampling plan is defined when it is possible to state every sampling unit’s probability of being in the sample. The specific random processes proposed for sampling the frame will lead to different costs and consideration of efficiency in estimation. It is necessary to come to an agreement on what plan is workable and adequate. Each sampling unit has a measurable multivariate response variable and it is necessary to know the dimension and nature of this variable. Are certain dimensions categorical and others continuous? Are certain dimensions observed correctly with certainty or with certain probability? Can the proposed plan produce information leading to attainment of study objectives?

ESTIMATION

Estimators of target population parameters have to be formulated and their statistical sampling distributions must be determined. This may be done algebraically in closed forms or it may be necessary to use ap-
proximations based on serial expansions. It may be necessary to develop data analytic procedures (e.g. the boot strap or the jack knife) which will produce confidence bounds.

COMPILATION

A standardized and mechanical procedure for inputing field collected data must be implemented which will survive changes in personnel. Such a system must be able to survive occasional errors and computer breakdowns. An administrative structure must be established for the personnel involved. The computer programs (or software package) must be designed or acquired to meet all foreseeable needs and technical staff needed to implement them must be determined. Basic long run decisions on acquisition of or access to computing facilities must be made.
The Minnesota Food Animal Disease Reporting System (MFADRS) was implemented in 1971 and in effect until 1979. During 1979-80 the system was validated following eight years of ongoing activity.

A National Disease Surveillance System developed by the United States Department of Agriculture was endorsed by the United States Animal Health Association in 1981. Its plan calls for a modification of the Minnesota Food Animal Disease Reporting System. This proposal indicates the need to identify up to 20 most prevalent diseases in each of the food animal species, beginning with cattle and swine.

MFADRS developed reporting in species and age (life stage) categories as follows:

**Swine:**
- Farrowed to finishing — (up to 8 months)
- Breeding stock — (sows, boars and gilts)

**Dairy Cattle:**
- Calves — (up to 1 year of age)
- Heifers — (1-2 years)
- Adults — (2 years and over)

**Beef cow-calf herds:**
- Calves — (up to 1 year of age)
- Heifers — (1-2 years of age)
- Adults — (over 2 years of age)

**Beef feedlot:** (all ages)

**Sheep:**
- Lambs — (up to 8 months)
- Breeding sheep — (over 8 months)

Between 1971-1980 the following information was collected by the Minnesota Food Animal Disease Reporting system: herd mortality, incidence per hundred herds, herd case incidence per hundred herds, herd mortality, incidence per thousand animals, and herd case incidence per thousand animals.

The following ranks the 20 most prevalent diseases and conditions reported in Minnesota swine during 1974-1980. Swine was selected as an example.
### SWINE HERD RANKINGS OF DISEASE INCIDENCE*

(Cases/1000 Head)

<table>
<thead>
<tr>
<th>Farrowed to Finishing Pigs</th>
<th>Breeding Swine</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mange</td>
<td>1. Mange</td>
</tr>
<tr>
<td>2. Colibacillosis</td>
<td>2. Mastitis, Metritis, Agalactia</td>
</tr>
<tr>
<td>3. Swine Influenza</td>
<td>3. SMEDI</td>
</tr>
<tr>
<td>4. Diarrheal Disease</td>
<td>4. Pediculosis</td>
</tr>
<tr>
<td>(Undetermined)</td>
<td></td>
</tr>
<tr>
<td>5. Pigs Killed by Sow</td>
<td>5. Arthritis Disease</td>
</tr>
<tr>
<td></td>
<td>(Undetermined)</td>
</tr>
<tr>
<td>6. Roundworms</td>
<td>6. Dystocia (Producer Delivered)</td>
</tr>
<tr>
<td>7. Atrophic Rhinitis</td>
<td>7. Abscesses</td>
</tr>
<tr>
<td>8. Pediculosis</td>
<td>8. Abortion (Undetermined)</td>
</tr>
<tr>
<td>10. Sudden Deaths (Undetermined)</td>
<td>10. Atrophic Rhinitis</td>
</tr>
<tr>
<td>11. Mycoplasma Pneumonia</td>
<td>11. Dystocia (Veterinarian Delivered)</td>
</tr>
<tr>
<td>12. Abscesses</td>
<td>12. Roundworms</td>
</tr>
<tr>
<td>13. Swine Dysentery</td>
<td>13. Parasites Internal</td>
</tr>
<tr>
<td></td>
<td>(Undetermined)</td>
</tr>
<tr>
<td>15. Streptococcal Arthritis</td>
<td>15. Sudden Deaths</td>
</tr>
<tr>
<td></td>
<td>(Undetermined)</td>
</tr>
<tr>
<td>17. Edema Disease</td>
<td>17. Swine Influenza</td>
</tr>
<tr>
<td>18. Arthritis Disease (Undetermined)</td>
<td>18. Erysipelas</td>
</tr>
<tr>
<td>19. Parasites Internal (Undetermined)</td>
<td>19. Polyserositis</td>
</tr>
<tr>
<td>20. Tail Biting</td>
<td>20. Porcine Stress Syndrome</td>
</tr>
</tbody>
</table>

*Ranked from highest to lowest from quarterly incidence rates (1974-1980).
A NATIONAL DISEASE SURVEILLANCE SYSTEM—STATUS REPORT
William R. McCallon

Last year this committee decided that APHIS should begin the development of a national animal disease surveillance system and report back this year as to time frame, potential for funds, and methodology.

TIME FRAME AND FUNDS

1983
We can begin a pilot project in two states. A final decision has not been made as to which two states, though several have been contacted and several have expressed interest. Some of the factors we feel to be important are: (1) a good mix of dairy cattle, beef cattle, and swine, (2) an interest in the project, (3) University and Laboratory Support, and (4) reasonably close to Washington as we will have to make frequent visits in the initial stages.

Currently our budget seems adequate to cover this stage.

1984
We can enlarge the project to five states—one in each of our five regions. We have requested adequate funds for this expansion and are reasonably confident they will be made available.

1985-88
We can continue the project in these states and perhaps add an additional state in each region. If the states are well selected, we may be able to begin making some inferences concerning prevalence incidence, trends, and economic significance.

We have no assurance of funds necessary for operations at that size, but are reasonably optimistic.

1990
At this time we should have the experience to implement a national system and a very good estimation of what we expect to get out of the system and what it will cost.

We can not predict whether or not the system will be funded.

METHODOLOGY

It should be made clear that the first years of the project will be devoted to developing, testing and refining methodology. The two major principles are that the sample herds must be representative of the herds in their area and the data must be collected in a uniform and timely manner. This strongly implies random sampling and data collection by State, Federal, and University employed Veterinarians, who have had special training.

THE SAMPLE

Initially, the system will cover beef cattle, dairy cattle, and swine.
A NATIONAL DISEASE SURVEILLANCE SYSTEM—STATUS REPORT
William R. McCallon

Last year this committee decided that APHIS should begin the development of a national animal disease surveillance system and report back this year as to time frame, potential for funds, and methodology.

TIME FRAME AND FUNDS

1983
We can begin a pilot project in two states. A final decision has not been made as to which two states, though several have been contacted and several have expressed interest. Some of the factors we feel to be important are: (1) a good mix of dairy cattle, beef cattle, and swine, (2) an interest in the project, (3) University and Laboratory Support, and (4) reasonably close to Washington as we will have to make frequent visits in the initial stages.

Currently our budget seems adequate to cover this stage.

1984
We can enlarge the project to five states—one in each of our five regions. We have requested adequate funds for this expansion and are reasonably confident they will be made available.

1985-88
We can continue the project in these states and perhaps add an additional state in each region. If the states are well selected, we may be able to begin making some inferences concerning prevalence incidence, trends, and economic significance.

We have no assurance of funds necessary for operations at that size, but are reasonably optimistic.

1990
At this time we should have the experience to implement a national system and a very good estimation of what we expect to get out of the system and what it will cost.

We can not predict whether or not the system will be funded.

METHODOLOGY

It should be made clear that the first years of the project will be devoted to developing, testing and refining methodology. The two major principles are that the sample herds must be representative of the herds in their area and the data must be collected in a uniform and timely manner. This strongly implies random sampling and data collection by State, Federal, and University employed Veterinarians, who have had special training.

THE SAMPLE

Initially, the system will cover beef cattle, dairy cattle, and swine.
Potential benefits will not be fully realized unless the results are used by livestock owners in formulating health plans. We intend working very closely with the Extension Service and the trade press.
REPORT OF THE COMMITTEE ON MORBIDITY AND MORTALITY

Chairman: G. C. Poppensiek, Ithaca, NY
Vice Chairman: G.P. Combs, Puerto Rico

P.N. Acha, D.C.; V.C. Beal, Jr., MD; H.E. Binks, MD; A.A. Case, MO; J.J. Doherty, WA; J.G. Flint, MN; F.M. Hamdy, NY; F.E. Hemphill, MD; C.M. Hibbs, NM; M.E. Hugh-Jones, LA; D.E. Hughes, SD; N.E. Hutton, OR; A.F. Kaufman, GA; L. King, TX; D.C. Kradel, NC; L. Mark, VA; H.A. McDaniel, MD; W.R. Miller, AL; L.G. Morehouse, MO; T.G. Murnane, DF; J.C. New, TN; S.R. Nusbaum, NJ; E.I. Pilchard, MD; J.R. Reagan, TN; Philip Ross, D.C.; L. Russell, TX; V.A. Seaton, IA; G.H. Snoeyenbos, MS; C.D. Van Houweling, VA; K.D. Weide, MO; S.L. Diesch, MN; R.K. Anderson, MN; J.L. Hyde, MD; R.F. Kahrs, FL; W.R. McCallon, MD; E.H. McCauley, MT; C.M. Hibbs, NM.

The chairman called the meeting of the Committee on Morbidity and Mortality Reporting, Food-Animal Diseases, to order at 1:30 p.m. Monday, November 8, 1982. The following six papers were presented to the committee:

Dr. Robert K. Anderson—"Historical Review of and Meaningful Data for Animal Disease Surveillance."

Dr. Frank B. Martin—"Random Sampling: Why Do It and How It Should Be Done."

Dr. William R. McCallon—"Fallacy of Drawing Inferences From Biased Data," "Status of the APHIS 5-year plan on the National Animal Disease Surveillance System."

Dr. Stanley L. Diesch—"Retrospective Indentification of the Twenty Diseases of Highest Incidence from the Minnesota Disease Reporting System."

Dr. Roger S. Morris—Gave a discussion on the collection of animal production data.

Discussion of the papers followed their presentation. The committee then excused all guests and went into executive session.

The committee makes the following recommendations:

1. That the time table as presented in the APHIS five-year Animal Disease Surveillance plan be followed.

2. That all State and Federal Veterinarians in charge, American Veterinary Libraries, and all committee members be provided a copy of the Minnesota Food Animal Disease Reporting System Validation Study by USDA, APHIS.

3. That public communication with interested groups be made a part of the five-year plan.

4. That a subcommittee be formed to study existing state regulations.
concerning reportable animal diseases. This committee is to report its findings and attempt to develop by next year a model regulation for guidance of state officials. Dr. Sidney R. Nusbaum was named chairman of this subcommittee.

The committee demonstrated approval of the statistical random sample methodology for the Animal Disease Surveillance plan currently under development by APHIS.

Two resolutions were made for transmission to the executive committee.

The chairman adjourned the meeting at 5:45 p.m.
When the original Food, Drug and Cosmetic Act was passed in 1938 manufacturers duly lined up to register their drug products with the Food and Drug Administration (FDA). Requirements for New Animal Drug Application approvals were quite modest. Most applications consisted of a few dozen pages and were approved within a week or two while more difficult files were handled in a month or so. Regulatory action against firms who failed to register their existing products were unnecessary because all pre-38 drugs were exempted from registration.

Congress also provided in the 1938 Act two classes of drugs: 1) new animal drugs and 2) all drugs other than new. New drugs were defined as those not generally recognized as safe and effective by qualified experts. No definition was provided for other than new drugs because they were exempted from registration and thus no definition was needed.

As the years passed the FDA became increasingly demanding in its requirements for registration of new animal drugs. Applications for approval of new substances grew to 300, sometimes 500 or 600 pages by 1955. Meanwhile enforcement actions against firms who produced the same products they had made prior to 1938 were simply avoided because, after all, Congress had excluded them from the registration provisions of the Act. Nevertheless, the therapeutic revolution had changed the character of the regulated industry and FDA gradually felt increasingly compelled to take action against firms who copied recently approved drugs and marketed them as "other than new." Simultaneously, new firms appeared in the industry and they began to produce accurate and not so accurate copies of the pre-38 drugs which FDA had traditionally viewed as "grandfathered" through exclusion from registration.

By 1965 FDA had a fairly healthy effort going to take legal action against firms who marketed new versions of pre-38 drugs (especially if the pre-38 drug had fallen into disfavor in the scientific community) and against firms who copied but failed to register drugs which had only recently been approved.

In the 1970's the enforcements division of FDA's Bureau of Veterinary Medicine (BVM) developed into a major part of BVM's activities and by the end of the Carter administration enforcements were at a feverish pitch. Regulatory letters increased year by year and by 1980 hundreds of traditional drugs and me-too versions of traditional drugs had been removed from the market through seizures, litigation and voluntary compliance.

While this was occurring, FDA's requirements for evidence of safety continued to evolve so that by 1962 applications for new drugs usually consisted of six or seven volumes of data from studies specifically per-
formed for the purpose of identifying the toxicity and pharmacology of the proposed new product. In that same year Congress amended the FD&C Act in such a manner as to require FDA to assure each new drug was also effective. Thus applications for approval continued to grow in size after '62 and by 1970 most applications were 15 to 30 volumes per copy. As it had done in 1938, Congress again exempted drugs marketed prior to the Act from its provisions provided that such drugs were generally recognized as safe. FDA then required all new drugs which were being marketed be reviewed for effectiveness and notified holders of approved NADAs that they should submit their drugs for effectiveness review by the National Academy of Science (NAS). This was duly done by most firms on those products for which NDA or NADA approvals were in effect but few, if any, of the pre-38 and "old drugs" were submitted for review. Most of the drug products found "effective" or "probably effective" by the NAS remain on the market today.

But in the '70s FDA decided that practically all those copies of NAS reviewed drugs and all those pre-38 drugs needed to be regulated as if they were new drugs and so the expansion of enforcements was necessary. A very confused industry reacted by crying "foul" whenever FDA decided to take away another copy of its pre-38, pre-62 or "generally recognized as safe" old drugs.

FDA's actions were isolated, disorganized and often explainable only as pure coincidence even though frequently viewed by smaller firms as vindictive. A better brew for litigation can hardly be imagined. But surprisingly it was a human house rather than a veterinary house that first challenged the idea that all drug products required specific FDA approvals. That house was Premo Pharmaceutical Laboratories, Inc. The Second Circuit Court of Appeals upheld FDA's position and Judge Lacey took the opportunity to "sermonize on the grave social necessity" of having FDA specifically approve each product because of the important role excipients play in bioequivalency...and thus in safety and effectiveness.

But the dust from the Premo case had hardly settled when a Florida firm decided to challenge the very same principle in the Fifth Circuit. This time the result was the opposite. Now the firm, Generix Drug Corporation, successfully argued that under FDA's interpretation of the Act (and Judge Lacey's) all drug products are new drug products. That was exactly what J. Richard Crout had sworn before Judge Lacey in the Premo case and that was the fatal error in FDA's view. One need not be a lawyer or scientist to realize that if there are no old drugs there can be no new drugs. "Newness," James Dickinson said in an editorial in Pharmaceutical Technology;*"inevitably begets oldness ...", as with the words "near" and "far," the word "new" has no meaning if the word "old" is

stricken from our vocabulary. Since Congress established at least two classes of drugs when it passed the FD&C Act, either FDA is mad or the U.S. Congress is mad. And in judicial circles it is still unpopular to officially declare the U.S. Congress to be mad!

Meanwhile in October of 1981 the United States Pharmacopeial Convention convened a distinguished panel of 13 outside experts and asked them to comment on the "Federal Drug Approval Process." To the horror of FDA's regulatory enthusiasts the panel in its interim report of 20 October sided squarely with Premo and Generix. Indeed, the Panel in effect accused FDA of wasting time and energy by requiring approval of old drugs and told FDA instead to rely on U.S.P. standards, Current Good Manufacturing Practice regulations, FDA plant inspections, and the Drug Listing Act to assure adequate quality for copies of approved drugs.

In this way both pharmaceutical science and the courts came together against FDA's interpretation of the Act. It is not necessary to be critical of any individual or group of individuals when one says the FDA attempts to regulate to the limits permitted by the Act. Expansionist tendencies are seen in all bureaucracies. But FDA went entirely too far and found itself boxed in by both science and law. It was "lift by its own petard."

But the Fifth Circuit opinion in Generix's favor has some far reaching implications. It makes the term "drug" in the FD&C Act refer only to active ingredients, not products. Thus when a drug substance, say oxytetracycline, becomes generally recognized by experts as safe and effective for any use at any dosage, FDA could no longer declare any product containing oxytetracycline to be a "new drug." Such a product might be adulterated because the wrong excipients were used (or were used in the wrong way) or such a product might be misbranded because the claims or dosage were wrong. But regardless of the formula, regardless of the claims, if the product contained oxytetracycline FDA could not demand NDA or NADA approval on the grounds that the product constituted a new drug.

Unfortunately, FDA's commitment to using "new drug" status as its regulatory nite-stick over the last 20 years has brought about an organization and structure which can not be readily changed. Instead of a massive Washington bureaucracy, FDA would have to restructure itself to improve in-plant inspections, increase it's surveillance of the marketplace, give real emphasis to the Drug Listing Act and prove its cases in court under more complex provisions of the Act. FDA had to appeal the decision to the U.S. Supreme Court.

Now let us look at the science of the matter again. Was the USP panel of outside experts correct that compendial standards and GMPs are adequate to assure good drugs? In the majority of the cases it is, but the exceptions in veterinary drugs (I can not speak with confidence about human drugs) give one pause. Compendial standards are not written and can not be quickly written for the 430 active ingredients approved for use in animal drugs not to mention several hundred which are used but not
GENERIC COMPETITION IN ANIMAL HEALTH PRODUCTS

specifically approved by FDA. Because minor differences in bio-
equivalency may be magnified in the pattern of tissue residues, such
standards may have to be far more comprehensive than anyone has yet
imagined. For example, I witnessed an instance in which an apparently
identical formulation of an antibiotic for cattle produced a very different
pre-slaughter withdrawal time. It is conceivable to trust the regulated
industry to conduct reliable tests outlined in the USP without oversight,
but is it really feasible to trust the generic drug industry to conduct
reliable tissue residue studies without oversight? My experience
suggests at least a few firms would abuse such freedom. No doubt FDA
views the matter as dangerous to the integrity of the Republic itself and
the agency has so argued in its brief before the U.S. Supreme Court. A
final ruling on the Generix case is due within the next six months. There
is no doubt the future of FDA as we know it hangs in the balance.* There
is doubt about the impact of the decision on the safety, effectiveness and
cost of our drug supply.

Since the word "cost" has now crept into this paper we can no longer
ignore the economic impact of Generix, Premo, and the upcoming U.S.
Supreme Court Decision.

As noted earlier, FDA's requirements for NADA approvals have
steadily increased over the years and today the industry's reported costs
for clearing a new drug substance are from $5 to $15 million over a 5 to 8
year period. This fearful initial investment must be recouped through
pricing the drug product well above actual manufacturing costs for
several years. Unless a firm's management can clearly foresee a
reasonable return on their investment, they obviously must not develop
the drug. Should Generix prevail at the Supreme Court, the pricing of
drugs "well above actual manufacturing costs" would be possible only if,
and only so long as, patent protection keeps competitive generic houses
out of the market. Remember, if Generix prevails any generic house
could copy and sell any approved drug, animal or human, without NDA or
NADA approval. Only patents could legally keep generics out of the
market. Thus the first effect of a Supreme Court decision favorable to
Generix would be the appearance in the market of a host of generic drugs
at substantially reduced prices. For people who purchase and use the
products, veterinarians, farmers, pet owners, and the like, this would be a
welcome blessing indeed. But unless patent laws are modified to permit
recovery of that $5 to $15 million initial cost, the next generation might
very well view it as a curse. The fountain from which modern medicine
has sprung would mostly dry up. This is simply because the drug in-
dustry has for the last 25 years depended in large part on the FDA, not
patents, to maintain their monopoly long enough to recover their in-
vestment and to make a reasonable profit by demanding full NDAs and/or
NADAs for each generic copy of recently approved brands. Even if there

*Assuming, of course, no change in enabling legislation, a very big assumption indeed.
are no patents, the initial cost of $5-$15 million for FDA approval keeps competitors away and those firms who do decide to spend the money must themselves charge high prices for their products if they too are to recover their initial costs.

Similar rationale apply (on a lesser dollar scale) to the addition of new claims, the introduction of new dosage forms, and the on-going costs to answer new questions raised by FDA about safety and effectiveness. The fact that the pharmaceutical industry does not conduct important research on vitamins, such as vitamin C, should be a fair warning that many promising new drugs and many important uses for existing drugs will be lost if industry can not recover its investments. If all drugs are to become as readily available to manufacturers and consumers as vitamin C and are to be as inexpensive as vitamin C, then the therapeutic revolution will be essentially ended. Drugs will be cheap but future generations will have to more or less make-do with what we have now. Drug research will practically halt in the United States. Certainly research will continue in at least a few other countries, but the great fountain of medical wonders, the United States of America, will cease to be a major factor. FDA has always been a more formidable obstacle than any U.S. patent.

A ruling against Generix could institute an equally distressing problem. If FDA is to require full NDAs and NADAs for all generic drugs then there will be no generic drugs. A permanent monopoly is a great incentive to pharmaceutical research but without competition the costs of drugs would obviously be sustained at a high level.

Presently, this is almost exactly FDA's posture except for drugs marketed before the 1962 amendments to the FD&C Act. While FDA has said it will approve NDAs for duplicates of post '62 drugs on the basis of published literature, this policy has done little to increase competition in veterinary drugs but has inhibited publication of drug research and is of questionable legality. Apparently Commissioner Hayes has appointed a task force to deal with the problems of approving these "paper" NDAs. But one must genuinely wonder how much progress can be made until the FD&C Act is reworded by Congress.

Thus the issue of drug cost transcends economics. The cost of a drastic reduction in drug prices today will probably be paid by our children and grandchildren. A simple decision by the Supreme Court for Generix is no more in the public interest than a simple decision against Generix. Congress never meant for FDA to treat all drugs as "new" and Congress never meant for FDA to destroy the pharmaceutical industry by treating all copies of approved drugs as "old." Congress did mean for FDA to compel research on new drugs to establish their safety and effectiveness and Congress meant for approved drugs to be exempted from the pre-

market registration procedures after such drugs became generally recognized by experts as safe and effective. Newness begets oldness with time. FDA's attempt to treat everything as new is, in my opinion, contrary to the intent and the precise language of the Act, for example, in 40FR26142-. It is certainly contrary to FDA's own previous interpretation of the Act. The controversy over Generix and Premo is the natural result of an agency attempting for force the Act to serve bureaucratic ends.* Drugs can and should drop out of active regulation as new drugs with the passage of time and if FDA personnel will honestly stick to this intent there will be no need either for all drug products to be the subject of approved files or for all generics to be exempted from registration. Each drug product must be viewed through the eyes of experts and each must be viewed under the bright lights of time and in the context of the amount and extent of use. FDA followed this procedure from 1938 to 1962. Since 1962 they have gradually expanded their jurisdiction but half the animal drug products marketed today remain unapproved.

The argument that unless FDA requires registration of each product another "Elixir Sulfonilamide" incident is likely is more of an adversarial legal tactic than a scientific concern. Even the least sophisticated formulaters of 1982 have vastly more information at their disposal than anyone had in 1938. The probability of someone using something like antifreeze as a solvent in drugs today is 1 in 10 million or less. Are we to believe FDA is really trying to preclude that 1 in 10 million risk of a grotesque mistake in formulating while they themselves take far greater risks in approving ANDAs of a dozen or two pages? No, the issue is as remarkably clear as one will ever see. This issue is one of bureaucratic determination to exercise a new regulatory function without enabling legislation, a function that would add to its prestige and enlarge its needs for staff and budgets, a function which would vindicate the agency for having constructed a massive Washington bureaucracy when instead a dispersed bureaucracy for monitoring drug manufacturing was both needed and consistent with the Act. FDA has worked for many years to build up case law under the FD&C Act to give the agency discretion to specifically license each and every drug before that drug goes on the market. The agency knows it can't really do this (it doesn't even want to) but, having the authority to do so has become something of an obsession, an obsession which has precipitated the power struggle known as the Generix Case.

Reading FDA's "Petition For A Writ of Certiorari" in the Generix case

---

*The October 20, 1981 USP Advisory Panel to the Commission on Federal Drug Approval Process also noted that FDA has in recent years conducted "major efforts" to build up case law so as to extend its jurisdiction to "every strength and every dosage form of every drug." FDA's efforts here are in direct conflict with its own position on monographs for OTC human drugs in 21CFR330.10.
is a traumatic experience for the scientific mind. Adversarial pleadings such as this lead one into a shadow world where attempts to mislead are in vogue and while intentional outright falsification is not acceptable, the parties are actually expected to allege circumstances and conditions that only the naive can take seriously. For example, FDA argues (p. 18), "If generic drugs are not subject to premarket approval (by FDA), a physician or pharmacist will have no assurance that generic products would be as equally safe and effective as the brand name drugs..." Such a statement is obviously misleading and, since half the drug products are unapproved today, it is outrageously misleading. Yet it is acceptable "argument" in adversarial proceedings.

As I was preparing these remarks the postman delivered to my desk a copy of the 1982 Fall Catalogue from Omaha Vaccine Company, a distributor which is typical of mail order houses around the U.S. The catalogue fell open to pages 34-35 and the magnitude of the FDA farce is all too exemplified by the fact that these two pages offer for sale 37 products: one medical device, one pesticide, one grooming aid, three FDA approved animal drugs, and thirty-one, that's right thirty-one unapproved animal drugs. FDA's shrill and unqualified contention that the Supreme Court must rule in FDA's favor or else unapproved new drugs will enter the market is absurd...not false, just intensely absurd.

Let me close these remarks by guessing at the future. The Supreme Court is pretty naive about pharmaceutical science but I expect they will only partially swallow FDA's bait. I even suspect they will lecture FDA severely for its conduct, but the spectre of dangerous drugs is terribly intimidating. The Court will feel compelled to give FDA most of what it wants, that is, essential jurisdiction to register all drug products. But the Court very well may take away with one hand what it gives with the other. Just how it will limit FDA's wanton enforcement of the new drug provisions of the Act remain to be seen. I can't guess, but I suspect it shall be limited. The Court will probably rule in a manner so as not to wreck the pharmaceutical industry from either direction. Ultimately, Congress needs to look at this law again and write an entirely new one to modernize the language and intent. Let's hope that time isn't far away.
MONENSIN—RUMENSIN/COBAN, INDICATIONS AND ADVERSE EFFECTS

George T. Edds, D.V.M., Ph.D., R. Bortell, M.S.
University of Florida, Gainesville, Florida

The anticoccidial agent, monensin, was first introduced to the poultry industry in 1971 as Coban Premix. Monensin is produced by the actinomycete Streptomyces cinnamomensis. It is effective against both intestinal and cecal coccidia but has little or no antibacterial activity. Monensin was introduced as a feed additive for ruminants as Rumensin in 1975 to improve the efficiency of rumen fermentation by reducing energy losses associated with volatile fatty acid formation. It increases the amount of propionic acid produced. Cattle obtain their daily energy needs from a smaller quantity of ration. The net effect is to lower daily feed intakes about 10%, maintain similar weight gains and improve feed efficiency. Feeding of 200 mg. Rumensin daily as supplement daily to cattle on pasture increased average daily gains 0.2 lb. or 16.3%.

With these very positive indications for both poultry and cattle one recognizes that industry has added a very effective drug for use by the agricultural producer. In developing a new drug, the company must be assured as to efficacy as well as the safety for the therapeutic agents. Monensin has a broad range of safety for both poultry and cattle. When Coban premix formulations were incorporated at the approved usage level of 0.0121% no toxicity was observed. If the level was increased 5x, i.e., 0.0605% a reduction in feed intake occurred along with decreased weight gains in poultry.

PRECAUTIONS FOR COBAN:

"For replacement chickens intended for use as cage layers only. Do not allow horses or other equines access to formulations containing Coban. Ingestion of Coban by equines has been fatal."

Warning: “Discontinue feed containing Coban Premix 72 hours before slaughter. Do not feed to laying chickens. Do not feed to chickens over 16 weeks of age. When mixing and handling monensin, use protective clothing, impervious gloves, and dust mask. Operators should wash thoroughly with soap and water after handling."

Acute toxicity trials indicated the LD₅₀ dosage for cattle was 21.9 mg/kg body weight. This compared to an optimum therapeutic dosage of 200 mg/head/day. Signs of intoxication included anorexia and diarrhea, depression, ataxia and tachypnea. Hematology and clinical chemistry values showed no hematologic but some significant chemistry changes by the 7th day.

Product liability relates to manufacturers or suppliers exercising reasonable care in formulation, manufacture, inspection, testing warnings, labels, instructions and sales of the products. Also, violations of statutes or governmental regulations can be considered negligence and a
basis for liability. The contracts for sale implies a warranty that the product is safe when used according to label directions. Disclaimers or contraindications should be in writing and conspicuous. The duty to warn of danger continues after the product is sold. Antidotes should be identified, if known.

Rumensin is toxic in horses with an estimated LD$_{50}$ of 2-3 mg/kg bodyweight. Horses given Rumensin at 3, 4 and 20 mg/kg died within 24 to 48 hours. Clinical signs included anorexia, muscular incoordination with ataxia, stumbling, exaggerated stepping or hypermetria; sweating was periodically profuse. Affected horses would lie down and rise repeatedly until they could no longer rise. Necropsy lesions included toxic tubular nephritis, toxic hepatitis and hemorrhage into the spleen. Two horses succumbed to as little as 1 gram of rumensin. The cattle premix contains 6 g/lb, thus a horse could die from as little as 2-3 teaspoonsfuls of this premix. A diluted premix, i.e., 1 pound Rumensin "60" to 2 lbs ground feed would require 6-9 tsp. to be toxic. If the Rumensin is added at 200 grams per ton, then 10 pounds of cattle feed could cause death of a horse.

Therefore, do NOT allow horses to gain access to either the Coban 45, Rumensin 60 or any feed combinations.

REFERENCES

1. Ordidge RM; Schubert FK; Stoker JW Death of horses after accidental feeding of monensin (letter) Vet Rec 1979 Apr 21; 104(16):375
INADVERTENT RESIDUES IN FOOD ANIMALS
RESULTING FROM CONTAMINATION OR ADULTERATION OF FEEDS
William B. Buck, D.V.M.

Feed-related problems are predominant sources of noninfectious diseases and chemical contamination of livestock and poultry. These problems vary from nutritional imbalances in major and minor ingredients to contamination with excessive drugs, chemicals, and natural substances such as fungal and plant toxins. During recent years, producers have been plagued with violative antimicrobial residues (sulfonamides and antibiotics) and with residues associated with other feed additives such as arsenicals and growth promotants. Pesticides, exemplified by the organochlorine insecticides, aldrin and dieldrin, and more recently certain organophosphorus compounds present a double hazard to livestock and poultry producers causing 1) violative residues in meat, milk, or eggs, and/or 2) overt toxicoses resulting in illness and death of exposed animals.

When violative residues occur in food animals, there is not only a potential human health hazard but also a true economic emergency exists for the producer. Governmental regulatory agencies such as the USDA Food Safety and Inspection Service (FSIS), the Food and Drug Administration, and the various state regulatory agencies are charged with the responsibility of assuring that foods of animal origin are residue-free. Economic losses from a single feed contamination incident may extend to millions of dollars because of: 1) livestock and poultry death loss, 2) additional laboratory expenses incurred by the regulatory agencies and the producers involved, 3) loss of revenue because the producer cannot market the product, and 4) legal fees associated with assessing liability.

Livestock and poultry producers have major capital investments in their operations. Each producer literally operates a business in which raw materials (feedstuffs) are used in the manufacture of finished products (meat, milk and eggs). Most successful manufacturers expend considerable funds for quality control of their product—to protect the consumer and their own investments. Yet the majority of livestock and poultry producers have no means of quality assurance of feedstuffs used in their operation to prevent violative residues in their product—to protect the consumer and, thus, their capital investments.

When a producer suddenly becomes unable to market his product, he rapidly faces bankruptcy. Federal and state regulatory programs leave a gap at this point. Although there is a good surveillance of livestock and food products of animal origin, there is no mechanism for assisting the producer in the decontamination of his feed and livestock so that he can rapidly get back into the market. Furthermore, there are no adequate programs to assist livestock and poultry producers in detecting contaminated feeds in order to prevent their inclusion into livestock and poultry rations.
ANIMAL FEED SPECIMEN STORAGE (BANKING) AND SCREENING

During the past 2 years, we have been developing procedures for sampling and storage of livestock and poultry feeds. The objectives have been to develop procedures for representative sampling, labelling, preservation, and storage of feed specimens to enable prospective and retrospective identification of drug/toxicant contamination and adulteration. Accomplishments have included:

1) Development of techniques for sampling, identification, and preservation of specimens.

2) Development of procedures for storage of specimens on producer premises.

3) Development of microscopy procedures for identifying ingredients and adulterant contaminants.

4) Development of limited analytical screening procedures for contaminants.

The pilot feed specimen banking program has confirmed its feasibility and set the stage for additional developmental studies with varied food-animal management/feeding systems. The Illinois Feed Analysis and Residue Monitoring (FARM) Program is designed to work closely with selected producer-cooperators in the development of a feed quality assurance program that eventually will be operated and funded by subscriber producers. The program must be developed initially, however, with the aid of public funding to enable the establishment of a statewide Feed Analysis and Residue Monitoring Laboratory; to develop prototype techniques for feed sampling and storage; laboratory studies; and computer-assisted modeling of management systems for residue avoidance. Once cost-effective protocols have been developed they can be utilized nationwide through private or public funding.

TOXICOLOGY HOTLINE AND EMERGENCY RESPONSE INVESTIGATION SERVICE

Working through the Veterinary Diagnostic Laboratory we officially established an Animal Poison Control Emergency Service in 1979. Callers can obtain information and consultation about known or suspected cases of poisoning or chemical contamination. The Hotline is manned 24 hours a day, 7 days a week. Veterinary toxicologists are on call during regular office hours. During evenings, weekends, and holidays the calls are taken through a telephone answering service and returned by the veterinary toxicologist on duty.

The team members have available a comprehensive file on chemicals, feed additives, drugs, pesticides, environmental contaminants, and plant and mold toxins. The file is being continuously updated and contains comparative toxicity data and recommended therapeutic and decontamination measures. This data bank, eventually expected to contain
more than 100,000 entries, facilitates rapid response to all types of poisoning/contamination episodes in animals.

Concomitant with establishing the Animal Toxicology Hotline, we also initiated a rapid response investigation service consisting of faculty of the Veterinary Diagnostic Laboratory, the Colleges of Veterinary Medicine and Agriculture, and extension specialists ready to investigate suspected or potential poisoning or chemical contamination problems in livestock and poultry. When telephone consultation is inadequate or the problem is potentially of major proportion, a team of investigators is dispatched to assist in diagnosis, collecting appropriate specimens, and solving the problem. A total of 44 field investigations were conducted in 1981. The problems ranged from plant poisoning and mycotoxicoses to contamination of grain by carbon tetrachloride/carbon disulfide fumigants (See Sample Case Histories 1, 2 and 3). About 450 case calls are handled monthly by the Animal Toxicology Hotline involving all species of domestic animals and birds.

CONCLUSION:

The Illinois FARM Program is the first producer-oriented feed quality assurance program to be proposed in the United States. Its major thrusts (to develop procedures for producers to sample and store feeds and ingredients; develop a battery of microscopy techniques and screening tests for detecting adulteration and violative residues; and computer modeling for guidance to producers in residue avoidance and management practices) are directed toward the creation of a cost-effective program that can be utilized anywhere there is livestock and poultry production.

Not only will this program serve to stabilize the economic position of individual producers but will be a major factor in creating consumer confidence in foods of animal origin, thus, expanding markets both at home and abroad.

The Illinois prototype program will provide a basis for regional and nationwide FARM programs. In doing this we will have an effective mechanism for preventing catastrophic disruption of meat, milk and egg production in the event of major contamination of livestock and poultry feeds by natural or man-made contaminants. This has strong national defense implications. If a foreign power were to release contaminant(s) so that it resulted in generalized livestock feed contamination, the proposed program would already be in place to facilitate detection, containment, decontamination and protection of our nation's food supply.

CASE #1:
SWINE LOSSES
COTTONSEED MEAL (GOSSYPOL) TOXICOSIS

HISTORY

An Illinois swine producer had a 30-40 sow farrow to finish partial
confinement operation during the summer of 1982. His management involved farrowing and growing the pigs up to approximately 100 lb. in total confinement after which they were moved to an open dirt finishing lot. During the latter part of June and all of the month of July the grower and finishing animals sickened and approximately 150 of 300 animals died. In addition about 20% of the remaining animals manifested evidence of poor health. The death loss had occurred throughout the 4-6 week period. Clinical signs included respiratory distress with short, choppy breathing (thumping), coughing, distended abdomen and death. Some animals would die shortly after manifesting clinical signs. Others would live several days and some were found dead without having manifested clinical illness.

The attending veterinarian conducted post mortem examinations, sent tissues and other samples to a neighboring state veterinary diagnostic laboratory and requested assistance from the neighboring state diagnostic laboratory who conducted a field investigation of the problem. Gross post mortem findings included fluid accumulation in all body cavities, severe cardiomyopathy, and pulmonary edema. The following is an excerpt from the laboratory report:

Gross Findings—two 4 month old pigs were submitted for necropsy with a history of death which occurred within a several hour time period. Both pigs were castrated males and were in good body condition. The skeletal muscle of one pig had a varied color pattern within the muscle bundles. Red and pale areas were present. There was marked pulmonary edema and hydrothorax in both pigs. The ventral margin of the lungs was red but not firm. Hydropericardium was also present. Multifocal subendocardial pale areas were present in the heart. Both pigs had multifocal, approximately 2 cm in diameter, white foci in the liver. The liver had an accentuated lobular pattern. One pig had centrilobular congestion. Both pigs had mild ascites. The ventral nasal turbinates of one pig were slightly atrophic.

Histopathologic Findings—The primary morphologic alterations in the heart are coagulation necrosis, hemorrhage, congestion and degeneration of myocardial fibers. A few fibrin thrombi are present in heart and lining vessels. Severe pulmonary congestion and edema are present. A focal area of hemorrhage and inflammatory cell infiltrate is present in the superficial mucosa of the stomach. The liver has increased perilobular fibrosis and inflammatory cell infiltrate. Fibrosis, edema and inflammatory infiltrate are also present in the mesenteric lymph nodes.

There were no significant bacteriology or virology findings.

A diagnosis of selenium—vitamin E deficiency was made and prescription to add increased selenium premix to the diet was suggested on June 28. By July 09 losses were continuing and three more animals were submitted to the same laboratory. Similar findings were made as with the first two animals and a diagnosis of cardiovascular collapse.
initiated by an extreme vasculitis with an outpouring of fluid was made. During the ensuing two week period losses continued and on July 27 the Animal Poison Control Center was requested to make a field investigation by an Illinois veterinarian who had been called into the case. A team of veterinary toxicologists and pathologists conducted the investigation that day. A number of dead animals were observed in the finishing feedlots and about 20% of the live animals were exhibiting evidence of poor weight gains, respiratory distress upon exercise and general muscular weakness. One animal was killed in the field and a necropsy was performed. In addition three other animals were submitted to the Veterinary Diagnostic Laboratory, College of Veterinary Medicine, University of Illinois. A total of six feed samples were obtained from feeders located in the grower and finishing lots where animals were sick or had died. Clinical pathologic analyses were performed on blood from two live animals that were submitted to the laboratory. All animals were necropsied.

LABORATORY FINDINGS

The major gross lesions were severe cardiomyopathy with hydropericardium, hydrothorax, ascites and centrilobular hepatic congestion. All four of the animals necropsied were either in fair or poor nutritional condition. Histopathologic changes included diffuse myocardial fiber atrophy with perinuclear vacuolation. There was mild subendocardial fibrosis and focal areas of mild edema and fibrosis between muscle bundles especially near the epicardium. The pericardium was markedly thickened by edema and mononuclear inflammatory infiltrates as well as by young connective tissue, which proliferated into adjacent muscle fibers and into nerves and purkinje fiber bundles. The epicardial surface was covered by cellular debris and neutrophils as well as by fibrin, especially severe in the atria. Skeletal muscle fibers were also undergoing degeneration characterized by swelling, granular disintegration and mild hypercellularity. There were marked hepatic centrilobular congestion, loss of hepatocytes and early connective tissue formation. There was mild to moderate inflammatory infiltration in the portal triads, sinusoids and areas of necrosis. There was diffuse thickening of pulmonary alveolar walls due to mixed inflammatory cell infiltration. There was mild peribronchiolar inflammatory cell accumulation and mild hypertrophy of smooth muscle around terminal bronchials and alveolar ducts. Several large areas of pulmonary consolidation characterized by diffuse infiltration of alveolar lumina by neutrophils were present.

Chemical analyses were performed on the six feed samples as follows: No mycotoxins (ochratoxin, vomitoxin, zearalenone, T-2, DAS, and aflatoxin), nor monensin was detected. Selenium concentrations ranged from 428 to 1,513 ppb. The liver of the first pig necropsied had 543 ppb wet weight and whole blood contained 233 ppb. The liver from the same pig together with a control pig liver were analyzed for 18 trace metals. A
significant finding was 90 ppm iron as compared to 164 ppm in the control liver.

Various tissues and body fluids were studied by three veterinary diagnostic laboratories and infectious agents which could cause such swine losses were not identified by bacterial and viral isolation procedures.

Microscopic examination of the feed specimens revealed the presence of cottonseed meal in concentrations ranging from 3 to 10%. A sample of 40% protein supplement contained 19% cottonseed meal. Three of the feed specimens were analyzed for free gossypol content. A sample of complete feed taken from a lot where most of the larger hogs had died contained 400 ppm. Another sample taken from the confinement pen where 12 animals had died contained 200 ppm and the protein supplement contained 1300 ppm free gossypol.

COMMENT:

Although the feed contained greater than recommended concentrations of selenium, the liver and whole blood concentrations (543 and 233 ppb respectively) along with details of the gross and histopathologic findings were not consistent with a diagnosis of selenium toxicosis. The history, clinical signs, pathologic findings and feed analyses are compatible with a diagnosis of gossypol toxicosis.

CATTLE LOSSES—POISONING BY CARBON TETRACHLORIDE/CARBON DISULFIDE GRAIN FUMIGANT*

HISTORY

In September, 1981 an Ohio farmer operated a grain-livestock farm producing corn, soybeans, and fat cattle. He had approximately 1250 Angus-Charolais cross steers ranging from 800 to 1200 lbs. located in 10 or 11 separate pens.

On September 8 the farmer sold 94 head of steers for slaughter. When the cattle were being loaded out he found three dead in the pen that had been separated out for slaughter and another animal dead in another pen. He had noticed the night before that a few animals had loose stools and tended to be a little bit slow in walking. He initially thought the death had occurred because of bloat. Subsequently two additional animals were found dead in two different finishing pens. His local veterinarian was called for assistance. Approximately 20 animals located throughout all but two of the finishing pens were exhibiting clinical signs including excessive salivation, dull eyes, hanging heads, depression, diarrhea, grinding of the teeth, anorexia, and subnormal temperature. A post mortem examination was performed on two animals. The findings included bloody fluid in the rumens and intestines ranging from the duodenum to the rectum; streaks of hemorrhages in the abomasum (but
not the mucosal or serosal surfaces); and the livers were mottled and friable.

The attending veterinarian notified the packing company that had received the 94 head for slaughter of the possibility that the animals had been exposed to a toxicant in the feed. Therefore, 74 head were sent back to the farm. The remaining 20 had already been slaughtered and were held for the USDA Food Safety Inspection Service disposition. The 20 carcasses were released on September 24. The Animal Poison Control Center was called at 11 a.m. on September 14 and invited to investigate the cause of the problem. A team of veterinary toxicologists from the Diagnostic Laboratory flew in a University owned airplane to the Ohio farm that afternoon. At the time of our investigation most of the animals in the 8 or 9 pens totalling approximately 1,050 animals were still off feed or at least not on full feed. Several had a diarrhea. Many of the animals were grinding their teeth. Some had foamy slobbering and a peculiar licking of the viborissal hairs about the mouth. Whole blood and serum samples were taken from three different animals in different pens and a total of 25 feed and/or ingredient specimens were obtained either at the time of our investigation or furnished by the veterinarian/farmer that were taken on September 8, the day after the initial losses were observed. In addition, the attending veterinarian had submitted two steers to the state diagnostic laboratory who subsequently submitted tissue, blood and rumen/abomasal contents.

On September 16 a second investigation was conducted by the University of Illinois and included a beef cattle nutritionist and veterinary clinician as well as a toxicologist.

The fattening cattle were being fed somewhat different regimens depending upon their weight and length of time on feed. Two pens containing 200 head were recently purchased cattle and were being fed high moisture corn, haylage and corn silage. None of these animals were affected. The other 9 pens of cattle were being fed corn silage, alfalfa haylage, a 50% protein supplement, and dry shelled corn purchased from a local feed elevator. Animals in all 9 of these pens were affected.

LABORATORY FINDINGS:

On September 9 a 7th steer was found sick and was sent to the Ohio Department of Agriculture Veterinary Diagnostic Laboratory along with another steer that had died the day before. The dead carcass was decomposed and was not necropsied. The live animal was electrocuted and the following lesions were described: streaks and blotches of hemorrhage in the wall of the rumen and reticulum (but not the mucosal or serosal surface), a few small hemorrhagic erosions in the abomasal mucosa, congested intestines with dark fluid contents. The liver was enlarged and friable. The urine had a greenish tint. No lesions were found in the thoracic cavity. Histopathologic changes in the rumen, reticulum and abomasum were similar. Superficial mucosa was intact and was within normal limits. The submucosa was characterized by a dramatic
influx of fluid, segmented neutrophils, and focal areas of hemorrhage. All vessels were markedly distended; in one instance there was evidence of fibrinoid change. The liver had acute multifocal necrosis with massive hemorrhage but no suppurative response or vasculitis. The kidney had mild vacuolar degeneration and early protein leakage from the glomerular tufts. It was concluded that the hemorrhage and necrosis in the liver and intense edema and acute inflammation in the upper digestive tract were suggestive of a toxigenic substance in the upper GI tract.

Well over 250 analyses were performed on the feeds and feed ingredients, rumen contents and tissues at the Illinois Department of Agriculture Veterinary Diagnostic Laboratory in Centralia, Illinois and the Veterinary Diagnostic Laboratory at the University of Illinois. Results are summarized as follows: All high moisture corn and dry corn specimens were negative for aflatoxins, zearalene, zearalenol, T-2 toxin, diacetoxyisocyrenol and vomitoxin. Twenty-one feed specimens including 50% protein supplement, high moisture corn and dry corn, mixed feed composites taken from numerous feed bunks, corn silage and haylage taken from silos, and grain dusts taken from bin chutes and conveyor belts at the local elevator were assayed in a xenobiotic screen and found negative. The xenobiotic screen inlcuded 30 organochlorine, organophosphorus and carbamate insecticides and industrial contaminants such as PCB's, and PBB compounds. Other specimens assayed in the xenobiotic screen found negative included several samples of rumen and abomasal contents taken from several animals and liver and kidney tissues. Three samples of the 50% protein supplement were analyzed for urea content and found to contain 6.3 to 10.7% (not considered abnormal).

The liver tissues were analyzed for lead and arsenic by the Ohio Department of Agriculture Laboratory and found to be within background levels. The Illinois laboratories analyzed 3 samples of paints taken from loading chutes and cattle pens. These contained less than .5% lead, while some pealing paint taken from a barn not in access to the cattle contained 14% lead. Three rumen content samples were analyzed for ammonia nitrogen content (ranging from 18.7 to 79.6) and pH (ranging from 4.5 to 5.0).

All of the corn, mixed feed samples and rumen contents were analyzed by the Illinois Department of Agriculture Centralia Laboratory for carbon tetrachloride/carbon disulfide. These chemicals were found in the dry corn specimens taken from the truck bed and unloading pit on the farm the day after the first losses (September 8). Carbon tetrachloride and carbon disulfide were not detected in rumen or abomasum contents, feed, dust or any other feed specimens. Samples of the dry corn were submitted to the National Animal Disease Center, APHIS, Toxicology Laboratory where both carbon tetrachloride and carbon disulfide were confirmed in the dry corn specimens by GC/MS instrumentation.

The Ohio Department of Agriculture Laboratory conducted bac-
teriology and viralogy studies on tissues and failed to find sufficient pathogens to account for cattle losses.

COMMENTS

These cattle took several weeks to come back onto full feed and a high percentage of those who did resume full feed failed to gain weight and some even continued to lose weight for several weeks after the acute problem. After about six weeks the first load of animals were sold for slaughter and although lighter than usual, they had a good yield.

Based on the clinical signs and lesions together with the results of chemical analyses, a diagnosis of carbon tetrachloride/carbon disulfide fumigant poisoning was made. During the initial investigation the local feed elevator was examined because it was the source of both the dry corn and protein supplement, the only common feed ingredients among all affected animals. Located in the hallway of the grain elevator were an estimated 40 to 50 five gallon drums of grain fumigant containing carbon tetrachloride and carbon disulfide at an 80:20 ratio. The feed elevator manager reported that this fumigant had been used in the treatment of the corn prior to shipment for export. We theorized that the fumigant had concentrated in the bottom of the grain bin from which the load of corn was taken and fed to the affected cattle two to three days prior to the onset of their illness. It is likely the cattle remained off feed and failed to make adequate weight gains subsequent to the acute problem because of the severe gastrointestinal and hepatic damage. It seemed reasonable that these animals would be suitable for slaughter for human consumption once they resumed full feed and made reasonable weight gains.

CASE #3

MARKET QUARANTINE OF BEEF CATTLE AND SWINE BECAUSE OF ALDRIN/DIELDRIN CONTAMINATION OF FEED

HISTORY:

In February, 1980 one of Iowa's largest beef cattle and swine producers was notified that one hog that had been routinely tested by the USDA, Food Safety Inspection Service, had been found to contain dieldrin in its body fat at a concentration greater than the actionable level of 0.3 ppm (the actual concentration was 0.58 ppm). The producer was notified that the next shipment of hogs would be held from entering the market channels until specimens from 30 had been tested for dieldrin and all shown to be less than the actionable level (0.3 ppm). The following week the producer shipped 200 head of fat hogs to the slaughtering establishment and was subsequently notified that approximately 20 of the 30 that had been sampled had violative levels of dieldrin and would therefore have to be destroyed. Furthermore, the producer was notified that he could not market hogs until his entire herd was shown to be free of violative residues. The producer had approximately 20,000 finishing swine in confinement on one premise weighing from approximately 100 to
BUCK

220 lbs. His routine practice was to market approximately 2000 hogs each week. On another premise he had approximately 15,000 grower pigs in confinement which were to subsequently be transported to the finishing facility.

On the same premises and adjacent confinement facilities the producer had approximately 3,400 head of finishing cattle weighing from 1200 to 1300 lbs. each that were ready to be marketed within the next 30 days.

On February 18 the producer telephoned the Animal Poison Control Center and requested assistance in determining the extent of the contamination problem, source of the contamination and to advise methods for decontamination and reestablishing marketing channels. The first field investigation was conducted three days later by two veterinary toxicologists who obtained 66 surgical fat biopsies from swine and 8 scrotal fat biopsies from the fat steers. The fat biopsies were taken so that they represented the entire 35,000 swine and 3400 head of cattle. In addition, numerous environmental (dust), pit, waste lagoon, and water samples were taken for aldrin/dieldrin analysis.

A subsequent field investigation was made by the Animal Poison Control Center veterinary toxicologists on March 1 and 2 during which time several hundred representative feed specimens were taken from the feed bunks of the swine and cattle together with various samples in and around the feed mixing facilities and grain storage bins. During the first two investigations it was learned that almost all of the corn being fed both the cattle and swine had been purchased from a local grain terminal elevator. Therefore, the elevator and a sister elevator in a neighboring town were visited with the idea of sampling corn storage areas including outside dirt pads where corn had previously been stored before it was delivered to the producer's premises. Subsequent field investigations were conducted on March 10 and again in May during which time numerous feed specimens were obtained from the feed elevators as well as from the producer's premises.

All of the tissues, feed specimens and environmental and water samples were submitted to the Illinois Department of Agriculture, Veterinary Diagnostic Laboratory at Centralia, Illinois where they were analyzed for chlorinated hydrocarbon insecticides, specifically aldrin/dieldrin.

LABORATORY FINDINGS

Analyses of the fat biopsies of both the swine and cattle indicated that all the swine and cattle on both premises had been exposed to aldrin or dieldrin probably within the last two-month period. Approximately 50% of the animals had residues below the actionable concentration (0.3 ppm) and approximately 50% had violative residues of dieldrin as high as 0.8 ppm. This was especially true of approximately 10,000 fat hogs that were about ready for market and the 3400 head of cattle. (Subsequent testing of both cattle and swine by obtaining surgical fat biopsies and submitted
Analysis of hundreds of feed specimens revealed that the corn purchased from the terminal elevator was contaminated with aldrin. There was no evidence that any of the other feed ingredients had been contaminated. Analyses of soil samples taken from where the corn had been piled on the ground revealed aldrin and dieldrin levels as high as 40 ppm in the top 2" of soil. This area had been a pesticide staging area for helicopter applications of agricultural chemicals. It became evident, however, during the investigation that not only had corn been contaminated from having been on contaminated soil and the soil mixed in with the shelled corn when it was scooped up by the large equipment; but also that corn had been contaminated in the terminal elevator itself by some other procedure which was never fully determined. It was postulated that corn in the terminal elevator had been accidentally treated with an aldrin or dieldrin preparation by mistake but this theory was never completely substantiated. Most of the corn specimens obtained on the producer's premises contained aldrin in concentrations up to about 300 ppb, however, screenings from corn that was being shipped for export from the terminal elevator contained up to 5 ppm aldrin. (An interesting sidelight to this problem was that two truckloads of screenings contaminated with aldrin were subsequently shipped to a feedlot in Oklahoma where they were fed to approximately 3500 cattle. Subsequent investigations of this contamination problem revealed that the feeder cattle had not received sufficient contamination to develop violative residues, although substantial disruption of the marketing of feeder animals occurred which resulted in considerable economic damage to the Oklahoma producer.)

DECONTAMINATION AND REESTABLISHMENT OF MARKETING CHANNELS

The Animal Poison Control Center veterinary toxicologists worked in consultation with the USDA, FSIS regulatory veterinarians, The Iowa State University Veterinary Diagnostic Laboratory toxicologists and the producer in deciding the most cost effective course to be taken in decontamination and reestablishing the marketing of the swine and cattle. It was decided that the cattle would be turned into a grass pasture for a six month period without a grain diet to enable them to lose fat and, therefore, the dieldrin contamination, then subsequently refattened for marketing. The cattle were moved to a ranch in Wisconsin where they were placed on a grass pasture. During the ensuing 4 to 5 months the animals lost an average of 200 to 300 lbs. By October, 1980 the animals were found to have excreted their dieldrin content to well below the actionable levels of 0.3 ppm. They were placed in a feedlot and refattened and sold for slaughter. The cost of transportation and decontamination of these animals was in the neighborhood of 1½ million dollars, however, their salvage value amounted to approximately 5 million dollars.
Since the larger swine weighing from 200 to 220 lbs. and ready for market had the highest concentration of dieldrin contamination, it was decided that approximately 6500 animals would be killed and rendered for meat scraps with the fat being discarded. This gave the producer an opportunity to decontaminate his feeders and pens using a high pressure water hose and detergent. He then was able to put the smaller swine, although previously contaminated, into clean pens and on clean feed and by the time they reached market age most were decontaminated to the point that they were approved for slaughter by the time they reached 200 lbs. body weight. The decontamination of the two premises holding some 35,000 head of swine was a major operation. Also, there was considerable damage to the finishing confinement facility because for a 2-3 month period no animals were marketed or slaughtered. Since all animals were becoming larger and larger, they outgrew the holding capacity of the facility and thus physically damaged the facility to the point that it was almost beyond reconstruction.

From the time that the first violative residue was encountered by FSIS in the swine until the farmer had settled with the feed cooperative for the damage that he had incurred, literally hundreds of regulatory and scientific people were involved in this case. Thousands of samples were analyzed by the food and drug administration, private laboratories and the State of Illinois. The producers saw his previously viable operation become a vacant, disheveled, worthless piece of property. Small town banks and large city banks were maneuvering to minimize their losses. Attorneys and consultant experts were expending thousands of hours trying to establish liability in this case. The case was not tried in court, however, because the producer and his creditors ultimately accepted an approximately 6 million dollar settlement for the losses and damages incurred.


2) Investigators included W. B. Buck, L. L. Berger, R. W. Coppock and W. A. Wolff.

3) Investigators included V. R. Beasley, D. J. Blodgett, W. B. Buck, R. V. Chalam and J. C. Haliburton.
REPORT OF COMMITTEE ON PHARMACEUTICALS,
PESTICIDES AND RELATED TOXICOLOGY
UNITED STATES ANIMAL HEALTH ASSOCIATION

Chairman: W.*A. Knapp, Jr., Raleigh, NC
Vice Chairman: G. D. Lindsey, Indianapolis, IN

D. A. Armstrong, MT; D. T. Bechtol, TX; W. B. Bixler, VA; Jerry Brunton, VA; W. B. Buck, IL; H. F. Burton, MD; F. Carter, MO; L. M. Crawford, GA; G. T. Edds, FL; J. B. Eller, D.C.; D. O. Farrington, IN; J. E. Fox, NY; D. A. Gable, VA; R. W. Gessert, VA; R. L. Gillespie, MD; J. S. Gloyd, IL; L. C. Harold, VA; J. S. Hayden, MO; W. Jochle, NJ; D. R. Mackey, CO; G. Murray, D.C.; G. D. Osweiler, MO; M. G. Scroggs, OH; T. K. Shotwell, TX; J. Silver, CT; Roy Strange, GA.

Dr. Ted Humphries, Veterinary Attache, Embassy of Australia, Washington, D.C. replaced Dr. Gardner Murray who returned to Australia after a three year assignment in the United States.

Dr. Wayne Weber, USDA Residue Planning Division, spoke on USDA's Residue Avoidance Program (RAP) which is designed to help America's livestock and poultry farmers avoid (prevent) tissue residues of drugs, chemicals, and other potentially hazardous substances through improved management procedures. Dr. C. D. Van Houweling responded to Dr. Weber's presentation by offering some concerns of industry about the RAP program.

The committee's response to both presentations was positive. A cooperative effort among producers and RAP appears indicated and is hereby recommended.

Dr. Thomas K. Shotwell presented a paper entitled "Generic Competition in Animal Health Products—The Generix Decision." This case is presently before the U.S. Supreme Court. If Generix prevails, the door will be open for any company to market a "me-too" version of any approved drug without FDA approval. If FDA prevails, then every drug product, regardless of age or general recognition by experts, will be the subject of an NADA. A decision is expected in early 1983. Dr. Shotwell's paper appears elsewhere in these USAHA Proceedings.

Dr. Gerald B. Guest, Deputy Director, Bureau of Veterinary Medicine, Food and Drug Administration, Washington, D.C., presented a status report on several regulatory proposals.

Dr. Guest reported that:

(1) A new animal drug approval process and a new appeals procedure were being drafted.

(2) The first animal drug to be approved in the Fast Track System took place in April, 1982.

(3) The Bureau of Veterinary Medicine (BVM) is seriously evaluating the so-called "drug lag" problem in the U.S.A.
(4) New Sensitivity of Method (SOM) document is expected to issue in early 1983. It will be in the form of guidelines instead of regulations as previously proposed.

(5) Minor-use drug document, (Low-Volume, Limited-Use Drugs for Use in Food Animals) is scheduled for early publication.

(6) Five tissue residue analytical methods validation trials are in process.

It was readily apparent from Dr. Guest’s report that the Bureau of Veterinary Medicine is planning for progress in 1983. It’s recent major reorganization suggests that a sound organizational base will not only support but also promote changes of considerable magnitude and significance.

William S. Buck, College of Veterinary Medicine, University of Illinois, Urbana, Illinois, presented an overview of the Feed Analysis and Residue Monitoring (FARM) Program that is being developed in Illinois. He also discussed the Illinois Animal Toxicology Hotline. A paper entitled “Inadvertent Residues in Food Animals Resulting from Contamination or Adulteration of Feeds” by William B. Buck appears elsewhere in these proceedings. Basically, the FARM Program in Illinois is designed to provide the livestock farmer and the small to middle size feed company with animal feed quality assurance capabilities. Dr. Buck stated that most livestock tissue residue problems are feed related and the Illinois FARM Program is designed to reduce this major source of contamination.

A paper by Dr. George T. Edds et al entitled “Monensin-Rumensin/Coban; Indications and Adverse Effects” was read in Dr. Edds absence by Dr. William A. Knapp, Jr. Monensin is toxic in horses. It has an estimated LD 50 in the horse of 2-3 mg/Kg body weight. Two horses in this study succumbed to as little as one gram of Monensin. This paper appears elsewhere in these proceedings. Therefore, it is important that the use directions and cautionary statements on the premix labels be carefully observed.

No motions and/or resolutions were made during this session of the Committee on Pharmaceuticals, Pesticides and Related Toxicology. The committee decided to have a mid-year meeting for the purpose of improving it’s programs and helping it to better achieve its goals and objectives.
INTRODUCTION

The term embryo transfer is used to refer to the recovery of early embryos from the reproductive tract of a donor and the subsequent transport to, and placement in, the reproductive tract of a recipient. The embryos are collected before they become implanted and, depending on the species, are usually 2 to 9 days of age. Embryo transfer has proven to be an effective procedure for expanding the pool of superior genetic material within a species and is of use clinically for obtaining offspring from senescent or infertile animals (1, 2). Due to the simplicity of the procedure and to its high frequency of success, embryo transfer is also considered to be a practical alternative to conventional methods for interstate and international transport of postnatal animals (3, 4). This is of concern to those with the responsibility for regulation of livestock movement (4). The reason for concern is that it is difficult to assess the potential of embryo transfer for infectious disease dissemination. There is little information in the available literature on the frequency or nature of the interactions that may occur between infectious disease producing agents and preimplantation embryos.

Several viruses have been shown to be detrimental to the development of early mouse embryos in vitro (5, 6). Degeneration of the embryos with the occasional recovery of virus is the evidence that has been offered for viral interaction with early embryos. However, detailed descriptions of the types of interactions that may occur between viruses and embryos have not been reported. It is possible that viruses may interact with an embryo without producing harmful effects. Previously, it was shown that pseudorabies virus (PrV) did not cause the degeneration of porcine embryos when they were exposed to the virus in vitro (7). To test whether PrV had interacted with the embryos in a benign manner, embryos that had been exposed to the virus were transferred to susceptible recipients (8). The results from those transfers indicated that PrV was transmitted by the embryos to their recipients. This stimulated a series of investigations in which early porcine embryos were exposed to PrV, porcine parvovirus (PPV), or either of two porcine enteroviruses, ECPO-3 or ECPO-6. The embryos were examined by electron microscopic techniques to discover the types of interactions that may occur between embryos and viruses.

MATERIALS AND METHODS

Embryos were surgically collected from donor sows or gilts. All of the embryo donors were free of detectable, PrV neutralizing, serum antibodies at the time of embryo collection. A standard virus neutralization
test was used at the 1:4 dilution of the sera (9). The embryos were all 2 to 3 days of age and of the 4- to 8-cell stages. A few of the donor animals were exposed to PrV after insemination and approximately 56 to 80 hours before embryo collection. Exposure of those donors was by intranasal, or intranasal and intrauterine injections of virus-containing materials.

The embryos that were collected from the exposed donors were washed by serial passage through 5 aliquots of cell culture medium supplemented 2% with new born calf serum. Following the washing procedure, the embryos were transferred to recipient animals that were free of detectable, PrV neutralizing, serum antibodies. Some of the embryos that were collected from the donors that had not been exposed to virus were placed on cell monolayers for cocultivation. The monolayers had been previously inoculated with PrV, PPV, or one of the enteroviruses. After 2, 24 or 48 hours of cultivation at 37°C in a humidified atmosphere of 5% CO₂ in air, the embryos were fixed in a 2.5% glutaraldehyde solution and processed for electron microscopic examination. The remaining embryos from the nonexposed donors were cultivated for 1 hour in medium that contained 10⁴ or 10⁸ cell culture infective doses (CCID) of PrV. These embryos were then washed and transferred to recipients.

RESULTS

The recipients of embryos that had been exposed for 1 hour to 10⁴ CCID of PrV did not produce detectable concentrations of PrV neutralizing, serum antibodies. However, all of the recipients of embryos that had been exposed to 10⁸ CCID of PrV had PrV neutralizing, serum antibodies at 21 and 35 days after the transfers were performed. In addition, 2 of the recipients of embryos that had been collected from donors exposed to PrV by intrauterine injection had PrV neutralizing, serum antibodies at 21 and 35 days after the transfers were performed.

Electron microscopic examination of the embryos that were cultivated on cell monolayers inoculated with PrV showed that the virus adsorbed to the outer surface of the zona pellucida. The number of virions observed per section of embryo increased with the time of cultivation. After 24 or 48 hours, the virus aggregates on the surface of the zona pellucida were of sufficient magnitude to be observed under a 10× objective of a light microscope. PrV was also found to enter sperm tracks within the zona pellucida. Washing the embryos that were exposed to virus for 2 hours did not remove all of the virus from either the surface of the zona pellucida or from the sperm tracks.

Electron microscopic examination of the embryos that were exposed to PPV or to the enteroviruses showed that those viruses also associate with the embryo. PPV was frequently observed enmeshed in cellular debris that was on the outer surface of the zona pellucida. The enteroviruses occasionally were seen associated with embryos in a similar fashion. Usually, though, the enteroviruses were located beneath the outer surface of the zona pellucida. It appeared that the enteroviruses had entered the pore-like structures that are present on the surface of
the zona pellucida. Both PPV and the enteroviruses were seen in proximity to sperm that were on or immediately beneath the outer surface of the zona pellucida.

DISCUSSION AND CONCLUSIONS

The results of these investigations suggest that PrV can be transmitted through the use of embryo transfer. Under experimental conditions, embryos that were exposed to PrV in vitro or in vivo caused the appearance of neutralizing antibodies to PrV in the sera of susceptible embryo recipients. Electron microscopic examination of the embryos exposed to PrV, PPV, or porcine enterovirus revealed that viruses can become associated with embryos through adsorption to the zona pellucida, entry into sperm tracks or pores within the zona pellucida, or by being conjoined to cell debris or sperm. It is possible that several of the viruses that infect swine may be transmitted to an embryo recipient through their association with an embryo by one of these mechanisms.

Clearly, a potential for disease dissemination exists when embryo transfer is employed. However, the opportunities the embryo has for contact with virus are certainly fewer than those of a postnatal animal. Therefore, the potential for disease dissemination through embryo transfer is probably less than that which is associated with the transport of postnatal animals. It is recommended that prior to embryo collection the donors and their herds of origin should be carefully examined for infectious virus. The embryo recipients should be isolated from their herds or origin, and they should be tested at repeated intervals during gestation for evidence of exposure to viruses that are of concern. By following these guidelines, embryo transfer would be as safe, if not safer, than other means of accession of new livestock.

Supported in part by ARS, USDA Cooperative Agreement No. 58-519B-2-1161.

REFERENCES

Bolin, et al.


Dr. Hinchman contacted me a few weeks ago to request that I present this report to the committee on behalf of Dr. Mack. The report further illustrates the diversity of our national health programs. It covers an event taking place in Northwest Texas, over 50 of the nation's major purebred swine breeders in one-third of the states in the continental U.S. involved, report prepared by a USDA veterinary epidemiologist from Texas, and being presented by a state-employed veterinarian from Illinois. This truly qualifies as a United States Animal Health Association report.

The narrative and slides, as I related, was prepared by Dr. Robert Mack from the Veterinary Services Office in Austin, Texas. When Dr. Mack was not available to present the report his materials were forwarded to Dr. Hinchman, who prevailed on me to present it. The information was not completely new to me, I must confess. I had worked with Bob Mack in the final stages of the hog cholera eradication program in Illinois, had maintained sporadic contact with him since he left Illinois, and during the initial stages of the Lubbock outbreak had about twice weekly contact with either he or Dr. Robert Daniel of the Texas Animal Health Commission. Further, there is general, but not 100%, agreement that the source of the outbreak was a herd in northwest Illinois.

Slide 1 — Title

The Southwest Type Conference is the major Spotted swine event of the year. This show and sale bring together the major Spot breeders of the country to display their stock and exchange valuable breeding animals. It is a source of genetic material for breeders and commercial producers.

Slide 2 — Swine Exhibited at the Conference

48 breeders from 15 states — 167 head of breeding swine.

Junior Barrow Show: open to junior exhibitors enrolled in FFA or 4-H.

19 barrows exhibited from 16 sources in 4 states (Texas, New Mexico, Oklahoma, South Dakota).

All barrows were released on Thursday, February 18, following the bred sow show.

Health Requirements, as specified by the breed association, applies to pseudorabies only:

"Each breeding animal and barrow must have:

a negative test for pseudorabies within 30 days of the show regardless of whether or not the animal is from a qualified free herd."
Texas Animal Health Commission reports that there were no infractions of Texas regulations regarding exhibitors at the conference. Health certificates have been checked and all were in order.

**Slide 3 — U.S. Map**

Shows the states represented at the conference and the number of consignors from each state.

**Slide 4 — Summary**

48 consignors, 58 buyers of 116 head

61 no sales—(these are animals which were exhibited at the Lubbock conference by the 48 consignors but were not listed as "sales" by the conference records)—10 no sales were later sold by private treaty at Lubbock.

26 consignors took their no sale animals home from the conference. No sale animals tested positive in 7 herds.

**Slide 5 — U.S. Map Showing Movement of Swine from Lubbock**

16 states received exposed swine from the Spot Conference.

**Slides 6 & 7 — Summary by States**

Tabulation of the outbreak in the 17 states involved.

The columns are:

1. Consignors to the conference
2. Buyers of animals at the conference
3. Breakdown of the positive cases resulting from exposure at the conference:
   - Direct — herds receiving animals which were at Lubbock, later found to be infected.
   - Secondary cases — animals or herds infected by exposure to Lubbock animals (Texas' 12 secondary cases were largely the result of sharing the use of an infected boar bought at the conference.)
   - Herd infection — these are herds receiving infected animals directly from the conference in which pseudorabies spread to other animals in the herd; e.g., in one Texas herd which first used a new Spot boar, 72 of 90 animals tested positive.
4. Consignors with No Sales

These are consignors to the conference who took their "no sale" animals back home. Seven of these subsequently tested positive.

**Slide 8 — Pen Layout in Fairgrounds Building**

Building — steel quonset, approx. 300' × 75'

Orientation — east and west
Ventilation—adequate, high ceiling, exhaust fans in ends of building. Weather was mild, 65° daytime.

Entrance—double drive-in doors on east and west ends (all hogs entered thru these).

Pens—248 pens (34 not used, all on north end), some pens on perimeter (not shown on diagram).
- steel posts and hog wire, 3 ft. high.
- 12 groups of 16 pens each—each group of pens has a wash rack pen designated as “W”—all were used. Two of the 12 groups have the smaller pen arrangement drawn in; the sequence is the same in the other groups.

Floor—Concrete and clean.

There were no pen assignments. Exhibitors selected pens of their own and usually didn’t change pens during the conference. There is no penning record except memory of the consignors.

The barrows were penned throughout the barn and were freely commingled with the breeders.

Number of hogs per pen varied. They were penned by compatibility, some alone, some grouped.

Bedding was one bale of straw per pen and it lasted for three days. More bales were available and stacked in the barn if the pens needed it. The straw was purchased from a local farmer.

Pens were not cleaned during the conference. Barn was cleaned after the conference by the fairgrounds crew.

Ring in center of barn was used for sale, show, judging and picture taking (tanbark floor).

Slide 9—Factors Contributing to Spread

1. Commingling of Swine

The animals were constantly commingled by the activity at the conference:

Moving to the wash racks
Moving to the ring
Moving to picture taking
Moving to scale area

Animals moving in alleys had nose-to-nose contact with animals in the wire pens.

2. Common Facilities Usage:

Wash racks: 1 wash rack per 16 pens. All animals were washed in the wash rack at least once. The watering hose for the pens came from the wash rack.

Feed pan for pictures: An individual picture was taken of all
animals in a class. Pictures were taken by a professional breed photographer. The animal's picture is taken while eating from an elevated (8 inch) feed pan. This is the pose breeders like to show off type and gives a uniform pose for all swine. The feed pan was placed at the north end of the ring on Friday and the animals driven to it. On Saturday, the feed pan was placed in the old wash rack area located south of the ring. NOTE: Not all animals that had pictures taken were positive for pseudorabies and not all positive animals had pictures taken.

Scales: All breeding swine were weighed in on arrival and paint-branded on the rump. A common scale located near the ring was used for this.

Loading Chute: A local Spot breeder brought his portable chute. This was a low chute usable with pickups and low trailers. A large truck chute is located on the east end of the barn but it was not used.

Vehicles: Some producers used a common truck or trailer to haul animals to and from the conference.

Show Ring: All the animals at the conference went through the ring at least once, usually commingled with animals of other origins.

Wire pens: The pen wire was large mesh and, in some places, torn. This allowed nose-to-nose contact between pens and from pens to alleys.

Alleys: Animal movement in the alleys was a continual event due to the nature of the activities at the conference; e.g., movement to ring, wash racks, chutes, vehicles.

Housing: 186 head of hogs in one common enclosed area allows for aerosol transmission of the virus.

3. Stress: Research has shown that stress can cause carrier animals to shed the virus. Uprooting animals from a herd and transporting them long distances in cold winter weather is stressful. Strange environments and commingling with strange hogs is stressful. Purebred breeders talk about the use of corticosteroid compounds by breeders at a type conference to give their hogs that added alertness and "pzazz" needed to win honors. Corticosteroids are stress producing.

4. Inadequate Isolation After the Conference:

Many buyers and exhibitors at Lubbock got into trouble because they skipped the recommended steps of isolation and retesting of all new or returned swine.

A 30-day isolation and test procedure is vital to prevent introduction of pseudorabies.
Swine producers know of the need for isolation and retest but they get anxious and take short cuts,—especially in this case with pseudorabies, since all animals were from qualified herds and tested negative to enter the conference.

What can be learned from this outbreak:

1. It can happen again and to any breed conference. The Spot Association took all the precautions to prevent it. Follow-up isolation and retest after leaving the conference can, hopefully, prevent a repeat of an outbreak of this type. It can't prevent a positive appearing but it can prevent spread of infection to the home herd.

2. Some states are unable to handle an outbreak of pseudorabies because of lack of regulations, laws, or manpower.
   
   Some states show a definite lack of interest in working an outbreak.
   
   Some states are unable to get their field veterinarian to do the necessary field work in a timely manner.

Summary—It is generally agreed that an Illinois purebred herd was the source of this pseudorabies outbreak.

The following are additional comments from Dr. Spencer relative to the Illinois suspected source herd:

One of the top Spotted herds in the U.S. The father is an “old-time,” highly respected breeder. He has a son who takes an active part in the operation. Confinement farrowing; gilts, pregnant sows outside.

HISTORY

1. PR qualified herd since December 31, 1979
   —approximately 80 breeding sows.

2. Last quarterly test was 1/26/82—negative.

3. Animals to conference also tested 1/26/81—negative.

4. Took 3 boars, 4 gilts to Lubbock
   1 boar, 1 gilt were “no saled” and brought home.


6. Animals returned to Illinois: Gilt readded to the herd. Later tested positive. Boar sold to an Illinois breeder about 3/15. Blood tested 4/10, 1:4 titer. Breeder was establishing a new Spotted herd on a farm two miles away from his purebred Chester White herd. Boar died of massive hemorrhage from a gastric ulcer on 4/16. Gilts in new herd, plus one boar from another source, tested 4/10 also—all negative. Owner sent all to slaughter. No problems in PR Qualified Chester herd to date.
7. Tests on Illinois Herd:

- 3/29: 11 bled, 11 positive
- 4/13: 74 bled, 67 positive
- 4/23: 114 bled, 103 positive
- 5/19: 5 bled, 3 positive
- 6/25: 30 bled, 9 positive

(Many of the titers were in the 1:128 and 1:256 range.)

8. Owners are now establishing a new herd through a modified offspring segregation plan, with the new herd going to a farm over 20 miles away.

9. Production sale held by breeder on 3/13/81. 5 Illinois, 1 Indiana, 1 Iowa herds believed to have become infected by purchases from this sale.

10. Comments:

a. Epidemiology would indicate that this was the most likely source for the outbreak.

b. Four known infected herds were disclosed within a five-mile radius of the Illinois herd in January-February, 1982—one farm is 1/2 mile away. Complaints were made on hogs being moved between farms by members of a family using road in front of the purebred breeder's farm. Both neighboring herds later found to be infected.

c. An extremely explosive virus was involved in this outbreak—Note the high percentage and high dilution of titers shown. Spread was phenomenal—but then, it had ideal conditions for spread at the Lubbock sale.

What have we learned now that the publicity has died down? This is going to happen occasionally whenever you have concentration of hogs from multiple sources and under stress conditions. —It did happen once and it will probably happen again.
SWINE EXHIBITED BY:
   48 BREEDERS FROM 15 STATES

SWINE EXHIBITED:
   167 HEAD OF BREEDING STOCK
   19 BARROWS (JUNIOR BARROW SHOW)
UPDATE ON DNA FINGERPRINTING OF PSEUDORABIES VIRUS DNA

William C. Lawrence¹ University of Pennsylvania,
School of Veterinary Medicine, Philadelphia, PA 19104

For a number of reasons a laboratory procedure which is capable of un-
ambiguously identifying different strains of pseudorabies virus (PrV)
would provide a useful epidemiologic tool. For example: 1) The ability to
detect the presence of one or more than one strain within a herd would in-
dicate that one or more than one source of infections was involved; 2) when
appropriate isolates were available it would be possible to defini-
tively trace sources of infection; 3) it would be possible to distinguish
field strains from live attenuated vaccine strains.

Several procedures have been employed in efforts to distinguish
strains of PrV. These have included plaque morphology, neutralization ki-
netics, heat stability, sensitivity to proteolytic enzymes and pathoge-
nicity for laboratory animals (1, 2). Some of these procedures are difficult
to carry out on a large number of viral strains, and it is not clear that any
of them can unambiguously identify field strains of pseudorabies virus.

It has been known for several years that restriction endonuclease anal-
ysis of viral DNA can reveal strain differences, and it has been shown
that for herpes simplex virus, restriction enzymes can provide definitive
identification of individual strains of virus (3), and more recently strain
variations in PrV DNA have been described (4, 5).

Several restriction endonucleases have been found suitable for typing
herpesviruses because they cleave DNA at specific sites and, thus, into a
relatively small number of fragments which can readily be separated and
identified by electrophoresis. To compare one virus strain with another,
viruses are propagated in the presence of ³²P or ³H-thymidine, purified,
and DNA extracted. Both viral DNA samples are treated with a given re-
striction enzyme and analysed by gel electrophoresis and autoradiog-
raphy of the dried gels. Differential characteristics which allow typing
are the loss or gain of restriction enzyme cleavage sites which result in a
gain or loss in the molecular weight of a DNA fragment and a corre-
sponding change in mobility of the fragment. If the viruses being com-
pared are the same strain, the banding patterns of DNA fragments (fin-
gerprints) are identical; if not the differences are readily apparent. It is
not necessary to know what the bands mean in terms of the physical map
of restriction enzyme fragments, but merely whether they do or do not
correspond from one fragment to another.

DNA FINGERPRINTS AS STRAIN SPECIFIC MARKERS FOR PRV

The results of restriction enzyme analysis of 8 epidemiologically unre-
lated strains of PrV are shown in Fig. 1. These 8 strains were indis-
tinguishable by serologic techniques such as neutralization and fluo-
rescent antibody tests. By comparing the fragment patterns in adjacent slots
of the gel it can be seen that the fingerprints for each strain are different
and serve as markers which can distinguish each. Where differences are not apparent or are ambiguous, analysis with one or more different restriction enzymes creates a composite of fingerprints which in most cases can resolve uncertainties. When two or more strains are found to have essentially identical DNA fingerprints with a panel of different restriction enzymes, this supports, but does not prove, the hypothesis that they are actually representatives of a single virus strain. Several observations (6, and unpublished data) indicate that these DNA fingerprints are stable strain specific markers, and are therefore useful for a variety of epidemiologic studies.

EXAMPLES OF THE USE OF DNA FINGERPRINTING FOR EPIDEMIOLOGIC STUDIES OF PRV

Pennsylvania Outbreak: In Pennsylvania, 65 PrV infected herds have been identified by serologic testing. Sixty three of these herds are located within a single 50 square mile area and many of the infected herds are neighbors. To date, DNA fingerprints of 3 isolates from 3 separate herds within this area have been compared and, as shown in Fig. 1 (labeled Pa1, Pa2 and Pa3) the DNA fragment patterns indicate that these represent 3 different strains of PrV. These data indicate that there are at least 3 different strains in the endemic area and that there were 3 separate sources of infection of the 3 herds from which the isolates were obtained.

Indiana Outbreak: This study involved 3 Indiana herds located within a five mile radius. Over a four month period mortality in these herds reached about 15% of a total of 3000 animals. The results of restriction enzyme analysis of isolates from each of these herds are shown in Fig. 2. Here the three isolates were analysed with four restriction enzymes. Based on the results obtained with this panel of restriction enzymes the simplest interpretation is that in two of the infected herds, there was a common source of infection and that the third isolate represented a completely different strain of PrV. Thus, the outbreak in this herd was clearly unrelated to the other two, and this rules out a common source of infection in this case.

Nebraska Outbreak: In this study, a Nebraska herd, thought to be PrV free, experienced stillbirths and neonatal deaths shortly after initiation of the use of live attenuated vaccine within the herd. An isolate from a neonatal pig was compared with the vaccine strain which was used. The results are shown in Fig. 3. Here, side by side comparisons clearly show that the two strains are different, and the results do not support the hypothesis that death was caused by the vaccine strain. Instead, the results indicate that PrV infection by a non vaccine strain exists in this herd.

SUMMARY

Restriction enzyme analysis (fingerprinting) of PrV DNA provides stable strain specific markers which permit a variety of epidemiologic studies not possible using classical virologic techniques, to be carried out.
Three field isolates from different herds were analysed with restriction enzymes DdeI, XhoI, SalI, and SphI. $\Phi$ indicates significant differences between fragments in adjacent slots.
**Fig. 3**

Restriction Enzyme Analysis of Vaccine and Field Strain of Pseudorabies Virus.
Vac, vaccine strain; Ne I, Nebraska field strain.
Fig. 1

Restriction Enzyme DdeI Fingerprints of PrV DNA

\( ^3H \)-thymidine labeled DNA from 8 strains of PrV was digested with DdeI. Fragments were separated by agarose gel electrophoresis, and visualized by fluorography. Auj, Aujeszky strain; the other strains are field isolates from Indiana, California, Nebraska, and Pennsylvania.
REFERENCES


REPORT OF THE COMMITTEE ON PSEUDORABIES

Chairman — Lowell W. Hinchman, Indianapolis, Indiana
Vice Chairman — P.E. Bradshaw, Griggsville, Illinois


The Committee on Pseudorabies met at 1:30 PM on November 8, 1982 in Nashville, Tennessee with 54 in attendance including 13 committee members.

Dr. Donald Gustafson presented "Observations on Pseudorabies Virus, Porcine Parvo and Porcine Enterovirus in Porcine Embryo Transfer" indicating the lack of current information in available literature on the frequency and nature of interactions between infectious disease producing agents and preimplantation embryos.

He indicated the pseudorabies virus (PRV) does not cause degeneration of porcine embryos when exposed to the virus in vitro. Recipients of certain exposed embryos did not produce detectable antibodies, however, the recipients of embryos collected from donors exposed to intrauterine PRV did elicit serum antibodies in 21 and 35 days after the transfers were made. PRV is absorbed by the outer surface of the zona pellucida and enters sperm tracts within the zona pellucida and washing did not remove the virus from these areas. Investigation does indicate that PRV can be transmitted through embryo transfer but transmission opportunities are less than in post natal animals.

Dr. William Lawrence presented "Fingerprinting of the Pseudorabies Virus" and indicated that some 200 restricting enzymes have been found that restrict the nucleic molecular cleavage, but that any definitive fingerprinting procedures require the use of at least 2 to 4 selected enzymes to regiment true strain identification. The practical application for fingerprinting allows for distinguishing between vaccine and field strains of PRV as well as potentiating the determination of the epidemiological source of PRV in area investigations.

Dr. Kenneth Platt presented "Subunit Pseudorabies Vaccines" and indicated that caution should be exhibited in the continued use of modified live vaccines to prevent the potential propagation of the virus. Subunit vaccines have specific antigenic capabilities which allow the pig to produce specific detectable antibodies for diagnostic procedures. Various techniques will allow genetic engineering and cloning with selected bacteria and isolate exact proteins to fabricate vaccines. The vaccine currently being studied requires two doses which create a serum neutralization titer of 1:2 in approximately 3 weeks.

Dr. David Thawley gave a detailed discussion of the Livestock Conservation Institute survey on clean up programs used in PRV infected
herds. The four basic programs used are, test and slaughter, depopulation with repopulation, herd vaccination and progeny isolation. Certain guidelines for each type of clean up program were described indicating the advantages and disadvantages of each. It was impressed upon the audience that selection of the clean up program must be designed to each individual situation to achieve the most effective method for the desired clean up goal.

Dr. E. A. Carbrey presented “An Update on ELISA for Pseudorabies” Comparative diagnostic efficiency between the serum neutralization test (SN) and the enzyme linked immuno-adsorbed assay test (ELISA) on 200 identified serum samples indicated that the percentages of agreement were 96.6 and 97.5 respectively. The advantages of the ELISA test were that more specific antigen can be prepared and standardized, no cell culture is necessary, it can be completed within a few hours and lower quality serum may be used. The disadvantages were a high cost of equipment, the potential costs of a commercial antigen, no qualitative measurement and the initial training expense.

Dr. Paul Spencer presented a paper detailing the exposure of swine to PRV which occurred at the Lubbock, Texas exhibition. The epidemiological surveys that were necessary to be conducted by the many states receiving swine resulted in a tremendous expense to those states as well as the resulting fiscal impact to the owner of the exposed herds. This fiasco clearly indicates that more rigid control measures are necessary to be developed at such exhibitions and also indicates the absolute need to isolate exhibition animals returned to the owner’s premise with a retest within a prescribed time before placing these animals back in the swine herd.

Dr. Leroy Schurrenberger reviewed the proposed pilot programs proposed by several states to control PRV. Illinois, Wisconsin, Iowa, North Carolina and Pennsylvania have developed individual programs of control to pursue, but it is very evident that these proposals will be difficult to pursue with the present fiscal restraints imposed to the U.S.D.A. budget. Wisconsin indicated that it was probable that their program would proceed without U.S.D.A. monies.

Dr. Schurrenberger then reviewed the proposed changes in CFR Part 85—Pseudorabies published in the Federal Register November 3, 1982. Dr. T. F. Zweigart made a motion to approve the proposed regulation and the motion was seconded by Dr. Paul Doby. In the discussion of the motion, the following sections of the regulation were discussed.

85.1 (1) (2) (i) The release of quarantine for infected herds with 1 negative test 30 days after the removal of the tested animals was thought by some to be inadequate.

85.1 (3) (iii) The privilege of determining epidemiologically those animals showing SN titers of 1:4 and 1:8 was accepted as a desirable procedure to determine laboratory error by conducting an additional test. The use of the
REPORT OF THE COMMITTEE

epidemiologist to determine which additional animals would be retested was thought prudent but the retest of SN titers of 1:8 was questioned.

85.1 (ee) The flexibility of monitoring qualified herds including the isolation and retest of exhibition swine was thought valid, however, most felt that C&D procedures should be maintained for infected premises.

85.1 (ff) The proposal to leave 10% of the herd unvaccinated in a Pseudorabies Controlled Vaccinated herd and testing these unvaccinated animals as an alternative monitoring procedure to progeny testing was discussed and some members felt this procedure inequitable in endemic areas.

85.1 (jj) The definition of an “official pseudorabies test” was thought to be adequate but discussion indicated that the recognition of the ELISA as an official test should be expedited immediately.

85.5 (a) The identification of slaughter swine on an ownership statement was thought to be an excessive demand until an effective method for identification back to the farm of origin was found to allow for enforcement.

85.7 (b) Many present found the flexible movement of feeder pigs of unknown origin for 4 interstate movements to be excessive.

When the question was called, the committee members voted 6 for the motion, 6 against the motion. In casting his vote against the motion, the chairman stated that in view of the depth of the discussion on the various points listed, that those present, both the industry and regulatory members, should objectively edit the proposed regulation to create an effective disease control program and express their desires to the Deputy Administrator.
RABIES DIAGNOSIS, A NEW APPROACH: SKIN BIOPSY METHOD FOR ANTEMORTEM DIAGNOSIS OF RABIES

Dennis R. Howard, MS, PhD

SUMMARY

Rabies virus antigen can be demonstrated in the skin of animals which have been naturally infected. Frozen sections of skin biopsies from the muzzle of animals were stained by the direct fluorescent antibody test (FAT). Specific fluorescence could be demonstrated in nerve bundles and nerve fibers surrounding hair follicles. The species studied included the bovine, equine, canine, feline, and the striped skunk.

INTRODUCTION

Rabies virus antigen has been demonstrated in numerous tissues, including skin, of naturally infected skunks.4,5 Rabies virus was detected in the skin of mice inoculated with rabies virus of bovine, feline or canine origin.7 Antigen was found as early as four days prior to the onset of clinical rabies.

Since then, rabies has been diagnosed in many species of animals using skin sections.3 Rabies infection was also predicted and diagnosed by skin biopsy in a litter of skunks.2

MATERIALS AND METHODS

A six mm punch is used to take the biopsy from the muzzle of animals. The biopsy should be taken to include several hair follicles and deep enough to include subcutaneous tissue. If the lateral sensory papillae are removed, an elliptical incision using a scalpel is most effective. The punch or incision should be closed with suture material.

On the bovine or equine, large sections are taken from the muzzle using a scalpel. The sections should be at least 3/4 inch long and 1/4 inch wide.

The skin section is frozen immediately and submitted to a laboratory for examination. Laboratory examination is described in previous papers.4,6

DISCUSSION

The skin serves as a major sensory organ. Dermal nerve endings supply the skin with sensations of touch, pressure, temperature, pain and itching and terminate principally in papillary dermis and around hair follicles.1

The use of skin biopsies or sections has proven to be an effective antemortem method for rabies diagnosis in several animal species. Specific fluorescence can be demonstrated in nerve fibers surrounding hair follicles.
REFERENCES


INTRADERMAL USE OF HUMAN DIPLOID CELL VACCINE FOR PRE-EXPOSURE RABIES VACCINATIONS OF HUMANS.*

David W. Dreesen, DVM, MPVM, John Brown, DVM, PhD, John W. Sumner, BS, and Douglas T. Kemp, Pharm. D.

SUMMARY

Rabies human diploid cell vaccine has now supplanted duck embryo vaccine for human use for pre- and post-exposure rabies immunizations. The current regimen for pre-exposure immunizations with human diploid cell vaccine requires that the individual receive three 1.0 ml doses administered intramuscularly in the upper deltoid region of the arm on days 0, 7, and 28. Recent studies have shown that this vaccine can be administered intradermally in low-dose (0.1 ml) quantities using the same time frame. Twenty-one days following the third intradermal dose, 100% of the recipients achieved a titer of $\geq 0.5$ international units per ml of serum, a titer the World Health Organization considers as having confirmed immunity against natural infection. Final approval of the intradermal regimen is expected in early 1983. This will allow economical use of a highly efficient and safe rabies vaccine.

INTRODUCTION

Pre-exposure immunization for individuals at high risk to rabies has been recommended since the introduction of duck embryo vaccine (DEV) in 1957. Rabies pre-exposure immunization reduces risk in persons at the greatest risk of exposure; it provides for a more rapid anamnestic response and eliminates the need for immune globulin should exposure occur. Duck embryo vaccine induces an adequate antibody response in only 70-90% of those receiving 3-4 doses prophylactically, but until recently, it was the only human rabies vaccine available in the United States. Also, DEV has induced severe systemic, neuroparalytic, and anaphylactic reactions in a small percentage of recipients. The vaccine is no longer marketed in the United States.

With the successful growth of rabies virus in human diploid cell strain WI38 and with the subsequent licensing of the human diploid cell vaccine (HDCV) by the Food and Drug Administration, U.S. Public Health Service, a much safer and apparently more efficacious vaccine was made available for both pre- and post-exposure immunizations. The HDCV has been found to be highly antigenic with rapid antibody response, and has been found extremely safe when used IM for either pre- or post-exposure immunization. Rabies HDCV is produced by the Institut Merieux in France, in the U.S. by Wyeth Laboratories, and in the Federal Republic

*Portions of this paper have been accepted for publication in the Journal of the American Veterinary Medical Association.
of Germany by Behringwerke A.G. The Behringwerke vaccine is not currently licensed for use in the United States.

When HDCV is administered intradermally (ID) or subcutaneously in low dose (0.1, 0.2, or 0.25 ml) amounts in various regimens, seroconversion is achieved in greater than 99% of recipients.\(^3,16,20\) Titer apparently will remain adequate for periods up to 1 year or more.\(^19,20\) In the present study, a 99% seroconversion rate for persons having \( \geq 0.5 \) international units (IU) of rabies neutralizing antibody per ml of serum was set as the minimum acceptable degree of protection. The titer of 0.5 IU/ml is recommended as the minimum to be attained to demonstrate seroconversion after use of HDCV.\(^21\)

Since faculty, staff, and students of veterinary colleges, among others, are high-risk candidates for exposure to rabies, especially in a rabies enzootic area such as Georgia, the administration at the College of Veterinary Medicine, University of Georgia authorized the use of HDCV for prophylactic treatment of all those at risk, at no cost to the individual. In view of financial considerations, which have been expressed previously\(^22\), and based on information from the studies cited above, it was proposed to administer the HDCV via the ID route rather than by the intramuscular (IM) route.

The standard to be achieved for acceptable protection against rabies virus following pre-exposure immunization is a titer of \( \geq 0.5 \) IU/ml of serum. The primary question that was to be answered in this study was whether individuals would meet this standard following a regimen of three 0.1 ml doses of rabies HDCV administered ID on days 0, 7, and 28.

**MATERIALS AND METHODS**

In the fall of 1981, students in the third- and fourth-year classes, graduate students, faculty, and staff at the College of Veterinary Medicine were offered the opportunity to volunteer for inclusion in the study. Each individual was required to sign a consent form approved by the University Human Subjects Committee. The consent form advised that HDCV was approved for IM use only and informed the signatory of potential reactions to HDCV administered ID. A medical history obtained from all volunteers was reviewed by a physician prior to initial vaccination. A vaccine reaction form was included for assessment of reactions to each of the 3 doses that were to be administered.

**Vaccine:** The vaccine used was a single lot (VO 456) of HDCV with an antigenic value of 3.41 IU/ml. It was produced by Merieux Institute. This vaccine is a sterile, stable, freeze-dried suspension of rabies virus prepared from strain PM-1503-3M obtained from the Wistar Institute, Philadelphia. Each vial of vaccine was reconstituted with 1.0 ml of sterile water provided in the pre-packaged material which includes the freeze-dried virus, diluent, syringe and needle. Doses of 0.1 ml were drawn into
INTRADERMAL USE OF HUMAN DIPLOID CELL VACCINE

1-ml disposable tuberculin syringes with 25-ga, 5/8" needles attached. The mean number of doses available per vial was 8.7 when syringes were reused with a new needle attached for each vaccination.

Serology: All 240 subjects who completed the entire regimen received the 3 doses of 0.1 ml HDCV ID in the upper deltoid region of the arm on days 0, 7, and 28. A 6-to-8-ml blood sample was obtained on days 0 and 49 for titer determination by the rapid fluorescent focus inhibition test (RFFIT). Titer was estimated by the Reed and Muench method and expressed in international units (IU)/ml of serum. The titer of the international standard antiserum (NIH lot no. R-2) varied between 1:60 and 1:180. The challenge virus standard was diluted to an estimated 50 TCID₅₀. A back titration was made for each test to determine the actual number of TCID₅₀ added. This number ranged from 32-100. Using these reference serum values, a reciprocal antibody titer of 1:15-1:45 corresponded to the minimal acceptable IU/ml value of 0.5.

Subjects: The ages of the 240 subjects ranged from 19 to 63 with a mean of 28.7. There were 108 women and 132 men. Each individual was assigned to 1 of 3 groups depending on whether they had received rabies immunization in the past. In nearly all cases, the previous immunization was with DEV. Group I included 62 individuals who had received rabies vaccine within the 2 year period preceding the initial ID dose of HDCV. Group II consisted of 92 people who had received rabies vaccine 2 years or more before the first ID dose of HDCV. Group III included 86 individuals who had never received rabies immunization. There was no significant difference in age range or sex distribution between any 2 of the 3 groups. For purposes of comparison with group III, groups I and II are at times combined as one group of "previous vaccinates."

The vaccine reaction form was completed by 224 of the subjects to ascertain postvaccinal reactions in 12 categories.

Statistical Methods: In keeping with other recent reports the international unit per milliliter IU/ml has been used to report RFFIT titer results. To facilitate analysis of data of this type the reciprocal of the RFFIT titer was transformed using the logarithmic transformation. Because of variabilities inherent in the results obtained by the RFFIT in high titer ranges, an index was devised to correlate results obtained from and between groups. A number of 1 was assigned to titers in the range of 0.5-0.9 IU/ml (minimal); the number 2 was assigned to titers in the range of 1.0-4.9 IU/ml (low); 3 was assigned to titers in the range of 5.0-9.9 IU/ml (moderate); and for titers ≥ 10.0 IU/ml (high), the number 4 was assigned.

The data that had been transformed using either the logarithmic transformation method or the index method were initially analyzed using a one-way analysis of variance. The geometric mean titers (GMT) for the transformed logarithmic data were compared using the Scheffe test.

---

*Monoject, Division of Sherwood Medical, St. Louis, MO, 63103.*
From within the 2 groups of previous vaccinates, 2 additional subgroups were identified. The first subgroup consisted of 54 individuals who were known to have developed antibody following rabies vaccination prior to this intradermal trial; the second subgroup (24 individuals) had not developed antibody from previous rabies prophylaxis or had no information concerning their past titer. For purposes of computation, a value of 0.02 was given to these undetectable or unknown titers. Geometric mean titers and standard deviations were calculated on an IU/ml basis for these 2 subgroups and significance of titer response at day 49 was determined using Student's t test.

RESULTS

As shown in Table 1, all 240 subjects given the intradermal HDCV developed antibody with a titer of ≥ 0.5 IU/ml, regardless of previous vaccination history. Group I vaccinates had 31 subjects (50%) with titers in the high range. In comparison, group II subjects had 34 (37.1%) in the high-titer range whereas group III subjects had only 18 (20.9%) with titers ≥ 10.0 IU/ml. The largest percentages of individuals in groups II and III were in the moderate titer range, 46.7% and 65.1%, respectively.

Utilizing the index concept (values of 1, 2, 3, and 4 assigned to IU/ml titer ranges of 0.5 - 0.9, 1.0 - 4.9, 5.0 - 9.9, and ≥ 10.0, respectively), the mean index value and 95% confidence intervals (CI) were: group I, 3.03 ± 0.26; group II, 3.19 ± 0.16; and group III, 3.07 ± 0.13. One-way analysis of variance demonstrated no significant differences among the 3 groups (F = 1.22, df = 2/237, not significant). A follow-up multiple comparison test (Scheffe) confirmed this finding. The mean index value for the combined "previous vaccinates" was 3.05.

Antibody titers were determined for 54 individuals who knew that they had developed antibody following rabies (DEV) immunization at some time prior to this trial and from 24 individuals who knew that they had not previously developed antibody or did not receive results from the serum testing. The GMT for the 54 subjects who had developed antibody was 11.04 IU/ml, with a 95% CI ranging from 8.66 to 14.00. The GMT for the other 24 was 8.79 IU/ml, with a 95% CI ranging from 5.97 to 12.80. When the GMT of these two subgroups was compared by Student's t test, it was found that these means were not significantly different (t = 1.044, df = 76, not significant [P > 0.05]).

Twenty-nine subjects had residual titers on day 0. When these titers were compared with the titers on day 49, an analysis of variance for linear regression showed there was no significant difference if dependency or association was assumed.

Table 2 shows the percentage of individuals with mild or moderate vaccine reactions. Groups I and II are combined as "previous vaccinates." Five individuals in the previously vaccinated group reported "severe" pain at the site of injection, muscle ache, or headache following the second (day 7) injection.
DISCUSSION

In November 1981, the Centers for Disease Control discontinued testing of sera from individuals who completed a series of pre-exposure IM rabies immunizations with HDCV, since all individuals tested had developed antibody.\textsuperscript{24} It is therefore imperative that persons at risk to rabies be given vaccine in a regimen that will assure an adequate antibody response in 99\% or greater of the recipients. In addition, the vaccine should be available at a reasonable price so that immunization is not cost-prohibitive.\textsuperscript{22} The HDCV now marketed in the U.S. by Merieux Institute meets the requirements of a safe, highly immunogenic vaccine. The regimen used in the study reported here (0.1 ml administered ID on days 0, 7, and 28) resulted in 100\% of individuals developing an antibody titer of $\geq 0.5$ IU/ml, a standard that the World Health Organization considers as having conferred an acceptable degree of resistance to natural infection.\textsuperscript{21} These small quantity 0.1 ml doses cost approximately $6.25/dose (about 8 doses/1 ml vial) or less than $19 per individual for safe protection.

On the basis of the index system of reporting, it is assumed that there was no difference in titer response regardless of rabies vaccine history. Whether a person had or did not have demonstrable antibody titer in the past from DEV had no bearing on the titer response achieved when a complete 3-dose HDCV primary series was administered ID.

It was further found that the antibody response of those few (29) individuals who had a residual titer on day 0 was independent of this original titer. In some individuals the titer rose to $> 10$ IU/ml, in some it remained virtually the same, whereas in a few the titer decreased.

As shown in similar studies, localized redness, pain, swelling, and itching at the site of an ID injection of HDCV are more common than with the IM route of administration of this vaccine.\textsuperscript{19} Although there were 5 severe reactions involving pain at the site of injection, muscle ache, or headache following the second injection, this represented only 0.69\% of the 720 inoculations. None of these 5 severe reactions were so intense as to prevent the subject from completing the 3-dose course of immunization.

Since the response to 3 ID doses of HDCV has been so effective, as demonstrated in this study, the Advisory Committee on Immunization Practices, USPHS has suggested that routine serologic testing for antibody response is no longer necessary whether the IM or ID route of administration is used.\textsuperscript{25} Although the ID regimen is an acceptable alternative for rabies pre-exposure immunization, the Food and Drug Administration, USPHS, is withholding final official approval until the manufacturer makes necessary changes in the packaging and labeling of the vaccine.\textsuperscript{25} Trials are currently underway at the College of Veterinary Medicine, University of Georgia on a new delivery system (syringes containing individual 0.1 ml doses) which should provide the data necessary for final FDA approval for the 3-dose 0.1 ml ID regimen.
For now the HDCV is only distributed in vials that reconstitute to 1.0 ml; therefore, for the most economical pre-exposure use of the vaccine, multiples of 6-8 people should participate in the immunizations at the same time. According to current recommendations of the ACIP, USPHS, individuals should receive a booster dose of HDCV every 2 years administered either 0.1 ml ID or 1.0 ml IM. Should a person who has received HDCV by the 3-dose regimen be exposed to rabies through a bite or other means of contact with a known or suspected rabid animal, it is recommended that two 1.0 ml doses of HDCV be administered IM 3 days apart.
Table 1. Distribution of rabies antibody titers (day 49) expressed in international units/ml of serum after pre-exposure immunization with three 0.1 ml intradermal doses of human diploid cell vaccine on days 0, 7, and 28.

<table>
<thead>
<tr>
<th>Group</th>
<th>Vaccine History</th>
<th>Distribution of titers among vaccinates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>&lt;0.50</td>
</tr>
<tr>
<td>I (n=62)</td>
<td>Vaccinated</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>&lt;2 Yrs. Ago</td>
<td>0%</td>
</tr>
<tr>
<td>II (n=92)</td>
<td>Vaccinated</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>&gt;2 Yrs. Ago</td>
<td>0%</td>
</tr>
<tr>
<td>III (n=86)</td>
<td>No Previous Vaccination</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td>TOTALS</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group</th>
<th>Vaccine History</th>
<th>Distribution of titers among vaccinates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>&lt;0.50</td>
</tr>
<tr>
<td>I &amp; II Combined</td>
<td>Previous Vaccinates</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0%</td>
<td>100%</td>
</tr>
<tr>
<td>III</td>
<td>No Previous Vaccination</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td>0%</td>
<td>100%</td>
</tr>
</tbody>
</table>
Table 2. Percent of individuals showing mild to moderate vaccine reactions following each of 3 I/D doses of 0.1 ml HDCV.

<table>
<thead>
<tr>
<th>Reactions following each of 3 injections</th>
<th>Previous Vaccinates (N=142)</th>
<th>No History of Previous Vaccine (N=82)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day of Immunization</td>
<td>Day of Immunization</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>1. Redness at Site</td>
<td>60.6</td>
<td>67.6</td>
</tr>
<tr>
<td>2. Pain at Site</td>
<td>14.8</td>
<td>24.6*</td>
</tr>
<tr>
<td>3. Swelling at Site</td>
<td>22.5</td>
<td>40.8</td>
</tr>
<tr>
<td>4. Itching at Site</td>
<td>23.9</td>
<td>43.7</td>
</tr>
<tr>
<td>5. Generalized Itching</td>
<td>1.4</td>
<td>2.8</td>
</tr>
<tr>
<td>6. Fever</td>
<td>0.7</td>
<td>3.5</td>
</tr>
<tr>
<td>7. Muscle Aches</td>
<td>4.2</td>
<td>6.3*</td>
</tr>
<tr>
<td>8. Lymphadenopathy</td>
<td>0.7</td>
<td>1.4</td>
</tr>
<tr>
<td>9. Malaise</td>
<td>4.2</td>
<td>6.3</td>
</tr>
<tr>
<td>10. Headache</td>
<td>5.6</td>
<td>7.0*</td>
</tr>
<tr>
<td>11. Dizziness</td>
<td>7.0</td>
<td>4.9</td>
</tr>
<tr>
<td>12. Nausea</td>
<td>3.5</td>
<td>2.8</td>
</tr>
</tbody>
</table>

* 5 Severe Reactions
REFERENCES


26. Winkler WG: Personal communication. Center for Disease Control, CDC, Atlanta, Ga.
REPORT OF THE COMMITTEE ON RABIES

Chairman: Leon Russell, College Station, TX
Vice Chairman: W. R. Miller, Auburn, AL

W. H. Beckenhauer, NB; John Brown, GA; R. R. Brown, AL; J. W. Glosser, MT; J. A. Gourlay, IA; B. Hancock, IA; D. R. Howard, KS; Bruce Kaplan, KY; O. L. Kelsey, AR; F. V. McCasland, TX; R. A. Robinson, MN; F. T. Satalowich, MO; E. L. Shroyer, NY; J. M. Shuler, IN; A. Strating, IA, Oscar Sussman, FL; W. G. Winkler, GA

The committee met on November 9 with 18 members and guests present.

Old Business: The committee reviewed recommendations made at the 1981 meeting and discussed the resulting actions during the past year.

New Business: Four formal paper were presented to the committee.


3. “A Proposed Index System for Reporting RFFIT Titers”, by J. Brown, University of Georgia, Athens, Georgia.

4. “Systemic Dissemination of Rabies Virus”, by D. R. Howard, Kansas State University, Manhattan, Kansas.

Positive discussion was stimulated by each presentation.

The committee considered additional new business as follows:

1. Handling food animals exposed to rabies. J. C. Prucha, FSIS, USDA, led discussion concerning procedures for handling animals exposed to rabies and subsequently submitted for slaughter at USDA Inspected slaughterhouses. The difficulty of writing regulations which cover all aspects of slaughter, including worker protection, was emphasized. Positive discussion resulted in understanding of the problems associated with the slaughter of rabies exposed livestock.

2. A Proposed Handbook of Professional and Technical Medical Personnel. D. R. Howard, Kansas State University, led discussion of the need for a handbook to guide medical and related personnel on how to handle rabies problems. (After extensive discussion, the committee agreed by common consent that a study should be made.) A sub-committee was appointed to study the feasibility and potential production of a “Rabies Handbook” by the USAHA. The sub-committee will be Dr. R. Howard, Chairman, J. M. Shuler and W. G. Winkler.

The committee was adjourned.
THE PREVALENCE OF SALMONELLA IN VARIOUS AGE GROUPS OF MARKET SWINE

J. W. Nelson, R. A. Robinson, M. M. Pullen, L. Ratananakorn,
College of Veterinary Medicine University of Minnesota, St. Paul, MN

INTRODUCTION

The purpose of this paper is to report on the level of salmonella infection found in various age groups of swine from a project just concluded in Minnesota. The importance of salmonella levels in swine lies in the great exposure potential to humans through the food chain. Along with the human health risk is the economic losses to the pork industry as a result of clinical swine salmonellosis.

The present study focused on the prevalence levels of salmonellas in slaughtered swine and to draw some inferences as to the factors involved in salmonella transmission within the swine population.

This study was done in conjunction with the U.S. Department of Agriculture to evaluate the role of the stockyard environment in the transmission of salmonellas to food animals as they pass through the yards. Since St. Paul has one of the largest stockyards in operation, with 1978 annual sales of 2.8 million livestock of which 60% were swine, it was a logical and convenient stockyard to evaluate. This stockyard is also located in close proximity to the large sources of swine originating from Minnesota, Wisconsin, South Dakota and Iowa. For example, in Minnesota alone 5.5 million pigs were slaughtered commercially in 1981.

HUMAN SALMONELLOSIS

Salmonellas are one of the major causes of bacterial gastroenteritis in the United States, being responsible for over one half of the food borne outbreaks reported. Food of animal origin is the primary source, and poultry, meat and dairy products are the most common vehicles of transmission.

The importance of pork as a vehicle of transmission apparently has not been significant in the United States although the 1978 Centers for Disease Control (CDC) Salmonella surveillance annual summary reported seven outbreaks of food borne salmonellosis from eating pork which produced illness in 850 individuals. Unidentified sources of infection accounted for 27% (14 of 51) of the outbreaks. In 1979 there was only one occurrence of food borne salmonellosis directly attributed to pork but approximately 1200 human clinical cases resulted. In 1979 of the reported food borne outbreaks 34% (23 of 67) were from an undetermined source.

European countries have reported that pork plays a significant role in human salmonellosis. In the Netherlands for example, pork and pork products were the most important source of human infection (Edel et al, 1967). Barrell (1982) reports that salmonellas were responsible for 75% of all bacterial food poisonings in England and Wales over a three year
period (1976-1978). Likewise Lee (1974) stated that in the period 1960-1970 meat and poultry products caused over 70% of the successfully investigated cases of human salmonellosis. Therefore, we see in England and Wales no change in the patterns of transmission over nearly 20 years of surveillance. Likewise in the United States, Silliker (1982) recently stated that in regard to salmonellosis nothing has changed as the disease in humans is increasing and the control measures which have been tried have not been overly successful.

There have been changes in the epidemiology of human salmonellosis: for example, Lee (1974) reported that since 1960 there are a greater number of human incidents caused by *Salmonella* serotypes other than *S. typhimurium* in both England and Wales. Barrell (1982) reported recently that a serotype being observed more frequently in food borne human outbreaks in England and Wales is *S. hadar*. In the United States in recent years we have seen new serotypes such as *S. hadar* surface in poultry, swine and man (Pomeroy personal communication) and *s. dublin* has moved from its enclave west of the Rocky mountains to infect cattle and enter the human food chain east of the mountains. (Blackburn, et al., 1981).

**PORCINE SALMONELLA INFECTIONS**

Isolations of salmonellas from various samples of slaughter swine as reported by workers in several countries are shown in table 1. Using cecal contents as a sampling site, the isolation rates ranged from 5.8- to 75%. While it is hazardous to make international comparisons because of variation in microbiological techniques, as well as the distances swine travelled to slaughter, the times swine were held in the pens prior to slaughter and feeding methods, one is left with the strong impression that slaughter swine could be an important source of salmonellas for humans.

The classic study of Galton et al (1954) demonstrated a marked increase in salmonellae isolation as the swine moved from the farm (70%), sales barn (9%), holding pens at the abattoir (26%), and at slaughter (51%) using fecal swabs. These results gave rise to what has been called the "build-up theory" where it is presumed that the pig has a low level of salmonella infection on the farm and as the pigs progress along the route to slaughter acquires infection from other pigs or the environment.

Williams and Newell (1970) demonstrated that in a swine herd where no isolations had been made on the farm, did, after transportation, commence shedding salmonelllas. This has given rise to the view that the pig acts as a "biological filter" and although clinical illness is not observed, infection occurs, and the pig becomes an asymptomatic carrier. As Haddock has stated (1970) the so called "build-up" may actually be a manifestation of a pre-existing infection.

Feeds, especially those containing animal protein supplements, have long been suspected as the major source of salmonellas for swine. For
example, Newell et al. (1959) traced infected swine at slaughter back to the farm of origin and found the same serotypes in the feed as were present in the pig.

The emergence of *S. agona* during the early 1970’s in Peruvian fish meal is a classic example of the movement of a salmonella serotype through both animal and human populations i.e. contaminated feeds and food (Clark et al, 1973).

Nagaraja and Pomeroy (1982) have demonstrated that it is possible to present to slaughter a salmonella-free bird. This can be accomplished by controlling feed contamination, either by removing all animal by-products from the feed or by pelletizing the feed with heat. Edel et al, (1966) demonstrated, in a controlled experiment with piglets, that by feeding pelletized feed they were unable to isolate salmonellas. These researchers gave evidence that by not feeding salmonellas to animals we can control or reduce prevalence levels.

Table 2 shows the level of salmonellae that have been isolated on swine farms by various investigators world wide. The isolation rate from examining fecal specimens is higher when compared with the use of a fecal swab (Haddock, 1970). A fecal swab does not get an adequate quantity of feces to place into enrichment broth.

These data demonstrate that the levels on the farm are lower than at slaughter. Unfortunately, some investigators failed to state what age group of swine were examined. For example, Ghosh (1972) grouped several age categories into one and stated that 423/773 (53%) of swine fecal samples collected were positive for salmonellas; however, later in his discussion he shows the separation into groups i.e. sows, boars, young boars, gilts, and young animals to 65% in the older sows and boars.

In most instances salmonellas are found at low levels in pigs soon after birth. Williams and Newell (1968) sampled 203 pigs just weaned and made only one isolate of salmonella. Guinee et al, (1965) sampled piglets every two weeks of life and found that 12.7% of the piglets were positive for salmonellas during the first eight weeks of life.

**SWINE MOVEMENT AND MARKETING PATTERNS**

The marketing movements of swine in a midwestern area are shown in figure 1.

Feeder pigs (recently weaned animals 6-8 weeks of age) originate in Minnesota and adjacent areas of Wisconsin and in turn move into a wider area of farms including Iowa, South Dakota and occasionally other states. A weekly feeder pig auction is held at the St. Paul Union Stockyards. Pigs arrive about 7:00 am, are vaccinated for Erysipelas, tagged and are sold and dispersed by 4:00 pm the same day. About 2000 pigs are sold weekly at this sale. Strict standards are maintained at this sale through: (1) a stringent veterinary examination of all animals at entry with rejection of any clinically ill consignments, (2) utilization of an area of the
stockyards isolated from other livestock, and (3) maintenance of holding pens in a good hygienic state.

Market age swine (200-220 lb. live weight) originate from farms in a four state area (Minnesota, Wisconsin, Iowa, South Dakota) and are transported: (1) to the stockyards, (2) to a buyer or buying station, (3) on occasions an auction market can function as a buying station, or (4) directly to the slaughter plant (see table 3).

The great majority of market swine are in the stockyards for less than 12 hours. Exceptions are a few unsold pigs at the end of each day or occasionally larger numbers held over a weekend. The slaughter establishment in this study purchased a large proportion of their swine from sources other than the St. Paul Union Stockyards. However, when the demand for swine exceeded the supply from other sources or if other factors, i.e. economics or weather, affected the flow of swine to the slaughter establishment, the buyers at the stockyards purchased pigs to meet the quotas.

Boars and sows are generally collected in stockyards or auction markets from a very wide area and then transported directly to the slaughter establishment. There are only three federally inspected slaughter establishments that kill boars in the North Central region of the United States. They are located in Minnesota, Illinois and Michigan. Therefore, these boars can be intransit or held in pens for 3-5 days prior to slaughter.

**SAMPLING PROGRAMS**

a) *Feeder Pigs:* Samples were collected on auction days during a 12 month period. Each day, pens were randomly selected and within each selected pen fecal swabs from five randomly selected pigs were obtained.

b) *Market Swine:* Because of handling problems and so as not to interfere with the commission firms operating in the stockyards, market swine were sampled at slaughter.

c) *Cull Boars:* Samples were taken on four occasions at the time of slaughter.

**SLAUGHTER HOUSE SAMPLING**

The sampling procedure consisted of removing one mesenteric lymph node (MLN) and a quantity of the cecal contents (CC). The slaughtering plants identification tattoo for the carcass being sample was recorded. The knife used in removing the MLN and in incising the cecum was sanitized in hot water between each collection.

**SALMONELLA ISOLATION TECHNIQUES**

The routine methodology throughout this study is demonstrated in Figure 2. The microbiological isolation techniques were selected as a compromise between efficiency of isolation and the ability to examine
large numbers of samples. There was no direct plating of samples. Some salmonella isolates were confirmed or serotyped at the National Services Laboratory, Ames, Iowa.

RESULTS

Feeder Pigs: Only 2/809 (0.25%) revealed Salmonellas (S. enteritidis and S. derby). See Table 4.

Market Swine: Salmonellas were isolated from 494/1920 (25.7%) animals sampled at slaughter (Table 4). The sites of isolation and the most common serotypes identified are shown in Table 5 and 6. In Table 7, swine are separated into two groups as to whether or not they had passed through the St. Paul stockyards. The latter group may have come directly from the farm of origin or have come via an intermediary buying station. There is a significantly higher salmonella isolation rate in the St. Paul stockyard passed animals utilizing either mesenteric lymph nodes or cecal contents as an indicator of infection. The salmonella serotypes isolated from swine did not appear to differ significantly depending upon whether they had passed through the St. Paul stockyard or came directly to slaughter.

Although sampling was not equally distributed over the work week on which slaughter was carried out, it does appear that a higher prevalence of Salmonella isolations was encountered during the first three days of the week (Table 8). The situation was confounded by two factors—some of the pigs killed on Mondays had been held over the weekend and during midweek the slaughter house often supplemented their source of pigs with extra large purchases from the St. Paul stockyards.

Monthly variation of Salmonella isolations at slaughter are shown in Table 9. There appears to be a higher isolation rate in the summer months. This again may well reflect the buying practices of the slaughter house since they do purchase more from the St. Paul stockyards during the summer.

Samples were collected from the St. Paul stockyards environment and the results are shown in Table 10.

Cull Boars: On four sampling days, 158/200 (79.0%) boars sampled yielded salmonellas from either cecal contents or mesenteric lymph nodes (Table 4). The five most common serotypes isolated were: Group B; G complex; S. agona; S. anatum; S. infantis and S. bareilly.

DISCUSSION

The differences in the Salmonella isolation rates are highly significant for the three age groups examined (Table 4). Based upon a single negative fecal swab, failure to demonstrate significant Salmonella excretion (0.25%) in an extensive sampling of feeder pigs over a 12 month period does not eliminate the possibility that some of these animals may be latently infected. Nevertheless, it does appear that the level of salmonella infection in these animals is extremely low. Presumably
piglets in their early life can be exposed to salmonellas from the sow, the environment or feed. While it is almost universal to offer creep feed to pre-weaned piglets they do not usually consume large amounts but often play with feed. Therefore, any salmonella exposure is likely to involve small numbers of bacteria with less likelihood of fecal excretion occurring later.

The significant rise in salmonella infection detected in market hogs (25.7%) indicates that from weaning to slaughter the exposure potential has changed significantly. The most likely culprit is feed containing salmonella contaminated animal protein.

Although the specific serotypes found in feeds may not correspond quantitatively with the types found at slaughter, this does not necessarily invalidate an association (Public Health Laboratory Service Working Group 1972). Some salmonella serotypes may well have advantages over others in environmental multiplication or persistence. Because of both economic and other disease factors, some producers in the mid-west have now ceased using animal protein in their swine rations. Future studies should include a detailed comparison of salmonella prevalence in swine on farms using protein derived from either rendered products or plant origin.

A number of studies have demonstrated that the handling practices after hogs leave the farm of origin affect the rate of salmonella isolation at slaughter. These include the time intervals, transport, feeding practices and environmental contamination. It was not possible to determine transportation times from farm to slaughter, but it was obvious that the holding times for the St. Paul stockyard pigs was longer than those trucked directly from farm to slaughter. A major contributory factor to the increased salmonella isolation rate from the St. Paul stockyard—passaged swine is most likely due to the fact that the majority of these animals are fed corn from the floor. The demonstrated environmental contamination of both feed, floor and water sources (Table 10) in the St. Paul stockyard obviously encourages fecal-oral transmission of salmonella. As mentioned earlier in the paper, recent Australian studies demonstrated a significant drop in cecal salmonellas isolated from pigs held in the pens prior to slaughter but not fed (Craven & Hurst, 1982).

In this study there were no days on which salmonellas were not isolated from market swine. The daily variation in salmonella isolation can be explained in part by some swine being held over the weekend and also by additional mid-week purchases from the stockyard. The explanation for seasonal variation in isolation rates may be due to the slaughter plant purchasing a greater percentage of their swine from the St. Paul stockyard during the summer months.

The significant differences between the isolation of salmonellas from the market swine in comparison with cull boars (25.7% vs 79%) is even larger than those reported by Keteran et al (1982) for adult sows and
market hogs. While longer transit and holding times prior to slaughter are the most likely explanation for these differences in both studies, the older animals have perforce been exposed to salmonellas in feeds for much longer periods that market swine.

While clinical enteric salmonellosis is only occasionally seen in preweaned pigs, septicaemic *S. cholerae-suis* infections are more often reported post weaning according to the Minnesota Veterinary Diagnostic Laboratory records. It is interesting to note in this study that the only isolates of the *S. cholerae-suis* made from slaughter hogs were made from the mesenteric lymph nodes.

While the present state of the art in rearing and feeding swine to achieve a salmonella free product has not been achieved, there are a number of procedures that are very likely to reduce both infection of the live animal and contamination of meat. These include a greater emphasis on Salmonella-free feeds, feeding and watering practices that do not encourage fecal-oral transmission, an all-in-all-out production system and readily cleaned and decontaminated housing. Transit times to the slaughter house should be reduced wherever possible and the added risks of holding and feeding pigs in salmonella contaminated environments recognized.

**SUMMARY**

Three age groups (feeder pigs, market swine and cull boars) in a large midwestern swine market or at slaughter were examined for *Salmonella* sp. Based on fecal swabs 0.25% (2/800) feeder pigs revealed salmonella while at slaughter 25.7% (494/1920) market swine and 79% (158/200) cull boars demonstrated a variety of salmonella serotypes either in the mesenteric lymph nodes or cecal contents.

There was a significantly higher isolation rate in market swine that had passed through the St. Paul stockyards prior to slaughter as opposed to animals that had moved directly from the farm. Higher isolation rates were obtained from slaughter swine at the beginning of the week which may be explained in part by animals being held over the weekend. Higher isolation rates were obtained from market swine during the summer months. It is suggested that feeding practices including potential feed contamination with salmonellas may be responsible for the increasing prevalence of salmonella isolations with increasing age.

**ACKNOWLEDGEMENTS**

The excellent technical assistance of Ms. L. Foss and Ms. H. Chao are acknowledged. The work could not have been undertaken without the excellent cooperation of the stockyards company, commission firms and packing house staff. These studies were supported by the Science and Education Administration, U.S. Department of Agriculture.
Figure 1

Movement of Swine in Three Market Areas

- Stockyards
- Feeder Pigs
- Slaughter Swine
- Buying Stations
- Slaughter Establishment
- Auction Markets
- Boars (Sows)

FARMS
- Minnesota
- Wisconsin
- Iowa
- South Dakota
Salmonella Isolation Protocol

Cecal contents

Enrichment (tetraphionate) 18-24 hours @ 37°C

Brilliant green agar 18-24 hours @ 37°C

Biochemical exam

MacConkey agar

Serology

Mesenteric decontaminated lymph nodes macerated
Table 1: Salmonella Isolations From Cecal Contents of Slaughtered Swine

<table>
<thead>
<tr>
<th>Investigators</th>
<th>Country</th>
<th>Year</th>
<th># positive</th>
<th># in Study(*)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Galton</td>
<td>USA</td>
<td>1954</td>
<td>46/71</td>
<td>(65.0%)</td>
</tr>
<tr>
<td>Williams</td>
<td>USA</td>
<td>1968</td>
<td>69/269</td>
<td>(25.7%)</td>
</tr>
<tr>
<td>Ghosh</td>
<td>UK</td>
<td>1972</td>
<td>34/586</td>
<td>(5.8%)</td>
</tr>
<tr>
<td>Lee</td>
<td>UK</td>
<td>1972</td>
<td>18/58</td>
<td>(31.0%)</td>
</tr>
<tr>
<td>Kampelmacher</td>
<td>Neth</td>
<td>1961</td>
<td>531/2100</td>
<td>(25.3%)</td>
</tr>
<tr>
<td>Guineé</td>
<td>Neth</td>
<td>1962</td>
<td>1091/8700</td>
<td>(12.5%)</td>
</tr>
<tr>
<td>Kampelmacher</td>
<td>Neth</td>
<td>1963</td>
<td>497/1566</td>
<td>(31.7%)</td>
</tr>
<tr>
<td>Chau</td>
<td>H.K.</td>
<td>1977</td>
<td>344/462</td>
<td>(74.5%)</td>
</tr>
<tr>
<td>Katsube</td>
<td>Japan</td>
<td>1973</td>
<td>18/40</td>
<td>(45.0%)</td>
</tr>
</tbody>
</table>

* % positive
<table>
<thead>
<tr>
<th>Investigator</th>
<th>Country</th>
<th>Year</th>
<th># positive</th>
<th># in Study(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Galton</td>
<td>USA</td>
<td>1954</td>
<td>27/374</td>
<td>(7.0%)</td>
</tr>
<tr>
<td>*Williams</td>
<td>USA</td>
<td>1968</td>
<td>1/203</td>
<td>(0.4%)</td>
</tr>
<tr>
<td>Gooch</td>
<td>USA</td>
<td>1969</td>
<td>68/370</td>
<td>(18.3%)</td>
</tr>
<tr>
<td>Newell</td>
<td>UK</td>
<td>1959</td>
<td>15/162</td>
<td>(9.0%)</td>
</tr>
<tr>
<td>Lee</td>
<td>UK</td>
<td>1972</td>
<td>11/136</td>
<td>(8.0%)</td>
</tr>
<tr>
<td>Ghosh</td>
<td>UK</td>
<td>1972</td>
<td>413/773</td>
<td>(53.0%)</td>
</tr>
<tr>
<td>Haddock</td>
<td>USA</td>
<td>1970</td>
<td>12/54</td>
<td>(22.2%)</td>
</tr>
<tr>
<td>Guinee</td>
<td>Neth</td>
<td>1965</td>
<td>172/858</td>
<td>(20.0%)</td>
</tr>
<tr>
<td>*Guinee</td>
<td>Neth</td>
<td>1965</td>
<td>49/390</td>
<td>(12.6%)</td>
</tr>
</tbody>
</table>

* pigs < 8 weeks of age

**% positive
### Table 3


<table>
<thead>
<tr>
<th>Source</th>
<th>Number Sampled</th>
<th>% of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>South St. Paul Stockyards</td>
<td>756</td>
<td>39.4</td>
</tr>
<tr>
<td>Minnesota*</td>
<td>612</td>
<td>31.9</td>
</tr>
<tr>
<td>Wisconsin*</td>
<td>96</td>
<td>5.0</td>
</tr>
<tr>
<td>Iowa*</td>
<td>310</td>
<td>16.1</td>
</tr>
<tr>
<td>So. Dakota*</td>
<td>146</td>
<td>7.6</td>
</tr>
<tr>
<td></td>
<td>1920</td>
<td>100.0</td>
</tr>
</tbody>
</table>

* These swine did not pass through the stockyards.
### Summary of Salmonella Swine Isolations by Age/Market Populations

<table>
<thead>
<tr>
<th>Class</th>
<th>Number of Positive Isolations</th>
<th>Number Sampled</th>
<th>Number of Samplings</th>
<th>Ave. daily Market or Slaughter</th>
<th>Yearly Averages (1981)</th>
<th>% Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feeder Pigs</td>
<td>2</td>
<td>809</td>
<td>13</td>
<td>1975</td>
<td>$10.2 \times 10^5$</td>
<td>0.25</td>
</tr>
<tr>
<td>Market Swine</td>
<td>494</td>
<td>1920</td>
<td>32</td>
<td>4200</td>
<td>$1.09 \times 10^6$</td>
<td>25.7</td>
</tr>
<tr>
<td>Boars</td>
<td>158</td>
<td>200</td>
<td>4</td>
<td>450-500</td>
<td>$2.5 \times 10^4$</td>
<td>79.0</td>
</tr>
</tbody>
</table>
Table 5: Salmonella Isolations by Site from 1920 Market Swine

<table>
<thead>
<tr>
<th>Site of Isolation</th>
<th>Total Isolations</th>
<th>% Pigs Positive</th>
<th>% of Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesenteric Lymph Nodes (MLN) Only</td>
<td>183</td>
<td>9.5</td>
<td>37.0</td>
</tr>
<tr>
<td>Cecal Contents (CC) Only</td>
<td>216</td>
<td>11.3</td>
<td>43.7</td>
</tr>
<tr>
<td>Both</td>
<td>95</td>
<td>4.9</td>
<td>19.3</td>
</tr>
<tr>
<td>Total pigs positive</td>
<td>494/1920</td>
<td>25.7</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Swine with more than one serotype isolated = 93/1920 (4.8%)
Salmonella positive pigs (MLN) = 278/1920 (14.5%)
Salmonella positive pigs (CC) = 311/1920 (16.2%)
Table 6

Five Most Common Salmonella Serotypes Isolated from Market Swine

<table>
<thead>
<tr>
<th>MLN*</th>
<th>CC**</th>
</tr>
</thead>
<tbody>
<tr>
<td>infantis</td>
<td>derby</td>
</tr>
<tr>
<td>derby</td>
<td>agona</td>
</tr>
<tr>
<td>agona</td>
<td>infantis</td>
</tr>
<tr>
<td>typhimurium</td>
<td>anatum</td>
</tr>
<tr>
<td>cholerae suis (va. kunzendorf)</td>
<td>st. paul</td>
</tr>
</tbody>
</table>

* MLN = Mesenteric Lymph Node  
** CC = Cecal Contents
**Salmonella Isolates in Market Swine**

St. Paul Stockyard vs Other Sources

**Mesenteric lymph nodes:**

<table>
<thead>
<tr>
<th></th>
<th>St. Paul Stockyard</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td>619</td>
<td>1023</td>
</tr>
<tr>
<td>Positive</td>
<td>141</td>
<td>136</td>
</tr>
</tbody>
</table>

\[X = 16.73 \quad p < 0.00001\]

**Cecal contents:**

<table>
<thead>
<tr>
<th></th>
<th>St. Paul Stockyard</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td>598</td>
<td>1009</td>
</tr>
<tr>
<td>Positive</td>
<td>162</td>
<td>150</td>
</tr>
</tbody>
</table>

\[X = 23.03 \quad p < 0.0001\]
Table 8: Origin of Market Swine by Day of the Week
St. Paul Stockyard vs All Other Sources

<table>
<thead>
<tr>
<th>Day of Week</th>
<th>St. Paul Stockyard</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Percent Positive</td>
</tr>
<tr>
<td>Monday</td>
<td>71/103</td>
<td>(68.9)</td>
</tr>
<tr>
<td>Tuesday</td>
<td>43/99</td>
<td>(43.4)</td>
</tr>
<tr>
<td>Wednesday</td>
<td>91/287</td>
<td>(31.7)</td>
</tr>
<tr>
<td>Thursday</td>
<td>42/187</td>
<td>(22.5)</td>
</tr>
<tr>
<td>Friday</td>
<td>5/80</td>
<td>(6.3)</td>
</tr>
<tr>
<td>Total</td>
<td>252/756</td>
<td>(33.3)</td>
</tr>
</tbody>
</table>
## Table 9: Source of Salmonella Positive Market Swine and Positives by Month at St. Paul Stockyard vs Other

<table>
<thead>
<tr>
<th>Month of Year</th>
<th># Positive</th>
<th># Sampled (%)</th>
<th># Positive</th>
<th># Sampled (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>St. Paul Stockyard</td>
<td>17/70 (24.3)</td>
<td>13/101 (12.9)</td>
<td>0/14</td>
<td>8/55 (14.5)</td>
</tr>
<tr>
<td>Other</td>
<td>17/170 (10)</td>
<td>5/79 (6.3)</td>
<td>40/226 (17.7)</td>
<td>37/240 (15.4)</td>
</tr>
</tbody>
</table>

**Total sampled during project** - 1920

**Total positive** - 494 (25.7%)
Table 10: Salmonella Isolations from Stockyard Swine Environment

<table>
<thead>
<tr>
<th>Frequency of Sampling (FS)</th>
<th>Location of Sample</th>
<th>Frequency of Isolation (FI)</th>
<th>FI/FS %</th>
<th>% Positive of the 35</th>
</tr>
</thead>
<tbody>
<tr>
<td>58</td>
<td>Feedbunk</td>
<td>10</td>
<td>17.2</td>
<td>28.6</td>
</tr>
<tr>
<td>49</td>
<td>Water fountain</td>
<td>7</td>
<td>14.3</td>
<td>20.0</td>
</tr>
<tr>
<td>126</td>
<td>Floor</td>
<td>7</td>
<td>5.6</td>
<td>20.0</td>
</tr>
<tr>
<td>16</td>
<td>Floor drain</td>
<td>2</td>
<td>12.5</td>
<td>5.7</td>
</tr>
<tr>
<td>15</td>
<td>Feed on Floor</td>
<td>2</td>
<td>13.3</td>
<td>5.7</td>
</tr>
<tr>
<td>38</td>
<td>Planks, ledges</td>
<td>3</td>
<td>7.9</td>
<td>8.6</td>
</tr>
<tr>
<td>33</td>
<td>Feces</td>
<td>3</td>
<td>9.1</td>
<td>8.6</td>
</tr>
<tr>
<td>1</td>
<td>Standing Water</td>
<td>1</td>
<td>—</td>
<td>2.9</td>
</tr>
<tr>
<td>4</td>
<td>Misc.</td>
<td>0</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>340</td>
<td></td>
<td>35</td>
<td>10.3%</td>
<td>100.0%</td>
</tr>
</tbody>
</table>
SALMONELLA IN VARIOUS AGE GROUPS

REFERENCES


Pomeroy, B. S. Personal Communications.


THE NATIONAL POULTRY IMPROVEMENT PLAN—AN UPDATE

R. D. Schar and I. L. Peterson*

The National Poultry Improvement Plan (NPIP) became effective in 1935 following a special appropriation from Congress. The Plan is a State-Federal cooperative program designed to improve poultry and poultry products. It is a voluntary program for both the State and the participating poultry member. At the present time, it is essentially a disease control program. The diseases, for the most part, that are controlled are egg transmitted and hatchery disseminated. The Plan controls diseases by identifying flocks, hatcheries, and dealers that meet certain disease control standards. The customer then has the opportunity of purchasing stock that are tested “clean” of certain diseases or that are produced under certain requirements certified to by the Official State Agency (OSA).

To properly understand the Plan and how it works, a brief review of its origin and history will be beneficial. In the early 1900's, the poultry industry was made up of small flocks kept essentially for egg production or multi-purpose chickens which could be utilized for meat as well as eggs. The chicks were obtained from a small local hatcheryman who had his own breeding flock of purchased hatching eggs from a local source.

The Extension Service and the local experiment station developed programs to improve breeding, management, and health. Differences in stocks became clearly evident as to health and egg numbers. In time, most of the States organized a poultry improvement board of council usually in cooperation with the State Poultry Association. A typical poultry improvement board consisted of at least one member from the poultry science department of the land grant college, the extension poultryman, a member from the State Department of Agriculture, a veterinarian from the diagnostic laboratory, and an elected member from each segment of the poultry hatchery and breeding industry.

Losses from bacillary white diarrhea or pullorum disease were high and widespread, and the state poultry improvement boards set up testing and control programs to prevent losses from this disease.

As transportation improved and as hatcheries shipped chicks great distances through the postal services, classification by breeding program and pullorum control had to be on a national basis. The International Baby Chick Association, which became the American Poultry and Egg Institute of America, was the prime industry organization pushing for a National Poultry Improvement Program or Plan. In 1935 when the Plan became effective, 19 States participated in the program by signing a

* Prepared by Mr. Raymond D. Schar and Dr. Irvin L. Peterson, Veterinary Services, APHIS, USDA, Hyattsville, Maryland, for the 1982 U.S. Animal Health Association Meeting, November 7-12, 1982, Nashville, Tennessee.
Memorandum of Understanding with the Department. The signatory on each Memorandum was the organization that had been carrying on the poultry improvement work in the years prior to 1935.

The first year of testing results showed nationally that 3.66 percent of the chickens were reactors to the pullorum test. Some individual States reported a reactor rate as high as 10 percent. Some prominent authorities were claiming that pullorum disease could not be controlled.

As the turkey industry developed, a similar need was evident and the National Turkey Improvement Plan was organized in 1944. The two Plans, having similar objectives, were combined into one National Poultry Improvement Plan in 1971 with separate subparts to allow for provisions unique to each species. We now have separate subparts or provisions for egg-type chickens, meat-type chickens, turkeys, and for waterfowl, exhibition poultry, and game birds.

Changes in the Plan are made by recommendations to the Secretary of Agriculture from voting delegates at the Biennial Plan Conference. Each State has one voting delegate for each of the subparts in which they have a participant with a maximum of four delegates. Each delegate votes only on those changes which affect his or her segment of the industry.

Proposals to change or amend the provisions of the Plan can come from any interested person. Proposals can be amended at the conference. Those receiving a majority vote are recommended to the Department. Following established procedures, they are published in the Code of Federal Regulations as amendments to the Plan.

One reason for the success of the Plan is that it is largely a self-help program carried out at the local level. The provisions are regulations adopted by the industry to improve their product. According to the latest estimate, the cost of the Plan programs is borne as follows:

- Federal — 2 percent
- State — 30 percent
- Industry — 68 percent

The Federal role is simply to coordinate or administer the program and provide the necessary diagnostic services and materials so that the States can effectively carry out the program.

The States carry out the day-to-day work of the Plan. The Official State Agency, through the NPIP Contact Representative, supervises the program. It provides for the diagnostic and testing services, inspection, and recordkeeping.

The participating industry members must be willing to cooperate and provide labor and the cost of blood testing and sanitation practices called for in the Plan provisions.

Any disease that is egg transmitted or hatchery disseminated and that has a practical diagnostic test for determining infection can be effectively controlled by the Plan. A customer for breeding stock, hatching eggs, or
baby poultry can purchase these products from a Plan participant with reasonable assurance as to their freedom from the diseases for which they are classified. No blood testing program is fool-proof, and a disease can be introduced into a flock after a test is made, but the total sanitation and monitoring program of the Plan provides the best assurance available that the product is indeed free of the disease.

This type of program has been very successful in the poultry industry. It permits interstate and international movement of hatching eggs and newly hatched poultry with minimal restrictions and paperwork, yet maximum assurance of freedom of disease.

We believe that the philosophy of this type of program could be applied to some other types of livestock or products.

A few charts (slides) will illustrate the progress and success of the programs of the Plan.

In regard to the pullorum and fowl typhoid diseases, the results are dramatic. These diseases are caused by *Salmonella pullorum* and *S. gallinarum* and are commonly referred to as pullorum-typhoid. There are several tests used for the detection of these diseases which are so closely related that a polyvalent stained K antigen is used for both diseases. The most common tests used in the pullorum-typhoid program are:

Rapid whole blood plate test—This test is especially useful for poultry flocks not participating in other programs and requiring a serum test. It can be performed by Authorized Testing Agents. Other tests are the tube test, serum plate test, and tests utilizing the microtiter system.

This chart summarizes the progress in the pullorum-typhoid program in the commercial chicken industry.

### Summary of Breeding Chickens Participating

<table>
<thead>
<tr>
<th>Year</th>
<th>Flocks (No.)</th>
<th>Birds Participating (Thousands)</th>
<th>Birds Tested (%)</th>
<th>Reactors (%)</th>
<th>Reactors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1936</td>
<td>9,191</td>
<td>4,329</td>
<td>100.0</td>
<td>158,516</td>
<td>3.66</td>
</tr>
<tr>
<td>1940</td>
<td>47,966</td>
<td>11,184</td>
<td>100.0</td>
<td>345,389</td>
<td>3.09</td>
</tr>
<tr>
<td>1950</td>
<td>111,422</td>
<td>37,237</td>
<td>100.0</td>
<td>269,115</td>
<td>.72</td>
</tr>
<tr>
<td>1960</td>
<td>37,857</td>
<td>37,030</td>
<td>100.0</td>
<td>6,812</td>
<td>.018</td>
</tr>
<tr>
<td>1970</td>
<td>8,340</td>
<td>35,890</td>
<td>48.9</td>
<td>162</td>
<td>.0009</td>
</tr>
<tr>
<td>1980</td>
<td>6,677</td>
<td>36,071</td>
<td>7.4</td>
<td>41</td>
<td>.0015</td>
</tr>
</tbody>
</table>

1. Note — increasing number of flocks until 1950 then a decreasing number of flocks yet maintaining same level of birds; i.e., larger and fewer flocks.
2. Note — decreasing reactor rate from 3.66 percent. Pullorum-typhoid essentially eliminated from commercial chicken industry by the late 1960's.

3. Note — change in program to provide for reduced testing.

The next chart illustrates the progress in the turkey industry.

Summary of Breeding Turkeys Participating

<table>
<thead>
<tr>
<th>Year</th>
<th>Flocks (No.)</th>
<th>Birds Participating (Thousands)</th>
<th>Birds Tested (%)</th>
<th>Reactors</th>
<th>Reactors (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1944</td>
<td>2,489</td>
<td>982</td>
<td>100.0</td>
<td>19,616</td>
<td>2.00</td>
</tr>
<tr>
<td>1950</td>
<td>4,717</td>
<td>2,340</td>
<td>100.0</td>
<td>9,172</td>
<td>.39</td>
</tr>
<tr>
<td>1960</td>
<td>2,614</td>
<td>2,510</td>
<td>100.0</td>
<td>243</td>
<td>.007</td>
</tr>
<tr>
<td>1970</td>
<td>1,062</td>
<td>3,145</td>
<td>75.2</td>
<td>6</td>
<td>.0003</td>
</tr>
<tr>
<td>1980</td>
<td>725</td>
<td>3,257</td>
<td>16.4</td>
<td>0</td>
<td>.0</td>
</tr>
</tbody>
</table>

1. Note — dramatic reduction of pullorum-typhoid from the turkey industry.

2. Note — changes in the industry; i.e., fewer and larger flocks.

1. Note — dramatic reduction of pullorum-typhoid from the turkey industry.

2. Note — changes in the industry; i.e., fewer and larger flocks.

This chart illustrates the increase in participation by the exhibition and backyard breeding flocks when emphasis was placed on bringing this type of poultry into the Plan.

NATIONAL POULTRY IMPROVEMENT PLAN

Waterfowl, Exhibition Poultry, and Game Bird Breeding Flocks

<table>
<thead>
<tr>
<th>Participating</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Birds</td>
<td>Flocks</td>
<td></td>
</tr>
<tr>
<td>1974-75</td>
<td>216,724</td>
<td>1,849</td>
</tr>
<tr>
<td>1976-77</td>
<td>274,880</td>
<td>2,247</td>
</tr>
<tr>
<td>1978-79</td>
<td>332,113</td>
<td>2,447</td>
</tr>
<tr>
<td>1980-81</td>
<td>409,177</td>
<td>2,769</td>
</tr>
</tbody>
</table>

A 7-year summary of pullorum-typhoid isolations indicates the number of infections detected in these flocks as they were tested under the Plan.
### 7-Year Summary of Pullorum-Typhoid Isolations

<table>
<thead>
<tr>
<th>Year</th>
<th>Pullorum-Typhoid Isolations</th>
<th>Isolations by Type of Flock</th>
<th>Gallinarum Isolations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Exhibition/Backyard</td>
<td>Small</td>
</tr>
<tr>
<td>1975</td>
<td>70</td>
<td>62</td>
<td>8</td>
</tr>
<tr>
<td>1976</td>
<td>63</td>
<td>59</td>
<td>-</td>
</tr>
<tr>
<td>1977</td>
<td>29</td>
<td>26</td>
<td>-</td>
</tr>
<tr>
<td>1978</td>
<td>67</td>
<td>47</td>
<td>10</td>
</tr>
<tr>
<td>1979</td>
<td>101</td>
<td>87</td>
<td>10</td>
</tr>
<tr>
<td>1980</td>
<td>138</td>
<td>115</td>
<td>16</td>
</tr>
<tr>
<td>1981</td>
<td>83</td>
<td>75</td>
<td>4</td>
</tr>
</tbody>
</table>

In the late 1960's, there was an effort by the USAHA and leaders in the Plan to make additional progress in eliminating the remaining foci of pullorum-typhoid and to reduce the work and cost of testing certain flocks. As a result, the classification “U.S. Pullorum-Typhoid Clean State” became available in 1974. This program provided for reduced or sample testing of primary breeding flocks and no testing of multiplier breeding flocks under certain conditions. The conditions necessary within a State in order to qualify for this classification are as follows:

**Conditions Within a State in Order to Qualify as U.S. Pullorum-Typhoid (P-T) Clean State**

1. All hatcheries under the Plan or equivalent State program.
2. All breeding flocks under the Plan or equivalent State program.
3. All imports P-T Clean or equivalent classification.
4. All P-T isolations reported to OSA within 48 hours.
5. All P-T isolations investigated by OSA.
6. All P-T infected flocks quarantined.
7. All publicly exhibited poultry, except waterfowl, tested for P-T.

As of 1982, 28 States have qualified as “U.S. Pullorum-Typhoid Clean State” as indicated on the following map.
We believe that this is the right approach to further progress in pullorum-typhoid control. We urge responsible poultry and animal health officials in the remaining 22 States to take the necessary steps to obtain the authority, support, or other action to qualify for this classification. Perhaps by 1990, we can designate these diseases as being foreign diseases to the United States.

Another success story is the control of *Mycoplasma gallisepticum* in the chicken meat or broiler industry and in the turkey industry.
The "U.S. M. Gallisepticum Clean" program became effective in turkeys, in 1966 and in meat-type chickens in 1968. Without the control of this disease in broilers and turkeys, these industries could not possibly exist as we know them today under present Federal inspection procedures.

This organism causes infectious sinusitis and airsacculitis in turkeys and airsac disease, chronic respiratory disease, or simply CRD in chickens. Infection by mycoplasma is detected by serological tests. The screening tests, a qualifying test before egg production followed by monitoring tests at regular levels, are either done by a serum plate test or a tube agglutination test. Additional laboratory work may be necessary to determine if a flock is infected. Suspicious flocks have been a continuing problem to the industry which often requires extra effort by a few scientist at universities, ARS laboratories, and the National Veterinary Services Laboratories.

The "U.S. M. Synoviae Clean" classification became effective in meat-type chickens or broilers in 1974. *M. synoviae* also causes airsacculitis in poultry as well as the disease infectious synovitis in chickens and turkeys.

Progress in *M. synoviae* control is illustrated by the following chart:

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Flocks Tested</td>
<td>237</td>
<td>1,063</td>
<td>1,219</td>
<td>1,254</td>
</tr>
<tr>
<td>Breeding Birds (Millions)</td>
<td>1.5</td>
<td>9.1</td>
<td>10.9</td>
<td>12.4</td>
</tr>
<tr>
<td>Positive Flocks</td>
<td>36</td>
<td>30</td>
<td>14</td>
<td>17</td>
</tr>
<tr>
<td>Percent Positive</td>
<td>15.2</td>
<td>2.8</td>
<td>1.1</td>
<td>1.3</td>
</tr>
</tbody>
</table>
To further illustrate the progress in the control of mycoplasma in the poultry meat industry, the following chart shows the percent of condemnation for airsacculitis in young chickens from 1962 to 1981.

You will note an increasing number of condemnations for airsacculitis until 1967. As the MG program became effective, there was a rapid drop in these condemnations. There was also a decrease in condemnation with the adoption of the MS program in 1974.

Most of the major chicken and turkey breeders have eliminated MG and MS from their primary breeding stock. A new classification "U.S. M. Gallisepticum Clean State Turkeys" will maintain or increase the emphasis necessary to make further progress in the control of MG.
North Carolina has recently initiated an aggressive and progressive program to reduce the incidence of MG in that State to protect their poultry industry from the dangers of this infection. Other States are considering similar practices.

Progress needs to be made in reducing the amount of MG in the commercial egg industry. Many multiple-age commercial egg farms are infected with MG and recent estimates have placed the annual economic loss due to this infection in this industry at over $97 million.

Recent attempts to develop an MG eradication scheme on these farms through the use of controlled exposure to the "F" strain of MG, use of MG bacterin, or both are encouraging. It is evident that the technology is now available to develop a scheme that will be successful in eradicating this organism from many of these multiple-age commercial egg farms.

Mycoplasma control programs for started pullets have recently been included in the Plan. This will provide certification by a disinterested Official State Agency that certain testing and other requirements have been met. These programs should assist the egg industry and the rest of the poultry industry in continuing to make progress in the control of mycoplasmosis.

Another mycoplasma program, "U.S. M. Meleagridis Clean," will become effective January 1, 1983. A good portion of the turkey industry has indicated a desire to participate in this program. This program should allow the industry to stop egg-dipping and to make substantial reduction in overall cost of production.

In conclusion, the Plan has been very successful in controlling several devastating diseases of the poultry industry. The control of pullorum-typhoid diseases and mycoplasmosis has been accomplished with minimal Federal effort. With control, the poultry industry has been able to grow into the dynamic industry that it is today and the consumer has profited enormously by enjoying a wholesome, high protein food at a reasonable price.

One problem in the future is to maintain support for the programs. Animal health officials must not become apathetic as we strive to make additional progress in reducing the incidence of these diseases.
SALMONELLA AND ARIZONA SEROTYPES FROM ANIMALS AND RELATED SOURCES REPORTED DURING FISCAL YEAR 1981

Billie O. Blackburn, DVM
Kathleen Sutch, BS
and
Rube Harrington, Jr., DVM, MPH
The Diagnostic Bacteriology Laboratory
National Veterinary Services Laboratories
Animal and Plant Health Inspection Service (APHIS)

SUMMARY

Serotyping of salmonella and arizona cultures from animal disease cases and epidemiologically related sources is reported for October 1, 1980 through September 30, 1981 (FY 1981). A total of 4924 cultures were serotyped. The most frequently identified salmonella serotypes were Salmonella typhimurium, S. cholerasuis var. Kunzendorf, S. typhimurium var. Copenhagen, S. dublin and S. heidelberg. The most frequently identified arizona serotype was 7a,7b:1,7,8. The most frequent sources of cultures in order of frequency were cattle, swine, turkeys, and horses.

INTRODUCTION

Data for this report were accumulated at the National Veterinary Services Laboratories, Animal and Plant Health Inspection Services, USDA, Ames, Iowa. Other laboratories contributing serotyping information were Paige Laboratory, University of Massachusetts, Amherst, Massachusetts, and the Animal Health Laboratories of the Wisconsin Department of Agriculture, Madison, and Barron, Wisconsin.

The data, except for serotyping results, were provided by the many laboratories requesting serotyping services. Most of these laboratories appreciate the importance of accurate data and made a concerted effort to provide quality input. Also, the reports were screened for obvious errors. However, it was not possible to verify each entry and the quality of the total report is a reflection of the cooperative spirit of these laboratories.

The purpose of this report is to make the data available to epidemiologists and others who have a need for it. The data are presented in tables as in previous reports in order that comparison can be easily made.

DISCUSSION

Salmonella and arizona cultures for serotyping were received from 48 states, the District of Columbia, Puerto Rico and Guam during FY 1981 (Tables 1 and 3). Several of the states were represented by a very small number of cultures. After one year with a significant number of reports, Michigan dropped back to one report.
The number of cultures serotyped, 4924, (Tables 2 and 4) was slightly more than for the previous year.

Sources of cultures submitted for serotyping shifted again in FY 81. Horses became more common than chickens as a source. Increases in cultures from horses have been observed for the last two years and cultures from chickens have reduced slightly.

The total number of serotypes identified was 140 (113 Salmonellae—Table 2 and 27 Arizonae—Table 4). The 11 most common serotypes (Table 10) accounted for 68% of the salmonella cultures. One serotype (7a,7b:1,7,8) accounted for 66% of the arizona cultures.

*Salmonella dublin* continued to appear more frequently (Table 10). It has gradually increased in rank for the last 6 years. Some of this increase may be due to an increase in cultures from cattle but some is also due to the spread of this serotype eastward as has been previously noted[11].

A great increase in *S. senftenberg* reports from turkeys, horses, and environmental samples boosted its rank from 14th to 8th. The data submitted with the cultures suggests that low morbidity and mortality was observed in affected flocks and herds (Tables 5 and 9). It may be significant that 94 of the cultures were from North Carolina (40) and Pennsylvania (54). This could mean that repeat isolations were involved to some degree in the increase.

On first look the increase in *S. muenster* from 25 in FY 80 to 101 in FY 81 is very striking. Most of the change was in cultures from cattle (11 to 77) and involves the states of Indiana and Pennsylvania. We know that a monitoring program was conducted in Indiana but the Pennsylvania cultures appeared to be from clinical cases. Reported morbidity in cattle was 18.4% and mortality was 2.7%.

We first identified *S. hadar* in FY 1979. During that year the serotype was reported once from Illinois and once from Minnesota. Both isolates were from turkeys. In FY 1980 it was reported from four additional states and two other animals with a total of 14 reports. The 63 reports this year indicate that this serotype, which is relatively new in the United States, has spread rapidly. It was the 5th most common serotype from turkeys (Table 5). However, no reports have been received from North Carolina and California which are large turkey producing states.

Another relatively new serotype from animals in the United States is *S. mbandaka*. Reports for the past five years have been 1, 0, 2, 12, and 39. The FY 1977 isolate was from a chicken in Illinois. This year the serotype was reported from 7 different animal species and feed with 12 states, Guam and Puerto Rico, involved. Although the details are far from complete, this pattern reminds one of what happened with *S. agona* several years ago[11].

While there appeared to be a significant increase in *S. typhisuis* reports, this was due mostly to special efforts to study some problems in
Massachusetts swine. None of the reports were from areas where *S. typhimurium* had not been previously reported.

*S. pullorum* continued to be the most common serotype from chickens. In fact, the gap between *S. pullorum* and the next most common serotype became greater. In addition to explanations given in the previous report, one should also consider the fact that infection with *S. pullorum* is a reportable disease in the United States while infections with most other serotypes are not required to be reported. Many of the reports of *S. pullorum* isolation resulted from laboratory investigations of serologically positive birds. Most of these were the result of a recently instituted mandatory testing program for exhibition poultry and "back yard" breeding flocks in one state.

Arizona 26:30 seems to have established itself in the sheep population. A sizeable number of reports have been received for several years. The data on this serotype are incomplete at this time, but most reports were associated with intestinal or reproductive problems.

Two serotypes were reported which apparently have not previously been reported from animals in the United States. *Salmonella lawndale* was reported from a mature bovine animal in Missouri. No data concerning the disease problem was provided. This serotype was originally reported in 1961 following isolation from the stool of a three-month-old female infant with acute gastroenteritis. The isolation was made at Cook County Hospital, Chicago, Illinois. *Salmonella orientalis* was reported from a mature canine animal on Guam. No data about the disease was provided with this isolate either. The original report of this serotype appeared in 1946. The isolation resulted from a survey of repatriated American prisoners of war from the Orient.
SALMONELLA AND ARIZONA SEROTYPES
ml

6

u

*

-

C
L

*

rn

e

f

m
I

b
0,

o

e

C
w

.

a

C

m

u
r

C

c

c

a
E

E

0

Y

0

z

-

N

e

L

I

.

SALMONELLA AND ARIZONA SEROTYPES

c

r

Y

459


360

c

a

.

L

c

~

-

~

~

~

m

~

~

~

~

-

~

~

m

o

~

~

~

o

-

n

o

o

BLACKBURN, SUTCH AND HARRINGTON

E
U dI

~ ~
c
.

x u

0”

r

-

o

~

o

~

x--ooooeooo~ooo000~0~-0r~000000000-00~0~oo~mon

Y A
9-

W

oa

o

o

o

~

o

o

~ o ~ - o o ~ o o o o ~ ~ o ~ o ~ ~ ~ o - o e o o o o ~ o o o o o o o ~ o o o o - ~ o

Y

m

a
m

w ~ O C C O O O O o O c 0 0 0 a - S e - e o e o - o o ~ - - e e o ~ e ~ ~ o o o ~ o o o o

Y

Y

r

*

1

-

v..I

-

~ o ~ ~ ~ ~ o o ~ o o ~ ~ c ~ ~ m ~ - - o - o o - ~ - o o o ~ ~ ~ o r r - - o o ~

C

Y

a

-00mo-0v-00-00-000~0o*ooo--enoorn9eoooo.=ooo**o

E P
Y.r001000*00000000-C-00000-0-0-0-01000--0000010

L
ea


<table>
<thead>
<tr>
<th>SEROTYPE</th>
<th>AZ</th>
<th>AR</th>
<th>CA</th>
<th>CO</th>
<th>ID</th>
<th>IL</th>
<th>IN</th>
<th>IA</th>
<th>KS</th>
<th>KY</th>
<th>MA</th>
<th>MD</th>
<th>MI</th>
<th>MN</th>
<th>MO</th>
<th>MT</th>
<th>NE</th>
<th>NC</th>
<th>OH</th>
<th>OK</th>
<th>OR</th>
<th>PA</th>
<th>SD</th>
<th>TX</th>
<th>UT</th>
<th>VA</th>
<th>WY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, 4,123-21</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,129-21</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,11,-2,5</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,11,-2,6</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,11,-2,7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,11,-2,8</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,11,-2,9</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,11,-2,10</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,11,-2,11</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4,11,-2,12</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 1: DISTRIBUTION OF ARIZONA SEROTYPES BY STATE - 1981**

**TOTALS**  2  3  14  39  6  1  1  1  1  2  1  7  4  6  6  4  5  1  11  20  16  7
<table>
<thead>
<tr>
<th>Table 4. Distribution of Arizona Serotypes by Source - FY 1982</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>Bacteriophage</td>
</tr>
<tr>
<td>Type</td>
</tr>
<tr>
<td>Type 1</td>
</tr>
<tr>
<td>Type 2</td>
</tr>
<tr>
<td>Type 3</td>
</tr>
<tr>
<td>Type 4</td>
</tr>
<tr>
<td>Type 5</td>
</tr>
<tr>
<td>Type 6</td>
</tr>
<tr>
<td>Type 7</td>
</tr>
<tr>
<td>Type 8</td>
</tr>
<tr>
<td>Type 9</td>
</tr>
<tr>
<td>Type A</td>
</tr>
<tr>
<td>Type B</td>
</tr>
<tr>
<td>Type C</td>
</tr>
<tr>
<td>Type D</td>
</tr>
<tr>
<td>Type E</td>
</tr>
<tr>
<td>Type F</td>
</tr>
<tr>
<td>Type G</td>
</tr>
<tr>
<td>Type H</td>
</tr>
<tr>
<td>Type I</td>
</tr>
<tr>
<td>Type J</td>
</tr>
<tr>
<td>Type K</td>
</tr>
<tr>
<td>Type L</td>
</tr>
<tr>
<td>Type M</td>
</tr>
<tr>
<td>Type N</td>
</tr>
<tr>
<td>Type O</td>
</tr>
<tr>
<td>Type P</td>
</tr>
<tr>
<td>Type Q</td>
</tr>
<tr>
<td>Type R</td>
</tr>
<tr>
<td>Type S</td>
</tr>
<tr>
<td>Type T</td>
</tr>
<tr>
<td>Type U</td>
</tr>
<tr>
<td>Type V</td>
</tr>
<tr>
<td>Type W</td>
</tr>
<tr>
<td>Type X</td>
</tr>
<tr>
<td>Type Y</td>
</tr>
<tr>
<td>Type Z</td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>SEROTYPE</td>
</tr>
<tr>
<td>------------------</td>
</tr>
<tr>
<td>HEIDELBERG</td>
</tr>
<tr>
<td>ARIZONA 7A,7H:1,7,8</td>
</tr>
<tr>
<td>SAINT-PAUL</td>
</tr>
<tr>
<td>SEVENTEENBERG</td>
</tr>
<tr>
<td>VANDAR</td>
</tr>
<tr>
<td>MONTEVIDE</td>
</tr>
<tr>
<td>AVATIM</td>
</tr>
<tr>
<td>AGONA</td>
</tr>
<tr>
<td>SANTA-DIEGO</td>
</tr>
<tr>
<td>HEADING</td>
</tr>
<tr>
<td>ALL OTHERS</td>
</tr>
<tr>
<td>SEROTYPE</td>
</tr>
<tr>
<td>-----------------------</td>
</tr>
<tr>
<td>PILLONIUM</td>
</tr>
<tr>
<td>INFANTIS</td>
</tr>
<tr>
<td>TYPHIMURIIUM</td>
</tr>
<tr>
<td>HEIDELBERG</td>
</tr>
<tr>
<td>TYPHIMURIIUM (COPENHAGEN)</td>
</tr>
<tr>
<td>AGONIA</td>
</tr>
<tr>
<td>MONTEVIEDO</td>
</tr>
<tr>
<td>WHANGAeka</td>
</tr>
<tr>
<td>CERRIO</td>
</tr>
<tr>
<td>BREDENAY</td>
</tr>
<tr>
<td>ALL OTHERS</td>
</tr>
<tr>
<td>SEROTYPE</td>
</tr>
<tr>
<td>--------------------------</td>
</tr>
<tr>
<td>Typhimurium</td>
</tr>
<tr>
<td>Typhimurium (Copenhagen)</td>
</tr>
<tr>
<td>Dublin</td>
</tr>
<tr>
<td>Waegstein</td>
</tr>
<tr>
<td>Anatum</td>
</tr>
<tr>
<td>Ménévido</td>
</tr>
<tr>
<td>Milwaukee</td>
</tr>
<tr>
<td>Havana</td>
</tr>
<tr>
<td>Avenuey</td>
</tr>
<tr>
<td>Muenchen</td>
</tr>
<tr>
<td><strong>All Others</strong></td>
</tr>
</tbody>
</table>

SALMONELLA AND ARIZONA SEROTYPES
<table>
<thead>
<tr>
<th>SEROTYPE</th>
<th>TIMES IDENTIFIED</th>
<th>AVE. HERD SIZE</th>
<th>PERCENT HIGHEST</th>
<th>MORRISITIATIVE AVERAGE</th>
<th>PERCENT HIGHEST</th>
<th>MORTALITY AVERAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHOLERA SUIIS (KINZENDORF)</td>
<td>590</td>
<td>370</td>
<td>100</td>
<td>13.4</td>
<td>100</td>
<td>5.0</td>
</tr>
<tr>
<td>TYPHIMURUM</td>
<td>77</td>
<td>364</td>
<td>100</td>
<td>10.5</td>
<td>100</td>
<td>5.4</td>
</tr>
<tr>
<td>TYPHIMURUM (COPENHAGEN)</td>
<td>37</td>
<td>278</td>
<td>100</td>
<td>14.0</td>
<td>70</td>
<td>5.5</td>
</tr>
<tr>
<td>DERRY</td>
<td>37</td>
<td>439</td>
<td>85</td>
<td>9.3</td>
<td>50</td>
<td>4.4</td>
</tr>
<tr>
<td>TYPHI-SUIS</td>
<td>25</td>
<td>151</td>
<td>20</td>
<td>9.0</td>
<td>20</td>
<td>5.7</td>
</tr>
<tr>
<td>AGONA</td>
<td>24</td>
<td>459</td>
<td>75</td>
<td>9.0</td>
<td>17</td>
<td>1.6</td>
</tr>
<tr>
<td>INFANTIS</td>
<td>23</td>
<td>197</td>
<td>90</td>
<td>14.2</td>
<td>17</td>
<td>6.5</td>
</tr>
<tr>
<td>ANATUM</td>
<td>19</td>
<td>320</td>
<td>100</td>
<td>13.9</td>
<td>50</td>
<td>4.9</td>
</tr>
<tr>
<td>ENTERITIDIS</td>
<td>18</td>
<td>993</td>
<td>100</td>
<td>36.6</td>
<td>13</td>
<td>7.7</td>
</tr>
<tr>
<td>SAINT-PAIN</td>
<td>18</td>
<td>271</td>
<td>23</td>
<td>14.3</td>
<td>8</td>
<td>2.7</td>
</tr>
<tr>
<td>ALL OTHERS</td>
<td>96</td>
<td>450</td>
<td>100</td>
<td>17.1</td>
<td>100</td>
<td>4.4</td>
</tr>
<tr>
<td>Serotype</td>
<td>Type 4</td>
<td>Type 5</td>
<td>Type 6</td>
<td>Type 7</td>
<td>Type 8</td>
<td>Type 9</td>
</tr>
<tr>
<td>-----------</td>
<td>--------</td>
<td>--------</td>
<td>--------</td>
<td>--------</td>
<td>--------</td>
<td>--------</td>
</tr>
<tr>
<td>S. Saint-Paul</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S. Matching</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S. Anatum</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S. Indiana</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S. Agona</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S. Newport</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S. Anatum   (Cloned)</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S. Agona    (Cloned)</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 4: Frequencies of Boilermaker Salmonella and Arizona Serotypes
Table 10. Salmonella Serotypes Identified Most Frequently During FY 1981 with Comparison Data for 5 Years (All Sources)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Typhimurium</td>
<td>844*</td>
<td>790 (1)</td>
<td>533 (1)</td>
<td>559 (1)</td>
<td>549 (1)</td>
<td>671 (1)</td>
</tr>
<tr>
<td>Choleraesuis (A)</td>
<td>611 (2)</td>
<td>706 (2)</td>
<td>431 (2)</td>
<td>302 (2)</td>
<td>289 (2)</td>
<td>332 (3)</td>
</tr>
<tr>
<td>Typhimurium (B)</td>
<td>579 (3)</td>
<td>380 (3)</td>
<td>267 (3)</td>
<td>166 (7)</td>
<td>259 (3)</td>
<td>434 (2)</td>
</tr>
<tr>
<td>Dublin</td>
<td>246 (4)</td>
<td>197 (6)</td>
<td>116 (7)</td>
<td>105 (8)</td>
<td>87 (11)</td>
<td>131 (12)</td>
</tr>
<tr>
<td>Heidelberg</td>
<td>214 (5)</td>
<td>321 (4)</td>
<td>242 (4)</td>
<td>234 (3)</td>
<td>188 (5)</td>
<td>220 (5)</td>
</tr>
<tr>
<td>Anatum</td>
<td>191 (6)</td>
<td>179 (7)</td>
<td>183 (5)</td>
<td>171 (6)</td>
<td>246 (4)</td>
<td>243 (4)</td>
</tr>
<tr>
<td>Agona</td>
<td>158 (7)</td>
<td>203 (5)</td>
<td>162 (6)</td>
<td>210 (4)</td>
<td>173 (7)</td>
<td>190 (7)</td>
</tr>
<tr>
<td>Senftenberg</td>
<td>145 (8)</td>
<td>52 (14)</td>
<td>58 (14)</td>
<td>25 (27)</td>
<td>40 (18)</td>
<td>55 (21)</td>
</tr>
<tr>
<td>Infantis</td>
<td>119 (9)</td>
<td>124 (8)</td>
<td>89 (11)</td>
<td>93 (10)</td>
<td>130 (8)</td>
<td>168 (8)</td>
</tr>
<tr>
<td>Montevideo</td>
<td>117 (10)</td>
<td>61 (13)</td>
<td>39 (17)</td>
<td>91 (11)</td>
<td>59 (16)</td>
<td>114 (15)</td>
</tr>
<tr>
<td>Saint Paul</td>
<td>117 (10)</td>
<td>119 (9)</td>
<td>94 (9)</td>
<td>189 (5)</td>
<td>176 (6)</td>
<td>158 (9)</td>
</tr>
</tbody>
</table>

(A) = var. Kunzendorf, (B) = var. Copenhagen

*Number of times the serotype was identified

**Rank beginning with the most common
REFERENCES


REPORT OF THE COMMITTEE ON SALMONELLOSIS

Chairman: B. S. Pomeroy, Minnesota
Vice Chairman: G. H. Snoeyenbos, Massachusetts

Charles W. Beard, GA; B. O. Blackburn, IA; M. S. Cover, MO; M. L. Crandall, MD; Robert D. Glock, AZ; Rube Harrington, IA; Frank A. Hayes, GA; R. L. Hogue, IN; C. W. Johnson, IL; W. L. Kadel, KY; E. T. Mallinson, MD; C. S. McCain, OK; E. L. Menning, VA; E. V. Morse, IN; John P. Newman, MI; Robert T. Nicholas, CA; I. L. Peterson, MD; H. G. Purchase, MD; R. A. Robinson, MN; Raymond Schar, MD; Keith Van Steenberg, MO; Carl R. Weston, NH.

Ex Officio: D. D. King, MD; W. H. Dubbert, DC; Alan H. Bentley, Canada.

The Committee met at 1:30 p.m., November 9, 1982. Fourteen members and seventeen guests attended.

Mr. Maurice Strigeler, Bastrop, TX represented the National Renderers Association. Representatives from U.S. Public Health Service, CDC, Atlanta, Georgia had been invited to present a report on "The Current Status of Salmonellosis in Humans in the U.S." but were unable to attend.

The Committee this past year had been divided into subcommittees and reports will be summarized in these minutes. Accomplishments since publication of "Recommendations for Production and Control of Salmonellosis" as the report of the U.S. Advisory Committee on Salmonella, January, 1978, was used as a reference point.

Six general reports were presented to the Committee:

   Extended fingerprinting of plasmid nucleic acid from a number of isolates suggested "that resistant plasmids may be extensively shared between animal and human bacteria, and that spread of multiresistant strains of Salmonella among animal and human beings, as observed in Great Britain, may have been undetected in the United States for lack of comparable surveillance."

2. Dr. B. O. Blackburn reviewed the current status of Animal Salmonellosis in the United States. A complete report will be published in the proceedings of the 86th Annual Meeting of the USAHA. It is noted here that there has been a steady increase in the incidence and geographic spread of Salmonella dublin and that it has become a national problem. Salmonella hadar, a relatively new serotype in the U.S., appears to have become established in some areas. It was of interest that equines were the fourth most frequent host from which Salmonella was isolated. He also reported that S. arizona 26:30 was
the most frequent isolate from sheep and was associated with diarrhea and/or abortion.

3. Dr. J. W. Nelson (University of Minnesota) reported on epidemiological studies of *S. dublin* in Minnesota, Iowa, Wisconsin and South Dakota involving primarily dairy calves. The 30 herds studied suffered a 60% morbidity rate and a 48% case fatality rate. Some infections originated in stock from New York which was moved through Indiana and subsequently moved through a series of transactions which resulted in dissemination to herds in the states involved. Many of the herd infections were noted by veterinary practitioners as initially exhibiting respiratory signs. He noted the possibility that some *S. dublin* isolates could easily be misidentified as either *S. enteritidis* or *S. rostock* because of similar major antigens. Infections in the few swine herds involved were marked by meningitis and arthritis.

4. Dr. E. V. Morse reported on epidemiological studies of *S. dublin* in 26 herds of cattle and one herd of swine in Indiana. Extensive examination of the isolates indicated that they represented a homogenous strain. A few abortions occurred in the affected cattle and some developed an acute transient mastitis. *Salmonella muenchen* was also found with considerable frequency in cattle.

5. Dr. B. S. Pomeroy traced the introduction of *S. hadar* in Minnesota through turkey hatching eggs imported from Canada. The original breeding stock in Canada had originated from the United Kingdom. It was first recognized in 1979 on a turkey breeding farm and has since been found in twelve turkey farms supplying two hatcheries. *Salmonella livingstone* also appears to be a newly established serotype in turkeys in Minnesota. Neither of these serotypes appear to be clinically significant in turkeys but have significant public health importance.

6. Dr. R. A. Robinson reported on the response to a Salmonella surveillance questionnaire sent to state veterinarians. The response indicated that there was generally a very low level of surveillance of animal feeds and animal products, undependable reporting of diagnosis to other agencies and overall a lack of resources to carry on an active investigation program.

SUBCOMMITTEE REPORTS

1. Diagnostics and Data Collection

   Dr. B. O. Blackburn reported for the Subcommittee that the Annual Report of the NVSL is not an incidence report as many Salmonella isolates made by veterinarians and some diagnostic laboratories are not serotyped and many clinical cases are not verified by isolation of Salmonella and go undiagnosed.

   The Subcommittee suggested that comments in a newsletter or similar vehicle would be useful to emphasize the reasons for
serotyping isolates and providing good epidemiological data. Also
the manual on Culture Methods for the Detection of Animal
Salmonellosis and Arizonosis, Iowa State University Press, is still
available and useful. The Subcommittee indicated the need for the
services of an epidemiologist(s) for investigation of Salmonella cases
and for analysis of data including economic impact.

The full Committee on Salmonellosis passed a resolution which
was submitted to the Resolutions Committee recommending that
such epidemiological capability be provided by Veterinary Services,
APHIS, USDA.

2. Regulatory Programs

   Dr. I. L. Peterson reported for the Subcommittee on regulatory
programs for production that VS-APHIS had supported feasibility
studies in meat chickens in Georgia and Massachusetts and of
turkeys in Minnesota as well as epidemiological studies of S. dublin
in Indiana and Minnesota and studies on survival of Salmonella in
natural and simulated natural conditions in a swine environment in
Indiana. The studies indicated that rearing chickens free of
Salmonella was not now economically feasible and that competitive
exclusion was promising as a means of reducing infection rates in
both chicken and turkey flocks. Use of specific vaccines in con-
junction with competitive exclusion was a very promising means of
control of Salmonella in turkeys and chickens.

   Dr. W. H. Dubbert reported for Subcommittee on post harvest
(processing) that Food Chemical News recently carried a report that
the Canadian Agriculture Minister, Eugene Whelan, criticized the
U.S. for having "done nothing to implement the recommendations of
a Salmonella Advisory Committee that made its report in 1978." Dr.
Dubbert commented that many of the recommendations in respect
to processing had been addressed and noted that processing
microbiological standards and processing operations had been
markedly upgraded within the past year. In this context, Dr. Green
reported on the monitoring program for cooked meats instituted in
the last year.

3. Industry

   A. Feeds and Feed Ingredients

   Dr. M. S. Cover reported for this Subcommittee and introduced
two invited speakers.

   Dr. W. C. Patterson summarized the research activities at the
Russell Research Center, Athens, Georgia to develop standards
to eliminate Salmonella from poultry feeds by pelleting, other
process modifications and treatments.

   Mr. Maurice Striegler, on behalf of the National Renderers
Association reviewed the position of the rendering industry and
indicated their interest in producing a quality product. He fur-
other indicated that the industry would accept a voluntary Good Manufacturing Program.

Dr. W. B. Bixler reviewed the current interest of FDA in developing and promoting Good Manufacturing Guidelines for voluntary use by industry(s).

The full Committee on Salmonellosis approved a resolution calling for the development and distribution of good manufacturing guidelines for renderers, blenders and feed manufacturers. The resolution was referred to the Resolutions Committee.

B. Poultry Breeders

Mr. C. R. Weston reported for the Subcommittee that after surveying the major broiler breeders and a few major broiler producers that major changes in management systems for broiler breeders and broiler production would be necessary to allow elimination of Salmonella and that such changes are not now feasible. Competitive exclusion should be carefully examined as a possible tool to reduce the number of infected birds going to slaughter.

4. Research

Dr. D. D. King—ARS-USDA summarized federal/state funding for Salmonella research in meat producing animals as of FY 81 and noted that support for research in cattle and swine was very low. Currently (FY 83) there is no financial support by APHIS or ARS-USDA for field investigations and extramural research on Salmonellosis in the production area. Considerable funding of research is being made in the post harvest (processing) area both in ARS and State Agricultural Experiment Stations. One project on Salmonellosis was funded by Special Grants Program/CSRS-USDA in FY 82.

The Committee on Salmonellosis recognizes that knowledge and techniques are lacking to allow reasonable control of Salmonella at the production level and that research is essential to develop control capability. Funding by USDA as well as State Agricultural Experiment Stations and Colleges of Veterinary Medicine is essential for such research.

5. Extension-Education

Dr. E. T. Mallinson reported for this Subcommittee and summarized a series of industry trials on the production performance of broilers following rigorous periodic clean-up and sanitation of broiler houses. Production performance was significantly improved and the incidence of Salmonella infection within flocks was reported to be decreased.

The Committee is cognizant of lack of representation from many
segments of animal production industries and looks forward to more comprehensive representation in the future.

The following Subcommittees were formed:


**INDUSTRY:**

**FEEDS AND FEED INGREDIENTS:** C. W. Johnson and M. S. Cover, Chairperson.

**POULTRY BREEDERS:** Robert Nicholas and Carl Weston, Chairperson.


**EXTENSION-EDUCATION:** E. T. Mallinson, Chairperson.
Coronaviruses are considered to be significant enteric pathogens in a number of animals species, but their importance in sheep has not been established. We have detected coronavirus particles in diarrhetic feces of lambs over several years. Experimental inoculation of lambs with cell-free filtrates of fecal and colonic specimens from a lamb naturally infected with a coronavirus produced diarrhea and colonic lesions. Extensive attempts to isolate the coronavirus in cell cultures were negative. We believe that coronavirus may be a cause of diarrhea in lambs but additional research will be necessary to confirm this hypothesis.

Adenoviruses have been isolated from sheep in many countries but their pathogenic significance has only been established in a few. In the United States the only previous report of adenovirus isolation was by Dr. Howard Lehmkuhl, National Animal Disease Center, Ames, Iowa who isolated two serologically distinct strains from lambs with pneumonia.

We have isolated two adenoviruses. Isolate 475N was from the nasal swab of a 3-week-old lamb with pneumonia. When inoculated into lambs it produced severe bronchiolitis and pneumonia. Large intranuclear inclusion bodies were in many swollen bronchiolar epithelial cells. The experimentally inoculated lambs exhibited severe dyspnea and pyrexia following inoculation. Preliminary serologic characterization suggests that this isolate is similar to one of the strains isolated by Dr. Lehmkuhl.

The second adenovirus, designated 47F, was isolated from the feces of a 2-week-old lamb with pneumonenteritis. When inoculated into experimental lambs it caused pyrexia but little respiratory distress. At necropsy, the lungs contained multiple small depressed, reddened areas. Microscopically, there was mild to moderate interstitial pneumonia. Serologically this virus appears similar to a second adenovirus strain isolated by Dr. Lehmkuhl.

Isolation of two strains of adenovirus in both Oregon and Iowa suggests a wide distribution but additional research, especially serologic studies, will be necessary to establish their prevalence and possible relationships with clinical diseases.

*Presented at the 86th Annual meeting of the United States Animal Health Association, November 9, 1982, in Nashville, Tennessee.

Oregon Agricultural Experiment Station, Corvallis, OR 97331. Technical Paper No. 6703.
REPORT OF THE COMMITTEE ON SHEEP AND GOATS

Chairman: Michele C. Howard, California

Vice Chairman: Jack Pitcher, Maryland

Members present: Michael Jochim, CO, John Mare, AZ, F. James Schoenfeld, UT, Howard Whitford, TX, Olin Timm, CA, Stan Allen, UT, Thomas Snodgrass, TX, Dean Smith, WA, Richard Hall, GA, Joe Huff, CO, Percy Turner, TX, James Fox, GA, Miles Bairey, IA, and John Niemi, SD

The Sheep and Goat Committee met at 1:30 on Monday, November 6, 1982. There were 17 committee members present and 23 guests; a total of 40 were present.

The committee met as requested by the president of USAHA to consider the business of the committee, and submit the following report:

Dr. John A. Schmitz reported on Coronavirus and Adenovirus infections in lambs, stating that Coronavirus plus two Adenoviruses are newly discovered pathogens in sheep. There is also a possible bovine virus pathogen in sheep. Studies to support this conclusion were explained in detail.

Bluetongue virus epidemiology in California was discussed by Dr. Bennie Osburn. Information now available substantiates that an animal may be a carrier of Bluetongue Virus, but not show any signs of the infection. Regarding vaccination, Dr. Osburn reported that a modified live vaccine is available throughout the U.S. This, however, offers very little or no cross protection. There are currently vaccines for serotypes 10, 11 & 17 available in California only.

Dr. Jack Pitcher, Md., reported to the committee that at this time APHIS is no longer funding the cooperative agreement with California, Colorado and Florida Universities for Bluetongue research, and there are no immediate plans by APHIS for future funds.

Another subject addressed by Dr. Pitcher was the status of Ivermectin. Merke, Sharpe & Dohn currently have two anthelmentics on the market—one for equine, approved now in Canada and Europe, and soon in the U.S. The other, a cattle anthelmintic, which will include provisions for use in sheep also—unfortunately will not be available for at least one year—maybe longer.

A review of the foot rot problem was given by Dr. Blaine McGowan, Ca. Apparently, the variety of serotypes makes it difficult to nearly impossible to produce a vaccine. Dr. McGowan feels that a new approach needs to be generated by pooling current knowledge. He felt that right now we are not any closer to a solution to the problem.

Dr. Bud Turner gave a report on the Scrapie program. Dr. Turner asked the group to support the proposed revised program as outlined by research and sheep industry people in August of 1982. He was also
hopeful that a diagnostic research test could be formulated on the living animal, because of the 5 years or more incubation period in some cases.

It was added that Canada feels very confident with their program, which is almost identical to this new revised proposal.

Dr. Lloyd Konyha described a recent scrapie outbreak in Oklahoma, and the source flock relationship of the discovered cases.

The use of Selenium rumen pellets in sheep was reported on by Dr. Bennie Osburn, and he also gave a brief report on Border Disease. Dr. Osburn felt that it was very important that the USAHA lend support to getting products approved by FDA and Vet Biologies, and to urge the USDA to take a look at the Border Disease problem to attempt to determine the cost to the sheep industry.

A comprehensive report on the IR-4 program was given by Dr. Robert Kupelian. A time frame was outlined when various drugs for minor species would be available.

Foreign animal diseases and import regulations were reviewed by Dr. Buck Sharman, who also told of various training programs being conducted by APHIS for veterinarians in foreign animal disease diagnosis. Included in these are programs for veterinarians in the military. Regarding importations, Dr. Sharman reiterated the number of times an animal is tested for Bluetongue, and the length of the various quarantine periods before an animal is admitted to the U.S. Another subject under discussion led by Dr. Sharman was the import and export of embryos for research purposes.

As a matter of interest and record, Dr. R. A. Robinson of the Committee on Salmonella reported that Salmonella has been found in feeder lambs in Minnesota.

Also, the committee directed the chairman to work with the Parasite Committee to get the use of Ivermectin as soon as possible.

The Committee passed resolutions on Bluetongue Research, a proposed Scrapie Program, a survey on Border Disease, further research on disease transmission via embryo transplants, and a resolution of support for APHIS in their stand to protect the domestic livestock industry and the Nation's Wildlife resources as their number one priority.
ELISA AND FLOCK PROFILING IN MANAGEMENT AND DISEASE CONTROL

Virginia-Maryland Regional College of Veterinary Medicine
University of Maryland, College Park, Maryland 20740

The concept of flock profiling is not completely new to the progressive poultry industry. A number of the larger companies currently utilize it as a regular management tool. Profiling is based upon the sequential, serologic testing of flocks throughout their lifetime to monitor their immune response to vaccination and/or to detect infections by the broad array of pathogenic agents present in their environment.

The sequential results provide an outline or continuous health profile of each flock and thereby provide a means to make management decisions concerning preventive medicine and disease control. Conventional flock profiling, however, is cumbersome and expensive. It requires conducting a wide variety of serological tests to obtain useful information about numerous disease entities. Therefore, because profiling is expensive and the different tests are unwieldy to perform, large scale surveillance is presently limited to only some of the more affluent operations.

With the advent of enzyme-linked immunosorbent assay (ELISA) the feasibility of mass profiling has begun to change. Until recently, enzyme-immunoassays were basically regarded as interesting research methods, and were not considered seriously for routine use in either clinical or diagnostic laboratories. Progress in this field has now advanced to a point where such technology has been implemented for use on a regular basis and eliminates the need for diverse test systems. Innovative and automated ELISA techniques have become commonplace, especially with human systems. Commercial ELISA kits are available for the diagnosis of many human infectious diseases as well as for the detection of drugs and hormones. Unfortunately, at present, there are few ELISA reagents available for use in veterinary medicine. Dependable, automated equipment which expedites the ELISA procedure, is available and it is expected that it will soon be found in most of the well equipped diagnostic laboratories.

The features which make ELISA a valuable technique for flock profiling are 1) the need for relatively small amounts of reagents and serum or blood, 2) the requirement of only a single conjugate, 3) its high sensitivity, and 4) the rapidity with which it can be performed, once the test is standardized. These inherent properties make ELISA economical, efficient and a practical method to collect data for health profiling.

Added benefits can be realized by analyzing a single serum dilution coupled with the use of a standard curve to predict the titer instead of

* Scientific Article No. A-333. Contribution No. 6405 of the Maryland Agricultural Experiment Station, College Park, Maryland.
using numerous serial dilutions. This cuts reagent costs, minimizes dilution errors and permits more samples to be handled. Further, eluates of whole blood, collected and dried on filter paper can be substituted for serum to obtain meaningful results. This simplifies sample collection and shipping, eliminates the need for test tubes and syringes and allows less trained individuals to take the samples. This lessens the need for many farm trips by a serviceperson and helps to prevent the transmission of diseases between farms. ELISA also lends itself to automation and computerization of the system facilitates handling large sets of data. A program we have devised will compile and store raw data transmitted directly from the ELISA reader. The computer will then analyze the raw data and the program can be directed to automatically correct test absorbance values by subtracting the negative serum control "noise" from each sample on the plate. It can do this for either serial titrations or a single serum dilution. The computer can also be directed to average replicates at any location in six plates and to convert corrected absorbance values to ELISA titer by solving a regression line equation. The titer of each test sample can be stored and then later retrieved for comparative purposes or to update a flock profile. This simplifies and expedites data processing and when needed, this information can be printed out in list form or graphically which lends itself to easy interpretation by laymen.

Our goal is to replace the present diverse serologic tests with ELISA and take advantage of a uniform, single assay. A battery of antigens on a single plate can be used to rapidly screen test serum against the numerous antigens. The results will then provide an accurate profile reflecting the flocks' exposure, planned or unplanned, to the pathogenic agents of interest.

The blending of ELISA and flock profiling should prove to be an excellent management and disease control tool. Many applications in solving veterinary and agricultural problems have been described for ELISA and about a dozen different avian viral or bacterial ELISA systems have already been reported.

Some of the problems with the ELISA that confront us concerns the interlaboratory standardization of assays and reproducibility of results. These exist because of differences in antigen preparation, type of equipment used, test conditions and conjugates utilized. Perhaps a way for interested laboratories to achieve comparable results without standard methodology would be to have central designated laboratories prepare standard antigen and antiserum with which each laboratory could adjust its system.

If more commercial test reagents were made available perhaps more laboratories would use the system. The present drawback is that some laboratories are not equipped to handle some of the physico-chemical manipulations required for reagent preparation.
NEWCASTLE AND OTHER DISEASES OF IMPORTANCE

No cases of exotic Newcastle disease were reported in chickens or turkeys in the U.S. in fiscal year (FY) 1982 (FY 82 Oct 1, 1981 to September 30, 1982). However, exotic Newcastle disease was diagnosed on 13 premises involving cage birds. The total cost of these outbreaks during FY '82 was $196,151. Four of these infections were of major concern and required investigations, depopulations, and cleaning and disinfecting. Two infected premises resulted from a shipment of unbanded double Yellow-headed Amazons to Florida which were reported to have originated in California and transhipped from Arizona. Two other infected premises involved a shipment of psittacine birds reported to have been recently smuggled and shipped from California to Colorado.

The other nine infections resulted from specimens submitted to diagnostic laboratories. The diagnostic laboratories involved were: San Diego County Laboratory—four; California State Laboratory, San Bernardino—three; and Texas A&M University Laboratory, College Station—two.

Two isolates of VVND and one VND were obtained from personally-owned pet birds in USDA pet bird quarantine facilities. Seven isolates of VVND and one isolate of VND were obtained from Amazon parrots confiscated by U.S. Customs near the San Ysidro border crossing.

Dr. S. S. Richeson of the import/export staff of the USDA discussed the activities of their staff relative to USDA approved or removal of approval of import commercial bird quarantine facilities, changes in the 90 day ban, psittacosis preventive treatment, banding of imported psittacine birds, the smuggled bird program and importation of poultry and hatching eggs. A summary of the number of lots and total number of birds received and quarantined during FY82 in commercial bird and USDA facilities is presented in Table 1.
The National Veterinary Services Laboratories (NVSL), Ames, Iowa, examined 256 lots and 39,229 specimens from quarantine facilities for exotic Newcastle disease. Hemagglutinating (HA) viruses (590 isolates) were obtained from 55 lots of birds in quarantine facilities. Of the HA viruses other than Newcastle disease, 37.8 percent were paramyxovirus type 2 and 62.2 percent were paramyxovirus type 3. No isolates of influenza were obtained in FY82.

In view of the large numbers of paramyxoviruses other than Newcastle isolated from these specimens, the committee felt that more emphasis should be placed on these agents. Therefore, a subcommittee to study the significance of paramyxoviruses in avian species was formed.

NVSL examined 369 cases and 1,049 specimens for both VVND and psittacosis. Of these, there were 53 cases and 58 specimens diagnosed as positive for *Chlamydia psittaci* or 14.4 and 5.5 percent, respectively. Two positive cases of *C. psittaci* were diagnosed in turkeys by a California diagnostic laboratory.

There were 31 cases of pullorum and no case of fowl typhoid diagnosed by NVSL in FY82. The pullorum isolations were submitted from 11 different States. Three States submitted over 60 percent of the isolations. Except for two cases, the isolations were reported from mature chickens. The flock size ranged as follows: 1 from 1,482 birds; 10 from 100-500 birds; and the balance or 20 from flocks of less than 100 birds.

AVIAN INFLUENZA

Since the report of the Subcommittee at the 1981 USAHA meeting (Oct. 12, 1981) the diagnosis of avian influenza in turkeys has been made in five states either by isolation of the virus or serology (Iowa, Minnesota, Missouri, North Carolina and Texas). One new state (North Carolina) was added to the list of states reporting outbreaks of avian influenza since 1964. No outbreaks were reported in chickens. The avian serotypes identified in chickens, turkeys and other domestic fowl in the U.S. since 1964 are shown in Table 2.

California

Reported no outbreaks since October 1, 1981.

Colorado

Reported no outbreaks in 1981-82—However, the first recorded outbreak of Avian Influenza was in 1972 rather than 1981 according to a report from Southeast Poultry Research Laboratory.

Iowa

The NVSL-APHIS-VS reported the isolation of H1N1 from a turkey flock.
Minnesota

Minnesota reported outbreaks in the Fall of 1981 involving 50 flocks, 31 flocks infected with H5N2, 18 flocks with H6H8 and 1 flock with H10N7.

One company experienced outbreaks in two areas, 9 flocks on two farms in one area involving H6N8 serotype. Approximately 100,000 market birds were involved ranging in age from 4 weeks to 19 weeks. The estimated dollar loss was $46,726 from increased mortality and condemnations. The average mortality was 1.45% and range was 0% to 4.0% and average condemnation was 2.3% and range was 0.6% to 4.4%. The outbreak in the second area involved H5N2 and occurred on a multiple age complex. Ten flocks became infected with serotype H5N2 which indicated there was no connection between the two outbreaks.

Approximately 128,000 birds were involved ranging in age from 3 weeks to 17 weeks. The average mortality was 3.13%, range 0.7% to 9.4% and average condemnation was 4.2%, range 1.1% to 9.7%. The estimated dollar loss was $43,000. The farm has 6 brooder buildings and 12 grower buildings. The brooder buildings were depopulated, cleaned and disinfected before restocking, but the grower buildings were not depopulated before new broods were started in the brooder buildings. Movement of personnel and equipment was controlled in the brooder buildings and no new outbreaks occurred. The infection was ultimately eliminated from the farm as infected flocks were systematically marketed. Infected flocks were on the farm from 12/20/81 to 5/28/82.

In another area of the State in May, 1982 outbreaks occurred in turkey flocks belonging to five different companies. Both breeder flocks and market birds were involved. Serotypes H4N2 were involved in four of the outbreaks and the spread from the original site was probably related to dead bird pick-up by rendering truck and personnel that visited farms that became involved in the outbreaks.

Lateral spread occurred after the original outbreaks. One farm operation had ten flocks involved with 227,000 turkeys. The age of the flocks involved was from 10 days to 18 weeks. The average mortality was 9.1% and losses varied from 25% in the youngest group to no loss in older groups. The estimated financial loss was $106,000. The outbreak on the fifth farm involved a breeder flock with H1N1 serotype. Sick swine were present on the farm. No new outbreaks have been reported since July.

Missouri

Outbreaks were noted in breeders and market flocks in August and September, 1981 and April and May, 1982. H1N1 serotype was involved. Five breeder flocks and three market flocks were identified.

North Carolina

North Carolina reported the first outbreak of avian influenza in turkeys involving serotype H1N1. Six breeder flocks and one market flock were identified involving 43,000 birds.
Texas

Texas reported 11 turkey flocks infected involving approximately 54,000 birds. The serotype encountered was H5 and estimated loss was $215,000.

Influenza killed vaccine was used in California, Colorado, Iowa, Minnesota, Nebraska, North Carolina and Texas. Over 1.5 million doses were used with Minnesota the largest user.

A report and recommendations of the 1st International Symposium on Avian Influenza which was held in Beltsville, Maryland, January 21-23, 1981 was presented before the International Office of Epizootics (O.I.E.) Conference which convened in Paris, France in May 1982. Dr. John Lancaster, Canada, Chairman of the Special Commission on Poultry, reported that the following definition of Fowl Plague by this body was adopted.

Fowl Plague is a "clinical disease of chickens and turkeys caused by influenza 'A' virus regardless of antigenic type and which results in a serious mortality within a period of 10 days." A questionnaire on avian disease situation had been distributed by the Commission to the member countries of O.I.E. A summary of the replies was prepared by John Lancaster and Guy Meulemans and will be published and distributed to member countries.

Isolation of avian influenza "A" viruses or detection of antibodies against these viruses were reported from 11 countries. All isolates were of low pathogenicity. At the present time (May, 1982) there is no evidence of Fowl Plague in any part of the world.

MYCOPLASMOSIS

During the past year the Subcommittee has been actively involved with other associations and in national and regional symposia concerned with advancing the control of avian mycoplasmosis. The committee wishes to bring the following major concerns to the attention of the Association and to the leadership of USDA.

1. Surveys by Dr. Daryl Johnson of the American Association of Avian Pathologists (AAAP) have carefully estimated that the current prevalence of avian mycoplasmosis in commercial table egg layers costs the industry approximately $97,160,000 annually. (Table 3)

2. National Poultry Improvement Plan (NPIP) reports indicate that although 99% of Northeastern primary egg-and-meat-type breeder flocks are officially classified as "U.S. NPIP M. Gallisepticum Clean," only 19% of Northeastern multiplier breeding flocks are so classified. A similar situation exists nationally, and a somewhat worse situation exists with respect to participation in official classification programs for M. synoviae. (E. malli Proceedings 1982 Northeastern Conference on Avian Disease).

3. Continued difficulty exists in obtaining serologic or cultural con-
firmation of varient *M. gallisepticum* infection in suspect breeding flocks, and emergence of serious discrepancies between U.S. and foreign produced *M. synoviae* serum plate test antigens. (Scientific Advisory Committee, NPIP — August, 1982).

The committee lauds the high priority Veterinary Services, USDA has given to avian mycoplasma diagnostic referral services at the National Veterinary Services Laboratory (NVSL), and urges increased support as future demand indicates. Methods to obtain this referral service should be simplified and publicized to expedite state/industry utilization of the outstanding services available at NVSL. Research towards better diagnostic procedures, perhaps utilizing ELISA and egg yolk diagnostic systems is urgently needed.

The committee also has high praise for Veterinary Services, USDA involvement in epidemiologic investigations of avian mycoplasma outbreaks, and for involvement with industry in studies on the control and eradication of avian mycoplasmosis on multiple-age layer farms utilizing live vaccines and other modalities. These studies should also explore the merits and cost/benefits of newly licensed killed vaccines for the same purpose. This committee highly recommends that,

1. Veterinary Services explore the possibilities of a certification system and/or check-tests of laboratories performing serologic tests of flocks qualifying for official U.S. Mycoplasma Clean classifications.

2. Immediate investigation by USDA/APHIS VS into the causes of documented differences in specificity between mycoplasma antigens produced by NVSL, Salsbury Laboratories and Intervet Noblis, and initiation of protocol modifications to ensure optimal reliability of all mycoplasma test antigens.

3. Continuation of funding levels for avian Mycoplasma diagnostic referral and Mycoplasma typing services at NVSL, and expand funding to support similar work in Georgia and California in support of ongoing research activity into improved diagnostic procedures.

4. Exploration by USDA/APHIS VS NPIP into ways to accelerate participation of most multiplier breeders in official U.S. MG and MS clean classification programs.

5. Greater support of epidemiologic investigation of variant and standard MG, MS and MM breaks in breeder flocks, and techniques to clean-up large infected table egg complexes utilizing live and killed vaccines and other modalities.

CERTIFIED VVND NEGATIVE FLOCKS

This Subcommittee on the certification of primary chicken breeder flocks met on several occasions. A final subcommittee meeting was held on Monday, November 8, 1982 at the USAHA meeting in Nashville.
A. Introduction

The main objective at this meeting was to establish the essentials for a program. They are:

1. Criteria for determining a negative status.
2. Control of eggs from production through hatching.
3. Permits to move hatching eggs will be on a hatch-by-hatch basis.
4. Program presently limited to grandparent and earlier generations.
5. Requirements of the model program must be acceptable to Resident State, APHIS, the Receiving State, and Industry.

B. Essential Considerations for A Model Program

1. Flocks in a quarantine zone pose a risk of spreading VVND via hatching eggs, thereby requiring special measures for preventing any spread. Determination of degree of risk will be made by Veterinary Services, APHIS, USDA, which will also determine the required procedures to be applied.

2. A pre-quarantine program was not considered essential, but was recognized as a means of minimizing the time period required for flock clearance, and is recommended.

3. A premises is defined as a farm (ranch) that is maintained by a single set of services and personnel.

4. Provision should be made to prevent unrestricted movement of people and services. This may be in the form of fences, locked gates, water, and other natural barriers.

5. Breeders must be monitored by:
   a. Examination of hatchability, eggshell quality, production records, and graphs by house.
   b. Easily identified sentinels fully susceptible to Newcastle, monitored by signs, mortality, seroconversion and vent swabbing.

   1) Number of Sentinels Required
      a) Up to 300 birds per house — 10%
      b) Up to 1000 birds per house — 10%, with a maximum of 30
      c) Above 1000 birds per house — Add 1% of the excess above 1000 to the 30 birds up to a maximum of 100.

      EXAMPLE: 3600/house
      \[30 + 1\% \times 2600 = 30 + 26 = 56\] total

   2) Sentinel Distribution
      a) Sentinels must be equally distributed among all pens in each house.
      b) There must be at least one sentinel per pen.
      c) Sentinels may be co-mingled or caged. However,
sentinels must have access to common waterers and feces of the breeder flock.

NOTE: A VVND sentinel is a chicken that has not previously vaccinated (killed or live vaccine) or infected with, and is fully susceptible to Newcastle Disease virus, and has no evidence of sero-conversion.

c. Negative HI status of sentinels will be demonstrated at least every 30 days and a minimum of 5 days after the last day of each egg collection period (Hatch).

d. Necropsy and vent swab all morbid and dead birds. Vent swabs from all birds will be virologically examined promptly (within 10-14 days from submission of results).

6. All eggs will be fumigated immediately prior to movement from the egg production premises.

7. Hatching eggs shipped out of a quarantine zone must be shipped to an approved hatchery outside the quarantine zone, and must have prior approval of animal disease control authorities of the receiving state.

8. If a positive source flock is detected, all eggs and chicks within a receiving hatchery must be destroyed on the premises and the premises cleaned and disinfected under Veterinary Services supervision.

9. Alternative procedures for movement of primary breeder hatching eggs out of a VVND quarantine zone may be allowed by the National Emergency Programs Field Officer (NEFO), based on epidemiological evidence.

10. Eggs approved for shipment out of a VVND quarantine zone may be moved to a port of embarkation on approval of appropriate Animal Health Officials.

CAGE AND AVIARY BIRDS

The Sub-committee on Cage and Aviary Bird problems, continued with last year's committee actions to foster the formation of a VVND Symposium Steering Committee in pursuit of a dialogue between the poultry and pet bird industrymen, regulatory veterinarians, and researchers concerning possible resolution of problems in VVND prevention, control and eradication. A tentative agenda has been formulated. The time and place remains to be selected. The current need for wider VVND surveillance of severely diseased pet birds by veterinary practitioners and possible associated practitioner/client legal complications is an example of some of the issues that need to be aired.

The Sub-committee on the Model State Program for Pet Birds met many times during 1982, including meetings with 80 delegates of the National Poultry Improvement Plan (NPIP), in an effort to establish a
program which would bring a modicum of order to the present chaotic state of disparate laws and regulations, existent and non-existent, throughout our 50 states, whereby attempts are made to manage the different facets of the cage bird and its allied industries. Such a measure would benefit both the cage/aviary bird and poultry industries, and the food supply of the nation.

As a result the National Cage and Aviary Bird Improvement Plan (NCABIP) has been proposed. The NCABIP provides the first of a series of voluntary cooperative steps whereby breeders, producers, and distributors of cage and aviary birds can significantly improve the overall health and well-being of their birds and provide protection against the major direct and indirect ravages of viscerotropic velogenic Newcastle disease (VVND), avian influenza, (fowl plague), chlamydiosis, Pacheco's disease, pox, and other avian diseases.

The NCABIP will:

1. Curtail the movement of illegally imported birds, the principle source of VVND, by broad involvement of all sectors of the cage and aviary bird industry in a recognizable, approved, self-regulated, state-federally supervised program of health security, record keeping, and bird identification;

2. Encourage uniform cage and aviary bird regulations among states and federal government; and

3. Facilitate interstate movement of cage and aviary birds participating in the NCABIP. The program is modeled after the National Poultry Improvement Plan. The cage and aviary bird industry recognized the desirability of their involvement in and adoption of a similar plan whereby it too may ensure and encourage the establishment of standards and procedures that are approved by industry, government, and the scientific community.

PROPOSED PROGRAM

I. Authority, Objectives, Organization

A. Authority. Possibly under a portion of the Department of Agriculture Organic Act of 1944, as amended (7 U.S.C. 429), but different from those authorizing the NPIP. Utilize currently "reserved" Part 146, Subchapter F (Poultry Improvement), Chapter I APHIS, VS USDA Code. This part was formerly used for the National Turkey Improvement Plan.

B. Objective. Provide a cooperative State-Federal program through which new technology can be effectively applied to the improvement of cage and aviary birds throughout the country. The provisions of the program, developed jointly by industry members and State and Federal officials, would establish standards for the evaluation of cage and aviary birds with respect to sanitation and control of certain contagious and infectious diseases of birds and poultry. Participants and
birds' conforming to NCABIP standards would be identified by authorized terms that would be uniformly applicable in all parts of the country.

C. Amendment. The provisions of the proposed NCABIP would be changed from time to time to conform with the development of the industry and to utilize new information as it becomes available. These changes would be based upon recommendations made at a national program conference by official delegates representing participating aviculturists, importers, jobbers, and retailers from all cooperating states. Refer to Section VIII.

D. Voluntary. Acceptance of the NCA BIP would be optional with the States and individual members of the industry within the States. The program would be administered in each State by an Official State Agency cooperating with USDA.

II. Basic Program Considerations

A. Sanitation and Health Management

1. Housing shall provide:
   a. Protection against extremes and sudden changes in temperature.
   b. Effective sanitation.
   c. Security against pests and predators.
   d. Containment to prevent escape.

2. Caging shall provide:
   a. Construction characteristics commensurate with efficient, effective husbandry and sanitation.
   b. Minimal opportunity for the collection of droppings, spent feed, and other debris or waste products.

3. Food and water containers shall be kept clean. Soaking in household bleach or equivalent recommended weekly.

4. Good housekeeping practices.

5. Isolation of new additions.

6. Bird density. Enclosure size should be adequate for the species and conditions of confinement. Perches should be sufficient in number to further enhance exercise and to avoid over-crowding.

7. Pest control. Housing must be rodent proof and active control measures maintained.

8. Proper feed storage. Feed shall be kept in enclosed containers or in unopened bags of suitable materials.

B. Accurate record keeping shall be maintained on all birds brought to or sold from the premises. Records shall be maintained for a predetermined period enabling an adequate audit trail of all
purchases, sales, other movements, and losses. This is a major
deterrent to smuggling, prevention of VVND, and of paramount
importance in rapid control of recognized VVND outbreaks. Ef-
ficient, uncomplicated forms, similar to those used by NPIP, should
be used.

C. All birds shall be identified by an approved band or other approved
means. Closed banding is preferred over open banding for domes-
tically hatched birds.

III. Recognition Terminology and Participants Directory

A. "U.S. Registered" classification awarded to those facilities and
individuals in compliance with outline Sections II-A, B and C above.

B. "U.S. Hatched" a possible additional program classification
awarded to those in compliance with program mentioned in Section
IX-A below.

C. Registries/Directories listing importers, aviculturists, jobbers, and
retailers in compliance with various classifications would be
published and distributed to all interested persons and businesses.

IV. Classified and Unclassified Stock. An educational and grace period of
specified length would be provided to enable aviculturists and industry
to "gear up" to eventually meet requirements of each participant to deal
only in NCABIP classified stock or their official state equivalent.

V. Suspension, revocation and/or denial from participation shall be so
structured as to ensure fair, prompt disposition of reported violations.
This is essential for the credibility of the Program and the protection of
the public and poultry, cage and aviary bird industries.

VI. Supervision, Training and Inspection

A. National level. Expanded National Plans Office—(Beltsville,
Maryland)

B. State level. Ordinarily State Departments of Agriculture are
preferred. However, as with NPIP in some states, inspection ar-
angement with other state agencies and industry organizations
would be possible.

C. Training shall be required for inspectors and administrators
concerning cage and aviary bird inspection, management, and
disease control. This is a vital program consideration which is
absolutely essential for establishing rapport, creating credibility,
and gaining willing cooperation.

VII. Separate funding. National (4%) and the balance (96%) between
States and Plan Participants.

VIII. Amendment of the NCABIP. Delegates and Advisory (Steering or
Executive) Committees. Delegates should represent State or Regional
Bird Improvement Boards. Presentation of amendments and their con-
sideration should be patterned after NPIP. National Conferences should
be back-to-back with those of NPIP to ensure close communication between NPIP and NCABIP advisors and delegates, and provide for efficient use of travel funds by persons involved in both plans.

IX. Supplemental Program Possibilities

A. Recognition of domestically hatched stock.
B. VVND control
C. Chlamydia control.
D. Avian influenza, Pacheco's disease, mycoplasmosis, pox and other avian diseases.

OTHER TOPICS

Chlamydiosis

A report was presented from an Avian Chlamydiosis Committee that was appointed in 1981 as a joint committee of the American Association of Avian Pathologists and the Association of Avian Veterinarians. Dr. L. C. Grumbles is chairman and reported that a major topic for consideration is the treatment of imported birds. The committee is considering recommending that the treatment period be extended from the present 30 days to 45 days. Dr. Susan L. Clubb explained that she does not support the 45 days treatment period because of a lack of research supporting the fact that 45 days is better than 30 days. She explained that such an extension would add significantly to the cost of importing pet and exotic birds.

Serious Errors in Disease Reporting

Dr. I. L. Peterson reported on the importance of accurate reporting. Diagnosticians and personnel involved in reporting poultry disease diagnosis which will be summarized and published in journals such as Avian Diseases should make every effort to insure the accuracy of the final data before it is printed. This is especially true for certain reportable or program diseases. Although this has been a recurring problem for many years, it was especially in error in the last annual summary published in Avian Diseases. In this summary, six states were reported to have had a total of 89 isolations, or cases of Salmonella gallinarum, the causative organism of fowl typhoid during 1980. Upon investigation, there was only one state with two cases of fowl typhoid. Although the errors were probably made by a clerk or typist in recording or transmitting the data, the damage is done when the journal is published to be read and reported as fact for years to come. Accuracy is essential if this disease reporting effort is to be meaningful and useful.

The following subcommittees were formed:

AVIAN INFLUENZA: R. A. Bankowski, C. Beard, D. King, J. Newman, J. E. Pearson, I. Peterson and B. S. Pomeroy, Chairperson

MYCOPLASMOSIS: W. Dugan, K. Hand, D. Johnson, H. O. Opitz, B. S.
Pomeroy, I. Peterson, R. E. Pitts, R. Yamamoto and E. T. Mallinson, Chairperson


CAGE AND AVIARY BIRDS: S. Clubb, R. Cooper, C. G. Harrison, E. Mallinson, J. Pearson, A. C. Rissen, Jr., and R. E. Baer, Chairperson

NEWCASTLE DISEASE REEVALUATION: R. A. Bankowski, M. Brugh, R. P. Hanson, J. E. Pearson, and L. Grumbles, Chairperson

MODEL STATE PROGRAM FOR PET BIRDS: T. Angel, R. E. Baer, H. Goldstein, R. Hanessian, D. J. Ligda, E. T. Mallinson, M. Myers, R. Schar, J. L. Williams, and H. Kahan, Chairperson

PARAMYXOVIRUS EVALUATION: Bahl, M. Brugh, D. King, J. Newman, J. E. Pearson, and R. A. Bankowski, Chairperson
<table>
<thead>
<tr>
<th>Number of Quarantines</th>
<th>Total Received</th>
<th>VND Refused Entry</th>
<th>Other Refused Entry</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Birds</td>
<td>DOA</td>
<td>Died in Quarantine</td>
</tr>
<tr>
<td>Private Commercial Bird Stations</td>
<td>255</td>
<td>771798</td>
<td>55676</td>
</tr>
<tr>
<td>USDA Facilities</td>
<td>149</td>
<td>6282</td>
<td>81</td>
</tr>
<tr>
<td>Total Imports</td>
<td>404</td>
<td>778080</td>
<td>55757</td>
</tr>
</tbody>
</table>

*Velogenic Newcastle Disease (VND)*
### TABLE 2

**AVIAN INFLUENZA SEROTYPES ISOLATED FROM TURKEYS, CHICKENS AND OTHER DOMESTIC FOWL IN THE U.S. (1964-1982) OR BASED ON SEROLOGY**

<table>
<thead>
<tr>
<th>STATE</th>
<th>Year First Identified</th>
<th>Hemagglutinin Antigens Identified</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Turkeys</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>California</td>
<td>1964</td>
<td>H5, H6, H9</td>
</tr>
<tr>
<td>Massachusetts</td>
<td>1965</td>
<td>H6</td>
</tr>
<tr>
<td>Wisconsin</td>
<td>1965</td>
<td>H5, H6, H9</td>
</tr>
<tr>
<td>Minnesota</td>
<td>1966</td>
<td>H1, H3, H4, H5, H6, H7, H9, H10</td>
</tr>
<tr>
<td>Washington</td>
<td>1967</td>
<td>H6</td>
</tr>
<tr>
<td>Oregon</td>
<td>1970</td>
<td>H6, H7</td>
</tr>
<tr>
<td>Iowa</td>
<td>1971</td>
<td>H1, H4, H5, H6</td>
</tr>
<tr>
<td>Colorado</td>
<td>1972</td>
<td>H1, H5</td>
</tr>
<tr>
<td>Ohio</td>
<td>1975</td>
<td>H1</td>
</tr>
<tr>
<td>Pennsylvania</td>
<td>1976</td>
<td>H7</td>
</tr>
<tr>
<td>South Dakota</td>
<td>1978</td>
<td>H1</td>
</tr>
<tr>
<td>Texas</td>
<td>1979</td>
<td>H5, H7, H9</td>
</tr>
<tr>
<td>Missouri</td>
<td>1980</td>
<td>H1</td>
</tr>
<tr>
<td>Kansas</td>
<td>1980</td>
<td>H1</td>
</tr>
<tr>
<td>North Dakota</td>
<td>1981</td>
<td>H5</td>
</tr>
<tr>
<td>Arkansas</td>
<td>1981</td>
<td>H1</td>
</tr>
<tr>
<td>North Carolina</td>
<td>1982</td>
<td>H1</td>
</tr>
<tr>
<td><strong>Chickens</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alabama</td>
<td>1975</td>
<td>H4</td>
</tr>
<tr>
<td>Minnesota</td>
<td>1978</td>
<td>H6</td>
</tr>
<tr>
<td>District of Columbia</td>
<td>1980</td>
<td>H1</td>
</tr>
<tr>
<td><strong>Other Species</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pennsylvania</td>
<td>1969</td>
<td>Ducks NA</td>
</tr>
<tr>
<td>Minnesota</td>
<td>1974</td>
<td>Geese NA</td>
</tr>
<tr>
<td></td>
<td>1974</td>
<td>Guinea Fowl NA</td>
</tr>
<tr>
<td></td>
<td>1980</td>
<td>Pheasants H3, H7</td>
</tr>
<tr>
<td>New York</td>
<td>1978</td>
<td>Ducks H3, H4, H5, H6, H11</td>
</tr>
</tbody>
</table>

NA - Not Available
TABLE 3
PREVALENCE OF MG IN COMMERCIAL TABLE EGG LAYERS AND COSTS TO THE INDUSTRY

<table>
<thead>
<tr>
<th>State</th>
<th>No. of Layers (Millions)</th>
<th>% MG Infected</th>
<th>No. MG Infected (Millions)</th>
<th>Loss Due to MG</th>
</tr>
</thead>
<tbody>
<tr>
<td>California</td>
<td>30.0</td>
<td>70.0</td>
<td>21.00</td>
<td></td>
</tr>
<tr>
<td>Georgia</td>
<td>18.3</td>
<td>8.2</td>
<td>1.50</td>
<td></td>
</tr>
<tr>
<td>Pennsylvania</td>
<td>17.6</td>
<td>75.0</td>
<td>13.20</td>
<td></td>
</tr>
<tr>
<td>Indiana</td>
<td>17.0</td>
<td>20.1</td>
<td>3.50</td>
<td></td>
</tr>
<tr>
<td>Texas</td>
<td>13.4</td>
<td>90.0</td>
<td>12.00</td>
<td></td>
</tr>
<tr>
<td>Arkansas</td>
<td>12.4</td>
<td>5.0</td>
<td>0.62</td>
<td></td>
</tr>
<tr>
<td>Ohio</td>
<td>11.5</td>
<td>25.0</td>
<td>2.90</td>
<td></td>
</tr>
<tr>
<td>Florida</td>
<td>10.6</td>
<td>6.1</td>
<td>0.65</td>
<td></td>
</tr>
<tr>
<td>Minnesota</td>
<td>10.1</td>
<td>16.8</td>
<td>1.70</td>
<td></td>
</tr>
<tr>
<td>Alabama</td>
<td>9.5</td>
<td>5.0</td>
<td>0.50</td>
<td></td>
</tr>
<tr>
<td>North Carolina</td>
<td>8.6</td>
<td>3.5</td>
<td>0.30</td>
<td></td>
</tr>
<tr>
<td>Maine</td>
<td>6.9</td>
<td>10.1</td>
<td>0.70</td>
<td></td>
</tr>
<tr>
<td>Connecticut</td>
<td>5.0</td>
<td>92.0</td>
<td>4.60</td>
<td></td>
</tr>
<tr>
<td>Mississippi</td>
<td>4.0</td>
<td>40.0</td>
<td>1.60</td>
<td></td>
</tr>
<tr>
<td></td>
<td>174.9</td>
<td></td>
<td>64.77</td>
<td>$64,770,000A</td>
</tr>
<tr>
<td>National</td>
<td>262.6B</td>
<td>37.0</td>
<td>97.16</td>
<td>$97,160,000</td>
</tr>
</tbody>
</table>

A An MG infected flock produces 20 less eggs per hen housed @ 5¢ per egg or $1.00 per hen housed

B Commercial table egg layers according to Crop Reporting Service
PREVALENCE OF SWINE DYSENTERY, TRANSMISSIBLE GASTROENTERITIS, AND PSEUDORABIES IN IOWA, ILLINOIS AND MISSOURI SWINE

I. Turney Egan, D. L. Harris, and H. T. Hill

Sera were collected from pigs at three slaughter plants serving areas of Iowa, Illinois, and Missouri. These sera were assayed for antibodies to *Treponema hyodysenteriae*, the causative agent of swine dysentery (SD), pseudorabies virus (PRV), and transmissible gastroenteritis virus (TGE). Results showed that 39.5% of the herds sampled were positive for SD, 19.4% for PRV, and 54.2% were positive for TGE.

INTRODUCTION

The prevalence of swine dysentery (SD) caused by *Treponema hyodysenteriae* has not been reported. Such information is important for determining the economic effect and formulating control measures for the disease. Improved serological methods for detecting antibodies to *T. hyodysenteriae* have recently been reported (Joens et al. 1978, 1979, 1982). The enzyme-linked immunosorbent assay (ELISA) correctly identified 93% of the herds and 31% of the pigs from SD positive herds in a recent study, indicating its potential for use as a herd test to determine the prevalence of SD (Egan et al. 1982). Such a herd survey was designed and sera were collected from market age pigs at slaughter plants. The herd and county of origin were obtained for the pigs sampled, which represented the states of Iowa, Illinois and Missouri. As little information is available on herd prevalence for pseudorabies (PRV) and transmissible gastroenteritis (TGE) of pigs, these sera were also assayed for antibodies to these two viruses.

METHODS

Geographic Location and Time Period of Survey

Sera were collected at three hog slaughter plants. Plant 1 served southeast Iowa and northeast Missouri and was visited in mid-January 1982. Plant 2 served west central Illinois and east central Missouri and was visited in late March 1982 as was plant 3 which served eastern Iowa. A total of 2,571 sera were collected, representing 157 herds in 44 counties in the three states.

SAMPLE COLLECTION

Each plant was visited on 2 consecutive days. Pigs were identified to herd with colored spray paint as they were unloaded. The county location of the herd was obtained at that time, and pigs from herds which had been sampled the previous day or earlier on the same day were not included. The herds were not identified other than to their county location of origin. When all the colors had been used, the groups were penned and a new group begun. Each herd then was identified by a color and a number. Ten to 20 animals per herd were marked. When the pigs were
slaughtered, blood was collected and the color and group number noted, and the tube of blood placed on a rack with the corresponding color and number code. The blood was allowed to clot and the serum was harvested by centrifugation then stored at $-20^\circ\text{C}$.

Serological Testing

Swine Dysentery—The ELISA was conducted as described by Joens et al. (1982). Each sample was tested with each of two lipopolysaccharide antigens representative of the two serotypes (1 and 2) of *T. hyodysenteriae* reported to occur in the United States. Sera which reacted at an optical density (O.D.) reading of 1.3 or greater with either antigen were considered to be positive for antibodies to *T. hyodysenteriae*.

Pseudorabies—The microtitration serum virus neutralization (SVN) test was conducted as described by Hill et al. (1977). A serum sample that reacted at a 1:4 or greater dilution was considered to be positive for PRV antibodies.

Transmissible Gastroenteritis—Sera were assayed with the microtitration serum virus neutralization (SVN) test described by Snyder et al. (1981). A serum sample that reacted at a 1:8 or greater dilution was considered to be positive for TGE virus antibodies.

RESULTS

Swine Dysentery

Results of the prevalence survey are presented in Table 1. Of the 2,571 sera tested, 156 (6.1%) were positive. These samples represented 157 herds of which 62 (39.5%) were positive, and 44 counties of which 29 (65.9%) were positive. In Iowa, 6.2% of the pigs, 40.9% of the herds, and 57.9% of the counties were positive. In Illinois, 6.5% of the pigs, 31.0% of the herds, and 56.3% of the counties were positive. In Missouri, 5.3% of the pigs, 55.0% of the herds, and all of the counties sampled were positive. One hundred eight samples representing 7 herds were not identified as to county of origin.
PREVALENCE OF SWINE DYSENTERY

Table 1

Prevalence of Antibodies to *Treponema hyodysenteriae*

in Iowa, Illinois and Missouri Swine

<table>
<thead>
<tr>
<th>Counties</th>
<th>Herds</th>
<th>Pigs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iowa</td>
<td>11/19* (57.9%)</td>
<td>36/88 (40.9%)</td>
</tr>
<tr>
<td>Illinois</td>
<td>9/16 (56.3%)</td>
<td>13/42 (31.0%)</td>
</tr>
<tr>
<td>Missouri</td>
<td>9/9 (100.0%)</td>
<td>11/20 (55.0%)</td>
</tr>
<tr>
<td>State, county unknown</td>
<td>N/A**</td>
<td>2/7</td>
</tr>
<tr>
<td>Total</td>
<td>29/44 (65.9%)</td>
<td>62/157 (39.5%)</td>
</tr>
</tbody>
</table>

* Numerator indicates number of positive values; denominator indicates total number tested.
** Not applicable

The ELISA results by antigen serotype are presented in Table 2.

Table 2

ELISA Results on Antigens Prepared from Two Serotypes of *Treponema hyodysenteriae*

<table>
<thead>
<tr>
<th>Serotype</th>
<th>1</th>
<th>2</th>
<th>1 and 2</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iowa</td>
<td>41*</td>
<td>71</td>
<td>16</td>
<td>96</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>15</td>
<td>13</td>
<td>36</td>
</tr>
<tr>
<td>Illinois</td>
<td>27</td>
<td>21</td>
<td>8</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>4</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td>Missouri</td>
<td>12</td>
<td>5</td>
<td>1</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>11</td>
</tr>
</tbody>
</table>

* Number with positive reactions in the ELISA

The confidence limits for the herd prevalence figure of 39.5% was ± 8% when the sample size of 157 herds was taken into consideration.

Pseudorabies

Results of the PRV survey are summarized in Table 3. Forty of the 2024 serum samples tested were toxic at the 1:4 dilution and an experimental ELISA in use at the Veterinary Diagnostic Laboratory, Ames, Iowa, was used to determine the antibody titers of these samples.
Table 3
Prevalence of Antibodies to Pseudorabies Virus in Iowa, Illinois and Missouri Swine

<table>
<thead>
<tr>
<th>State, county unknown</th>
<th>Counties</th>
<th>Herds</th>
<th>Pigs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iowa</td>
<td>11/19*  (57.9%)</td>
<td>23/87 (26.4%)</td>
<td>141/1145 (12.3%)</td>
</tr>
<tr>
<td>Illinois</td>
<td>3/16 (18.8%)</td>
<td>4/40 (10.0%)</td>
<td>23/533 (4.3%)</td>
</tr>
<tr>
<td>Missouri</td>
<td>2/9 (22.2%)</td>
<td>2/21 (9.5%)</td>
<td>15/254 (5.9%)</td>
</tr>
<tr>
<td>Total</td>
<td>N/A**</td>
<td>1/7</td>
<td>16/92</td>
</tr>
</tbody>
</table>

* Numerator indicates number of positive values; denominator indicates total number tested.
**Not applicable

Transmissible Gastroenteritis

Results of the TGE survey are presented in Table 4. A total of 2014 serum samples were assayed; 10 samples were not assayed due to an insufficient quantity of serum or to toxicity of the serum.

Table 4
Prevalence of Antibodies to Transmissible Gastroenteritis Virus in Iowa, Illinois, and Missouri Swine

<table>
<thead>
<tr>
<th>State, county unknown</th>
<th>Counties</th>
<th>Herds</th>
<th>Pigs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iowa</td>
<td>16/19* (84.2%)</td>
<td>47/87 (54.0%)</td>
<td>373/1140 (32.7%)</td>
</tr>
<tr>
<td>Illinois</td>
<td>13/16 (81.3%)</td>
<td>22/40 (55.0%)</td>
<td>147/529 (27.8%)</td>
</tr>
<tr>
<td>Missouri</td>
<td>5/9 (55.6%)</td>
<td>10/21 (47.6%)</td>
<td>49/253 (19.4%)</td>
</tr>
<tr>
<td>State, county unknown</td>
<td>N/A**</td>
<td>4/7</td>
<td>53/92</td>
</tr>
<tr>
<td>Total</td>
<td>34/44 (77.3%)</td>
<td>84/155 (54.2%)</td>
<td>622/2014 (30.9%)</td>
</tr>
</tbody>
</table>

* Numerator indicates number of positive values; denominator indicates total number tested.
**Not applicable
DISCUSSION

It was felt that herd prevalence, rather than data based on the number of individual pigs affected over a period of time was important because in the diseases of SD, PRV, and TGE, the presence of one infected pig in a herd renders the whole herd suspect and that the herds affected gave a more accurate depiction of the dimensions of the disease problems.

Several factors need consideration in the interpretation of these data. The survey was originally designed for SD prevalence determination; therefore, samples were collected in the late winter, early spring in an attempt to sample animals marketed that would have been on the farm in late summer, early fall, a time when the incidence of SD is increased. Sampling at another time may produce different results for SD or for TGE and PRV.

There may be other serotypes of T. hyodysenteriae not recognized that would not have been assayed for specifically in this study. It was interesting to note that there were more positive reactions to serotype 1 ELISA antigen in Missouri where the organism used to produce the antigen was originally isolated. Conversely, in Iowa where the organism used to produce serotype 2 antigen was originally isolated, there were more positive reactions to that serotype.

It is not known what the effect, if any, drug usage has on the development or maintenance of titers to T. hyodysenteriae.

It was not determined whether the hogs sampled were raised in farrow to finish operations or purchased as feeder pigs. This information would aid in interpretation of the prevalence percentages obtained.

Regarding PRV and TGE; by sampling only market age animals, the problem of interference with maternal antibody or titers due to vaccination was avoided.

The PRV prevalence data based on individual pigs was 9.6% which compares to the 8.4% obtained in the recent U.S.D.A. survey (1981). The herd prevalence of 19.4% reported by this survey for PRV, as compared to previous studies, may indicate the more widespread dimensions of the disease problem in a hog dense area such as the states surveyed.

The herd prevalence of TGE, 54.2%, may reflect the occurrence of enzootic TGE as these animals would have to have had a fairly recent exposure to be positive at slaughter. If a younger age group of pigs had been sampled, the number of positive samples may have even been higher.

Such data provided information on the dimensions of the diseases of SD, PRV, and TGE. It should be useful for determining the economic effect of the diseases, aiding in establishing control programs, and providing baseline data for future studies.


STREPTOCOCCAL LYMPHADENITIS OF SWINE:
A REVIEW OF INCIDENCE AND RESEARCH ACTIVITIES

R. L. Wood
National Animal Disease Center
Ames, Iowa

REVIEW OF INCIDENCE AND RESEARCH PROGRAM

Streptococcal lymphadenitis of swine, more commonly known as swine abscesses or jowl abscesses, is a disease characterized by suppurative lesions of lymph nodes, primarily those of the head and neck, and caused by group E Streptococcus (GES).

In the early 1960’s, the swine industry became concerned about the incidence of losses of pork carcasses and carcass parts at packing plants due to swine abscesses. These losses had been increasing steadily since the late 1940’s. In December 1962, a meeting of swine industry leaders was called at Iowa State University to review the problem and determine a course of action. The meeting was attended by representatives of the pork packing industry, university research and educational personnel, and other concerned individuals. Reports from industry representatives concerning losses provided ample evidence of the need for action, but there was little information available with which to develop methods of control of the disease.

At the request of those attending the meeting, a national survey of the incidence of abscesses at packing plants was conducted in 1963 by Livestock Conservation, Inc. (LCI; now Livestock Conservation Institute). The survey established an estimated annual product loss of $12,000,000, which was expected to increase to $15,000,000 by 1970 if nothing was done to control the disease. In 1964, a report of these findings and a research proposal was presented to the Secretary of Agriculture's Advisory Research Council by the newly formed LCI Swine Abscess Advisory Committee. Although the proposal was given high priority by the USDA, specific funding was not immediately forthcoming. In the meantime, research on a preliminary basis was started at the National Animal Disease Center (NADC), U.S. Department of Agriculture, Ames, Iowa, under the direction of the late Dr. C. A. Manthei, with Dr. R. D. Shuman as principal investigator.

By mid-1966, after considerable effort by LCI, including some contact with congressmen, specific and continuing funding for research on swine abscesses was granted by the federal government. These funds have been used for in-house research at the NADC conducted by Drs. R. D. Shuman, G. E. Wessman, and R. L. Wood, and for cooperative research projects conducted by Drs. John R. Collier and Robert P. Ellis at Colorado State University, Charles H. Armstrong at Purdue University, and LeRoy D. Olson at the University of Missouri.

Dr. Collier, in previous research at Iowa State University (4), had
already established the identity of the causative organism, its route of entry, and some information on pathogenesis of the disease. In addition, significant research efforts were made at or before this time by industry. Fort Dodge Laboratories was working on a vaccine and American Cyanamid already had been testing the efficacy of chlortetracycline for treatment and prevention of abscesses (12). Similar work has been done by Diamond Shamrock (14), and probably other corporate researchers.

By 1969, sufficient information had accumulated on which to base some preliminary suggestions for minimizing the problem. These, along with useful facts about the disease, were published by LCI in a leaflet distributed to the pork packing and marketing industries, veterinarians, vocational agriculture teachers, and others. The leaflet soon became a "best seller;" distribution has totalled over 100,000. It has been revised several times to reflect the latest knowledge and is still available.

After reaching a peak in the mid-1960's, the incidence of condemnations due to swine abscesses has declined steadily. The reduction in incidence is reflected by annual reports from the Federal Meat and Poultry Inspection Service on number of parts condemned (Table 1) and number of carcasses passed after removal of parts (Table 2). We believe the reduction in rate of condemnations has been due largely to the application of knowledge gained from research.

Specific federal funding for swine abscess research continued to the end of fiscal year 1982, reaching a cumulative total of nearly $3.4 million (Table 3). Because of the reduction in incidence of abscesses, limited funds, and changing priorities, the U.S. Department of Agriculture in 1979 began to reduce its support of research on the disease. The cooperative projects at Purdue University, the University of Missouri, and Colorado State University have been terminated (in 1979, 1981, and 1982, respectively). In addition, the in-house swine abscess project at the NADC was terminated in 1982.

RESEARCH ACCOMPLISHMENTS

Research objectives established in 1967 by the Swine Abscess Committee of LCI consisted of the following:

1. Definition of the properties of the causative organism.
2. Determination of the cycle of the causative organism and sources and reservoirs of infection.
4. Development of methods of control, including vaccination, medication, and other means.

Significant information has been gained under each objective. Following is a summary of the most significant progress in swine abscess research by investigators in both government and industry:
1. Properties of the causative organism.

a. Etiology. The existence of 3 serotypes (II, IV, and V) of GES, the causative organism, is generally accepted. Types formerly designated I and III are no longer recognized. Virtually all clinical swine abscesses in the field are caused by GES of type IV (1). Not all strains of type IV GES are virulent, even though they were isolated from an abscess.

b. Pathogenesis.

(1) Group E streptococci do not induce abscesses in small laboratory animals (rabbits, guinea pigs, rats, mice) (9); therefore, all in vivo research on pathogenesis and immunity must be conducted in swine.

(2) Susceptibility of swine to abscesses is influenced by age. Very young (less than 10 weeks) and mature swine (2½ years and older) are the most resistant (15). The disease occurs primarily in post-weaning and fattening swine.

(3) Abscesses induced in swine by GES occur primarily in the lymph nodes of the head and neck following infection through the mouth or nose (4). Involvement of lymph nodes in other areas of the body is unusual, but may occur if GES enter the blood stream. After ingestion, the organisms are quickly carried to the regional lymph nodes, presumably by the lymphatic vessels that drain the mouth and pharynx, including the tonsils (11). Organisms appear in mandibular and cervical lymph nodes as early as 2 hours after oral exposure. Abscesses begin forming in a lymph node as early as 48 hours after exposure, and usually can be felt or seen externally within 2 to 4 weeks.

(4) Virulent GES develop and release a protein substance called antiphagocytic factor (APF), which may be a factor in virulence (8). The APF prevents phagocytosis of GES by swine leukocytes; it is produced by GES when grown in the presence of serum under artificial conditions, and presumably when growing in the body of the host animal.

2. Cycle of the organism and sources and reservoirs of infection.

a. Cycle of the organism.

(1) Swine can become infected with GES by nasal contact with infected swine, through feed and drinking water, and through feces (5). The presence of draining abscesses is also a source of infection, but is not necessary.

b. Sources.

(1) Group E streptococci are carried by swine in the tonsils and possibly other lymphoid tissue of the digestive system (6).

(2) Evidence indicates that recovered carriers account for the endemic nature of swine abscesses. The carrier state can be established with or without the appearance of noticeable lesions.
In exceptional cases, swine can continue to transmit the disease as long as 2½ years after initial infection, but usually the carrier state lasts 10 months or less (6). Some convalescent swine do not become carriers.

(3) Group E streptococci can persist in pasture and swine-pen soil for several weeks, especially at cold temperatures (16).

3. Diagnostic tests.
   a. Swine immune to GES possess serum antibodies that neutralize the APF and promote phagocytosis of the bacteria. These antibodies can be detected by a bactericidal (opsono-phagocytic) test and a "long-chain" test (19). These tests are useful in research, but are not practical for diagnostic purposes.
   b. A microtitration agglutination test specific for antibodies to type IV GES has been developed (2). This test is sensitive; false negative reactions have not been observed. It is believed that the test could provide a basis for eradicating abscesses from individual herds by a test-and-cull approach.

4. Methods of control.
   a. Immunity.
      (1) Swine become immune to GES following recovery from the disease, and their serum will provide temporary passive immunity when injected into susceptible swine (13). Infected sows transmit passive immunity to their pigs through colostrum.
      (2) Although serum opsonins play an important role in immunity to abscesses, cell-mediated immunity appears also to have a role (3).
      (3) A commercially-produced vaccine (Jowl-Vac®) (10), prepared from a living nonabscess-inducing strain of GES, is effective for protection of pigs against abscesses, according to experimental evidence (7). Commercial manufacture has been discontinued, apparently due to lack of demand.
      (4) Partial immunity of swine to vaccination with nonliving products of GES has been demonstrated (18). Swine vaccinated with a soluble autoclaved extract of GES combined with adjuvant developed 71% fewer abscesses after challenge than did non-vaccinated control swine. It is possible that further investigation could result in an efficacious nonliving vaccine.

b. Medication and other means of prevention.
   (1) Experiments have shown that chlortetracycline (50-100 gm/T of feed), oxytetracycline (50 gm/T of feed), or penicillin (600,000 units IM every 6 days) prevents abscess formation in infected swine (12, 14, 17). Antibiotic treatment has not been successful in abscessed swine or for elimination of the carrier state.
   (2) Best results in prevention are obtained when antibiotics are
given in baby pig starter, until the pigs are 10-12 weeks of age, and when sows are also being treated.

(3) It is possible that early weaning (before 6 weeks of age) and separation of young pigs from all older hogs will reduce the incidence of abscess at market age. However, this concept has not been proven experimentally.

In summary, we have acquired some tools to deal with swine abscesses: (1) An effective vaccine has been developed; (2) A diagnostic test has been developed (still unproven in the field, but we believe it would be useful); (3) Preventive medication is available; and (4) Considerable knowledge has been gained in epizootiology and pathogenesis of the disease, which aids in development of management practices.

Further research in the following areas would be beneficial:

1. Determination of conditions controlling production of the APF in culture, using a chemically defined medium.
2. Characterization of the APF and determination whether it can be used as a diagnostic or immunizing antigen.
3. Determination of the mechanism of resistance to the disease seen in very young pigs, and the role of colostral immunity.
4. Development of improved immunization methods.

SUMMARY

Research on streptococcal lymphadenitis of swine (jowl abscesses) conducted by investigators at the National Animal Disease Center, Colorado State University, Purdue University, the University of Missouri, and by industrial researchers has provided the following tools to deal with the disease: (1) An effective vaccine; (2) A diagnostic test; (3) Preventive medication; and (4) Knowledge of epizootiology and pathogenesis on which to base improved management practices.

The incidence of jowl abscesses, as reflected by annual reports from the Federal Meat and Poultry Inspection Service, has declined steadily to a level in 1981 less than 36% of the peak incidence in the mid-1960's. Specific federal funding for jowl abscess research, which totalled nearly 3.4 million from 1967 to 1982, has been discontinued because of reduced incidence of the disease, limited funds, and changing priorities.
Table 1. Swine Carcass Parts Condemned for Abscesses/Pyemia at Federally Inspected Plants, 1957-1976

<table>
<thead>
<tr>
<th>Year</th>
<th>No. parts (per 1,000 carcasses inspected)</th>
<th>Year</th>
<th>No. parts (per 1,000 carcasses inspected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1957</td>
<td>29.1</td>
<td>1967</td>
<td>32.2</td>
</tr>
<tr>
<td>1958</td>
<td>28.7</td>
<td>1968</td>
<td>31.4</td>
</tr>
<tr>
<td>1959</td>
<td>30.2</td>
<td>1969</td>
<td>26.8</td>
</tr>
<tr>
<td>1960</td>
<td>32.1</td>
<td>1970</td>
<td>28.6</td>
</tr>
<tr>
<td>1961</td>
<td>33.7</td>
<td>1971</td>
<td>21.5</td>
</tr>
<tr>
<td>1962</td>
<td>32.2</td>
<td>1972</td>
<td>21.3</td>
</tr>
<tr>
<td>1963</td>
<td>38.1</td>
<td>1973</td>
<td>18.7</td>
</tr>
<tr>
<td>1964</td>
<td>38.6</td>
<td>1974</td>
<td>16.0</td>
</tr>
<tr>
<td>1965</td>
<td>38.8</td>
<td>1975</td>
<td>14.8</td>
</tr>
<tr>
<td>1966</td>
<td>36.5</td>
<td>1976</td>
<td>13.7</td>
</tr>
</tbody>
</table>

Table 2. Swine Carcasses Passed after Removal of Parts Affected by Abscesses at Federally Inspected Plants, 1959-1981

<table>
<thead>
<tr>
<th>Year</th>
<th>No. carcasses (per 1,000 inspected)</th>
<th>Year</th>
<th>No. carcasses (per 1,000 inspected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1959</td>
<td>48.1</td>
<td>1970</td>
<td>42.2</td>
</tr>
<tr>
<td>1960</td>
<td>49.4</td>
<td>1971</td>
<td>29.3</td>
</tr>
<tr>
<td>1961</td>
<td>51.8</td>
<td>1972</td>
<td>30.3</td>
</tr>
<tr>
<td>1962</td>
<td>53.3</td>
<td>1973</td>
<td>29.0</td>
</tr>
<tr>
<td>1963</td>
<td>57.2</td>
<td>1974</td>
<td>14.8</td>
</tr>
<tr>
<td>1964</td>
<td>56.7</td>
<td>1975</td>
<td>23.3</td>
</tr>
<tr>
<td>1965</td>
<td>55.9</td>
<td>1976</td>
<td>22.0</td>
</tr>
<tr>
<td>1966</td>
<td>52.8</td>
<td>1977</td>
<td>20.1</td>
</tr>
<tr>
<td>1967</td>
<td>47.0</td>
<td>1978</td>
<td>21.1</td>
</tr>
<tr>
<td>1968</td>
<td>45.2</td>
<td>1979</td>
<td>20.2</td>
</tr>
<tr>
<td>1969</td>
<td>39.8</td>
<td>1980</td>
<td>20.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1981</td>
<td>20.3</td>
</tr>
</tbody>
</table>
Table 3. Total Federal Designated Funds for Research on Swine Abscesses, 1964-1982

<table>
<thead>
<tr>
<th>NADC</th>
<th>$2,261,050</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cooperators</td>
<td>$1,096,500</td>
</tr>
<tr>
<td>Colorado State Univ.</td>
<td>$360,000</td>
</tr>
<tr>
<td>Univ. of Missouri</td>
<td>$422,500</td>
</tr>
<tr>
<td>Purdue Univ.</td>
<td>$314,000</td>
</tr>
<tr>
<td>Grand Total</td>
<td>$3,357,550</td>
</tr>
</tbody>
</table>

REFERENCES


REPORT OF COMMITTEE ON TRANSMISSIBLE DISEASES OF SWINE

Chairman: J.P. Kluge, Ames, IA
Vice Chairman: D.G. Thawley, Columbia, MD

J.M. Alumbaugh, IL; P.D. Beard, IA; L.G. Biehl, IL; Neal Black, MN; C.E. Boyd, SC; John Brown, GA; Jesus Castaneda G., Venezuela; R.A. Crandell, TX; R.L. Daniel, TX; P.B. Doby, IL; J.A. Downard, MD; Gene Erickson, IA; D.P. Gustafson, IN; R.E. Hall, WI; D.L. Harris, IA; G.W. Hausman, IA; H.T. Hill, IA; R.E. Horton, NJ; C.L. Kanitz, IN; M.H. Lang, IA; Norman Lichtman, NJ; Vincent Marshall, NB; J.W. McVicar, NY; K.E. Myers, IA; P.A. O' Berry, IA; Carson Rogers, NB; G.M. Schloer, NY; L.W. Schnurrenberger, MD; W.C. Stewart, IA; R.E. Thompson, AR; H.W. Towers, DE; Hsi-tang Tung, IL; C.D. Van Houweling, VA; J.P. Villari, NJ; B.D. Ward, NY; Fred Wertman, IA.

The committee on transmissible diseases of swine was convened at 1:30 pm on Tuesday, November 9, 1982. Twenty-three committee members and 21 guests were present for a total of forty-four.

Five reports were given to the committee. Firstly, Dr. R.J. Gerrits spoke on plans and projections of current Swine Disease Research supported by Federal Programs. He stressed that currently more pork products are consumed worldwide than meats from any other animal species. He described data available within the USDA Inventory of Agricultural Research Document of 1982. This data indicated that in 1982, $43 million will be spent on swine research within the U.S.A. This figure is approximately equally divided between federal and non-federal funding sources. Dr. Gerrits described the ARS Research Program and the 1982 budget allocated to both domestic and exotic disease research projects. He also described areas of research which will likely receive high priority for funding in the future. These included projects involving genetic manipulation or engineering for the development of disease control products as well as for disease resistant strains of animals. Anticipated future areas of swine research which will receive emphasis will be of the basic long term high risk type.

The second report was given by Dr. D.L. Harris who spoke on the topic "Steps to be Taken for the Eradication of Swine Dysentery." A review of the epidemiology, pathogenesis, diagnosis, immunology, and therapeutics of swine dysentery was presented. He noted a problem still existed in that diagnosis of infected, asymptomatic pigs is still not possible and that diagnosis must be on a herd basis. The problem is compounded in herds where drugs are employed which mask clinical disease. He stressed that certain drugs may be used to eradicate the disease from herds of swine, and noted that in Iowa alone the disease causes an economic loss of at least $30 million annually. Prevalence data indicates approximately 30-50% of swine herds in midwestern states are infected with *Treponema hyodysenteria*. He proposed a concept for the control of *T. hyodysenteria*. 

511
The concept is based on sale price premiums for feeder swine which originate from herds which are not using drugs that are effective against *T. hyodysenteriae* infection. No state or federal veterinarians or diagnostic laboratory capability would be needed in such a program which would be self-regulated within the swine industry. The program was proposed to be in two phases. First, an education phase in which the following points would be included: drug based eradication techniques; problems of drugs masking clinical signs; cost of drug control; and how to eliminate the disease from new herd introductions while in isolation. It was stressed that the organism can be eliminated from groups of swine within 6 days by treatment with appropriate drugs. Phase II would implement the premium plan, using a $1-2 premium for feeder pigs. This would result in a net saving of $3-4 per pig marketed if the use of drugs could be eliminated. Such a program would require no testing; and feeder pigs would be sold on the seller's insurance that they had not received a specified list of drugs over a designated time span. Details of the plan will be published elsewhere.

The third presentation was by Dr. L.W. Hinchman who gave a report from the Pseudorabies Committee and described proposed changes to the federal PRV regulations. The committee report will be presented in detail elsewhere.

The fourth presentation by Dr. Richard Wood of USDA was a review of the incidence of, and research activity on Streptococcal Lymphadenitis. He presented a history of USDA research activity on the condition, and noted that in 1982 the last remaining cooperative research project was terminated together with research activity at NADC. Over the years 1965-82, during which federal funds were used for lymphadenitis incidence was decreased by approximately 50%.

He presented a review of the properties of the causative organism, the transmission cycle, sources of infection, diagnostic tests, and recommended methods of control. Dr. Wood stated that interest in the disease has waned and the commercial vaccine is no longer available. He noted further research is still needed to improve immunization methods, to study the mechanism of young pig resistance and to determine the factors which stimulate the production of antiphagocytic factor. He emphasized that should there be a decrease in the use of antibiotics in swine management, we may well see an increase in the incidence of the condition. The paper will be published in the proceedings.

The last presentation was given by Dr. Bruce R. McClain, Director of Veterinary Services for Kleen Lean, Inc. He discussed concepts of health management which are important to swine production. He noted health management must be of prime concern to swine producers if they are to realize the benefits of current genetic selection of improved swine strains. Recommended vaccinations, slaughter checks, and serological and cultural monitoring were discussed together with emphasis on the need for isolation facilities for new introductions. The need to tailor
health maintenance plans to individual herd needs were stressed.

No resolutions were adopted at the business meeting.

John P. Kluge, Chairman
David G. Thawley, Vice Chairman
EFFECTIVENESS OF DISINFECTANTS ON *MYCOBACTERIUM PARATUBERCULOSIS*

R. S. Merkal and D. L. Whipple

**SUMMARY**

*Mycobacterium paratuberculosis* in aqueous suspension and in dried fecal smears were exposed to various disinfectants for varying lengths of time. In the aqueous suspensions, 5 min. exposure in most of the disinfectants was adequate to inactivate up to $1 \times 10^7$ colony forming units (CFU) of *M. paratuberculosis*. However, when the dried fecal smears were examined, less than $1 \times 10^3$ CFU were lost after 20 min. of exposure. Therefore, when using these compounds, it is necessary to clean the area adequately before disinfecting to effectively reduce the number of *M. paratuberculosis* organisms.

**INTRODUCTION**

Inactivation of *M. paratuberculosis* by some chemical agents has been studied using a laboratory adapted strain (2); however, the effect of a disinfectant on a laboratory adapted strain may not be the same as on a pathogenic strain. More information also was needed to determine the amount of time necessary to inactivate this organism in both aqueous suspensions and in nonsterilized organic material. We have studied the disinfectant activities of ethanol, sodium hypochlorite, and various commercial disinfectants on pathogenic *M. paratuberculosis* in aqueous suspensions and in dried fecal material.

**MATERIALS AND METHODS**

A virulent field isolate of *M. paratuberculosis* (NADC-211, grown on egg yolk agar with mycobactin (1), was used for this study.

*Disinfectants.* The disinfectants used were ethanol at various concentrations, sodium hypochlorite, a Guardall Iodine Disinfectant, b and the phenolic disinfectants Amphyl, c Con-O-Syl, c Instrument Germicide, c Lyso, c O-Syl, c Tergisyl, c Mikro-Bac, d and 1 Stroke Environ. c The sodium hypochlorite and commercial disinfectants were used at the manufacturer’s recommended concentrations except for the Instrument Germicide which had to be used at 50%.

*Aqueous Suspensions.* The aqueous suspension was exposed at 22°C to the disinfectant by adding 2 ml of the disinfectant to 2 ml of suspended *M.*

---

a(XOX bleach), Thoro Products, Co., Denver, Colorado.
bBio-Lab, Inc., Decatur, Georgia.
cNational Laboratories, Lehn and Fink Industrial Products Div. of Sterling Drug Inc., 225 Summit Avenue, Montvale, New Jersey.
dEconomics Laboratory, Inc. St. Paul, Minnesota.
eCeva Laboratories, 10560 Barkley Street, Overland Park, Kansas.
paratuberculosis. After mixing briefly, samples were taken at intervals of 0.1, 0.5, 1, 2, 5, 10, and 20 minutes. At each sampling time, 0.1 ml from the disinfectant-organism suspension was transferred to 0.9 ml of sterile water in a 13 x 100 mm culture tube. Ten 10-fold dilutions were made, then 0.25 ml from each dilution was used to inoculate egg yolk agar in a 25 cm² tissue culture flask. A set of dilutions was made and flasks were inoculated for a set of nontreated controls. All flasks were incubated at 37°C for 3 months before counting the colony forming units (CFU). The same procedure was used for all the disinfectants.

Dried Fecal Smears. A suspension of the M. paratuberculosis was mixed thoroughly into fresh bovine feces. After mixing, the feces were spread onto Schleicher and Schuell #520½ filter paper (0.5 mm thick), which had been marked into 2.5 cm x 2.5 cm squares. The feces was allowed to air dry before cutting into the squares. Each square contained approximately 11.6 mg of dried feces.

For each disinfectant, 6 squares were put into a 100 ml beaker containing the disinfectant. At intervals of 0.1, 1, 2, 5, 10, and 20 minutes, 1 square was taken from the disinfectant and dipped in distilled water. After dipping in water, each square was put into a 15 ml TenBroek tissue grinder with 10 ml of 0.3% benzalkonium chloride decontaminant.¹ The samples were ground, then the supernatant fluid was decanted into a screw cap tube and allowed to set overnight.

The next day, the samples were shaken and 0.35 ml was removed from each sample. Of this, 0.25 ml was used to inoculate a tissue culture flask (25 cm²) of egg yolk agar and 0.1 ml was added to 0.9 ml of sterile water. Ten 10-fold dilutions were made and 0.25 ml from each dilution was used to inoculate a flask of egg yolk agar medium. A set of controls was enumerated by putting the squares in water instead of disinfectant, and treating the same as the samples exposed to the disinfectants. Colonies were counted after 3 months at 37°C.

RESULTS

Aqueous Suspensions. Inactivation curves are shown in Fig. 1. Most of the disinfectants inactivated up to 10⁷ CFU within 5 min. The decimal reduction values (D-values) for all of the commercial disinfectants except Tergisyl and 1 Stroke Environ were less than 1 min. (Fig. 2). The Amphil, Mikro-Bac, and Guardall Iodine disinfectants were the most effective, having D-values of less than 0.1 min. Ethanol, at 50%, rapidly killed the organisms. Ten percent ethanol had no measurable affect. Thirty percent ethanol and sodium hypochlorite slightly reduced the number of viable organisms.

Dried Fecal Smears. In the dried fecal smears, less than 1 x 10² CFU were inactivated after 20 min. exposure to any of the commercial disinfect-

tants (Fig. 3). Only the Instrument Germicide and Amphyl inactivated the *M. paratuberculosis* enough during the time tested to have a measurable D-value. Fifty and 70% ethanol were very effective. The disinfectant quality of the sodium hypochlorite could not be assessed by this method because it immediately washed the fecal specimen off the filter paper.

**DISCUSSION**

Although most of the disinfectants effectively inactivated *M. paratuberculosis* in the aqueous suspensions, the dried organic material protected the organisms from the disinfectants. The Amphyl and Instrument Germicide, which were the most effective in the dried fecal smears, had the highest concentrations of detergent. The detergent probably helped the disinfectant penetrate the organic material. The Lysol, which was next most effective, had a lower concentration of detergent than Amphyl and Instrument Germicide. The 50% and 70% concentrations of ethanol were effective in killing *M. paratuberculosis* in dried fecal specimens, but their cost might be prohibitive for farm use. Sodium hypochlorite did not kill the organisms rapidly in aqueous suspension, but was very effective in removing the dried feces from the test specimens.

Dried fecal samples were used instead of fresh samples because in a natural setting the dried feces would be more of a problem. The cracks and holes in cement of farm buildings are difficult to clean thoroughly, and become collection sites for fecal material which dries. As these data show, the commercial disinfectants do not inactivate the organisms in the dried feces very well. Therefore, it is necessary to clean the area adequately before disinfecting so that the disinfectants can effectively reduce the number of *M. paratuberculosis* organisms.

**REFERENCES**


Fig. 1. Inactivation of *Mycobacterium paratuberculosis* in aqueous suspension.

![Graph showing inactivation of *Mycobacterium paratuberculosis* in aqueous suspension.](image)

Fig. 2. D-value (decimal reduction time), the time necessary to reduce the CFU by 1 log or 90%.

<table>
<thead>
<tr>
<th>D-VALUES</th>
<th>AQUEOUS SUSPENSION</th>
<th>DRIED FECAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>AMPHYL</td>
<td>0.83 min</td>
<td>15 min</td>
</tr>
<tr>
<td>INSTRUMENT GERMICIDE</td>
<td>1.67 min</td>
<td>16 min</td>
</tr>
<tr>
<td>CON-O-SYL</td>
<td>1.67 min</td>
<td>2</td>
</tr>
<tr>
<td>O-SYL</td>
<td>1.67 min</td>
<td>2</td>
</tr>
<tr>
<td>LYSOL</td>
<td>0.25 min</td>
<td>2</td>
</tr>
<tr>
<td>TERGISYL</td>
<td>0.55 min</td>
<td>2</td>
</tr>
<tr>
<td>MIKROBAC</td>
<td>0.080 min</td>
<td>2</td>
</tr>
<tr>
<td>1STROKE ENVIRON</td>
<td>0.20 min</td>
<td>2</td>
</tr>
<tr>
<td>GUARDALL IODINE DISINFECTANT</td>
<td>0.01 min</td>
<td>2</td>
</tr>
<tr>
<td>ETHYL ALCOHOL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10%</td>
<td>~ 0</td>
<td>~ 0</td>
</tr>
<tr>
<td>30%</td>
<td>0.80 min</td>
<td>8.0 min</td>
</tr>
<tr>
<td>50%</td>
<td>0.1 min</td>
<td>17 min</td>
</tr>
<tr>
<td>70%</td>
<td>NOT DONE</td>
<td>10 min</td>
</tr>
<tr>
<td>90%</td>
<td>NOT DONE</td>
<td>40 min</td>
</tr>
</tbody>
</table>
Fig. 3. Inactivation of *Mycobacterium paratuberculosis* in dried fecal smears.
DECONTAMINATION, MEDIA, AND CULTURE METHODS FOR MYCOBACTERIUM PARATUBERCULOSIS

R.S. Merkal, P.A.S. Lyle, and D.L. Whipple

Culturing still is the most reliable method of detecting paratuberculous animals, but the long incubation period of *Mycobacterium paratuberculosis* has been a serious obstacle to its use. We have been examining methods to shorten the incubation period, and also to reduce the number of contaminants and increase the likelihood of positive cultures from infected animals.

All other mycobacteria examined had been shown to produce one or more mycobactins, which are iron chelators necessary for growth in vitro. It had been assumed that *M. paratuberculosis* did not produce mycobactin because it is necessary to add mycobactin to the primary isolation medium for this organism. *Mycobacterium phlei* produces a mycobactin, mycobactin P, that readily crystallizes and hence is easy to purify. For many years we have produced the mycobactin P used in this country and many other countries. However, some strains of *M. paratuberculosis*, after many subcultures, eventually become mycobactin independent, which suggested that they must start producing some mycobactin themselves. After they become mycobactin independent, their incubation time is much reduced. We investigated whether we could produce a mycobactin from *M. paratuberculosis*. We used the same procedures that we use when producing mycobactin P from *M. phlei*, except that we used St. 18 *M. paratuberculosis*. A mycobactin was produced which we named mycobactin J (for the Johne’s bacillus). Mycobactin J differs from mycobactin P by having a shorter acyl group at the main hydroxamate center, in the absence of a methyl group in the benzene ring, and in the presence of an isopropyl group instead of an ethyl group at the R<sub>1</sub> position. Like all mycobactins except mycobactin P, mycobactin J does not crystallize and hence, must be purified by chromatography.

Biological assays showed that approximately the same amount of mycobactin J was needed in the medium as when mycobactin P was used. However, the cultivation time was reduced by approximately 3 weeks, and in most cases, more colonies developed on medium supplemented with mycobactin J than on the same medium supplemented with mycobactin P. We now have produced a large supply of mycobactin J and will be supplying it for future use.

The next area we investigated to improve culturing was a comparison of various decontamination procedures. We compared decontamination by oxalic acid, benzalkonium chloride, and hexadecylpyridinium chloride (HPC). The oxalic acid was used at 2.5% and 5.0% and at room temperature and 37 C for periods up to 4 hours. Benzalkonium chloride was used at 0.05%, 0.15%, and 0.25%, all at room temperature for periods up to 5 days, and the HPC was used at 0.05%, 0.15%, and 1.0% at room
temperature for periods up to 5 days. The beginning concentration of *M. paratuberculosis* was $1 \times 10^5$ for all treatments. Oxalic acid at both concentrations reduced the colony forming units (CFU) to approximately $1 \times 10^4$ by 4 hours at room temperature at both concentrations and at 37°C at 2.5%. Five percent oxalic acid at 37°C reduced the CFU to $1 \times 10^3$. At all concentrations, apparently due to clumping of the organisms, benzalkonium chloride reduced the CFU one log during the first hour, but from then until five days there was little further decrease in the number of CFU. HPC caused no measurable reduction in CFU during the entire period at any concentration.

The ability of benzalkonium chloride and HPC to eliminate contaminants and allow growth of *M. paratuberculosis* was compared on 117 fecal samples from cows and goats. Oxalic acid was not tested further because of its deleterious effect on the organisms and the large amount of effort required with its use. Duplicate aliquots of the water suspension of each fecal sample were treated with 0.3% benzalkonium chloride and with 0.75% HPC overnight at room temperature. Preliminary studies had shown that HPC concentrations between 0.1% and 0.75% were equally effective in reducing all contaminants except for rapid growing mycobacteria which sometimes tolerated 0.1% and 0.25% HPC, but not concentrations of 0.5% or 0.75%. The sediments from each suspension that formed after standing overnight (0.5 ml) were distributed onto 4 tubes of mycobactin-egg yolk agar and incubated at 37°C for 3 months. Among the 486 tubes inoculated with benzalkonium chloride treated sediments, 163, representing 52 samples, contained colonies of *M. paratuberculosis*, of which 91 were too numerous to count (TNTC), 234 contained no colonies of any kind, and 73 were contaminated. Among the 468 tubes inoculated with the HPC treated sediments, 204, representing 65 samples, contained colonies of *M. paratuberculosis*, of which 113 were TNTC, 196 contained no colonies of any kind, and 67 were contaminated. In samples from 15 animals shedding relatively small numbers of organisms, isolations were made only from HPC treated sediments and in samples from 2 animals, isolations were made only from benzalkonium chloride treated sediments. In one of these cases the caps had not been tightened, so the medium dried out and in the other case the HPC tubes were contaminated. Because of the consistently superior results of using HPC as a decontaminant, we now have switched to using it for all primary isolation work. The cost of using 0.25% HPC is 2 cents per sample compared with 1.3 cents per sample for 0.3% benzalkonium chloride.

For purposes such as drug assay, metabolite assay, etc., it frequently is desirable to grow *M. paratuberculosis* in liquid culture, and to be able to assess growth rate by optical density measurement. We have determined a number of the conditions for optimal growth rate of recently isolated strains of *M. paratuberculosis* in liquid culture.

Drug susceptibility assays such as those used to separate *M. tuberculosis*, *M. bovis*, *M. avium-intracellularare*, etc., usually are conducted in
test tubes filled 2 to 3 cm deep with the liquid medium containing the test drug. However, when *M. paratuberculosis* is inoculated in such tubes, little or no growth occurs. The same medium, in tissue culture flasks that are incubated flat allows good growth. We found that the optimum depth for liquid culture of *M. paratuberculosis* is approximately 3 mm.

In order to measure the optical density of liquid cultures in ordinary colorimeters, the culture must be in a tube rather than a flask, and if cultures frequently are transferred to tubes for measurement and then returned to the flasks for further incubation the likelihood of contamination is high. We tried a number of methods of attaching disposable culture tubes to the necks of the tissue culture flasks. We found that rubber tubing could not be used because it is toxic to the organisms and we could find no glue or cement that would make a perfect seal of the glass tube to the plastic flask. A solution was found in "shrink-tubing." This is a plastic tubing used in electrical work. It is not toxic to the organisms. When it is heated gently, it shrinks in diameter. By attaching one inch strips of shrink tubing to the ends of the glass tubes before gas sterilization, then attaching the other end of the strip to the neck of the plastic flask after it had been inoculated, a permanent attachment was formed so that repeated optical density measurements could be made without the chance of contaminating the culture.

Determination of generation time must be based on log phase growth. Fluid cultures inoculated with from 1 to 1 x 10⁷ colony forming units per 0.25 ml, then sampled at one or two day intervals, indicated that log-phase growth occurred until approximately 1 x 10⁷ CFU/0.25 ml. Optical density measurements of these cultures indicated that 1 x 10⁵⁵ CFU had to be present before they were detectable optically. By determining the time required to go from an optical density of 0.03 to 0.086, which represents a one-log increase in CFU, the generation time can be determined by optical density measurement. Generation time is defined as T/3.3 (log b/B) or the time for a 1 log increase divided by 3.3.

The speed of growth of most aerobic organisms can be increased by shaking. We examined the effect of shaking *M. paratuberculosis* both in the tissue culture flasks and in nephelometer flasks at speeds up to 150 rpm. As the speed of shaking was increased, the growth rate of *M. paratuberculosis* decreased. Hence, the optimum growth of this organism occurs in stationary culture.

The growth of some mycobacteria is stimulated by incubation in the presence of carbon dioxide. Duplicate cultures of *M. paratuberculosis* were incubated in the presence of 6% carbon dioxide and in normal atmosphere, both stationary and shaking at 50 rpm. Carbon dioxide did not increase the growth rate in either case.

One of the standard liquid media for culture of mycobacteria is Middlebrook 7H9 supplemented with either Dubos' Oleic-Albumin Complex (DOAC) or Oleic Acid-Albumin-Dextrose-Catalase (OADC) or Albumin-Dextrose-Catalase (ADC) enrichments. We compared these 3 enrich-
ments and found that growth was consistently faster with the OADC enrichment when the medium was prepared at the normal pH which is about pH 6.6.

Other media for the cultivation of mycobacteria usually have a pH of 7.0 or greater. To determine the optimum pH for *M. paratuberculosis* we adjusted the medium in increments of 0.2 to pH's of 5.0 to 8.0. The shortest generation time was in medium adjusted to pH 5.8 or 6.0. In medium at pH 5.9, the DOAC supplement allowed the most rapid growth.

By using the optimum depth, pH, enrichment, and stationary growth, the generation times were reduced from the previously measured 48 hours to only 8 hours. We now can do a complete drug assay in 7 to 10 days, without resorting to radioisotope studies. We hope in the near future to be able to use liquid culture for primary culture from clinical specimens.

---

*Fig. 1. Inactivation of Mycobacterium paratuberculosis* in aqueous suspension.
Fig. 2. D-value (decimal reduction time), the time necessary to reduce the CFU by 1 log or 90%.

<table>
<thead>
<tr>
<th>D-VALUES</th>
<th>AQUEOUS SUSPENSION</th>
<th>DRIED Fecal</th>
</tr>
</thead>
<tbody>
<tr>
<td>AMPHYL</td>
<td>083 min</td>
<td>15 min</td>
</tr>
<tr>
<td>INSTRUMENT GERMICIDE</td>
<td>167 min</td>
<td>16 min</td>
</tr>
<tr>
<td>CON-O-SYL</td>
<td>167 min</td>
<td>20 min</td>
</tr>
<tr>
<td>O-SYL</td>
<td>167 min</td>
<td>20 min</td>
</tr>
<tr>
<td>LYSOL</td>
<td>25 min</td>
<td>20 min</td>
</tr>
<tr>
<td>TERGISYL</td>
<td>55 min</td>
<td>20 min</td>
</tr>
<tr>
<td>MIKROBAC</td>
<td>080 min</td>
<td>20 min</td>
</tr>
<tr>
<td>1 STROKE ENVIRON</td>
<td>20 min</td>
<td>20 min</td>
</tr>
<tr>
<td>GUARDALL IODINE DISINFECTANT</td>
<td>01 min</td>
<td>20 min</td>
</tr>
<tr>
<td>ETHYL ALCOHOL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10%</td>
<td>~ 00</td>
<td>~ 00</td>
</tr>
<tr>
<td>30%</td>
<td>80 min</td>
<td>80 min</td>
</tr>
<tr>
<td>50%</td>
<td>01 min</td>
<td>17 min</td>
</tr>
<tr>
<td>70%</td>
<td>NOT DONE</td>
<td>10 min</td>
</tr>
<tr>
<td>90%</td>
<td>NOT DONE</td>
<td>40 min</td>
</tr>
</tbody>
</table>

Fig. 3. Inactivation of *Mycobacterium paratuberculosis* in dried fecal smears.
EPIDEMIOLOGICAL STUDY OF AN OUTBREAK OF BOVINE TB IN CONFINED ELK HERDS

Charles D. Stumpff, D.V.M.

Reports of tuberculosis infection in elk are rare in the United States. What reports that do exist are occasional incidents in elk involved under zoo conditions. Reports of tuberculosis in other members of cervidae are also few in number. One of the most complete reports was made in the March issue of the American Journal of Veterinary Research, 1965. This is a report on tuberculosis in captive fallow deer by Towar, Scott and Goyings.¹

The following case report will cover an outbreak of tuberculosis in captive elk herds located in North and South Dakota, including epidemiological considerations.

HISTORY AND DIAGNOSIS

Herd "O". In November 1980, herd owner purchased 42 bull elk from herd owner "H", both located in the State of South Dakota. These animals were placed in the same enclosure with approximately twenty other elk owned by herd owner "O". These elk were in approximately 320 acres of pasture surrounded by an eight foot woven wire fence. Animals were fed hay and grain in cattle feeders. Water was provided in regular stock watering tanks. Essentially, the elk were cared for under conditions similar to cattle. There were no other cattle that had fence line contact with this group of elk. However, there were cattle on adjoining farms. The elk were located in ranch country and maintained under ranch conditions.

CLINICAL SIGNS

Few clinical signs were observed. Poor conditioning was the first indication of illness. Also animals would lag behind the rest of the herd. Animals would eventually go down and die shortly thereafter in spite of treatment. One animal has an abscess in flank region. This abscess was drained but did not respond to treatment.

In January 1981, the first death loss occurred. Losses continued at three to four week intervals until a total of ten animals had died. Nine animals were autopsied with eight showing suspicious lesions. Two of these were confirmed as M. bovis at National Veterinary Services Laboratory (NVSL) on August 11, 1981 and August 14, 1981. The exact method of tuberculosis testing of many exotic animals is not fully understood. The method used for domestic cattle is often used in wildlife species. One such report of tuberculosis testing of captive fallow deer in

Michigan by Towar, Scott and Goyings lists tests on many animals with apparent accurate results. Similar testing methods were used in this report of elk herds.

A cervical test was conducted on the remaining 53 animals. Thirty animals were classified as reactors with 23 animals negative. Reactors were separated from the negative animals. The test consisted of the application of .1 cc. special PPD for cervical use applied to the cervical region. The site was midway in the neck in a clipped area prepared by Oster clipper using a No. 40 surgical blade. Skin thickness was measured prior to injection and at 72 hours post injection. Diameter size of the response was also reasured. Any response was considered as a reactor. Application and interpretation of this test was made in accordance with U.S.D.A. recommendations for cattle. Similar procedures were used by Scott, Towar, and Goyings in their report of tuberculosis in a captive deer herd.

Increase in skin thickness ranged from 1 mm. to 27 mm. Diameter measurements of responses varied from 5 mm. to 40 mm.

PATHOLOGY

The first animal involved had a large abscess in the flank region. This animal was restrained and the abscess treated. Complete healing never occurred. Autopsy has been made of nine animals, eight exhibited lesions suspicious of tuberculosis. All animals had granuloma type of lesions in the lung tissue with little or no lymph node involvement. At least one animal had two huge mesenteric node abscesses. M. bovis was isolated from two specimens submitted to NVSL. One animal died about a week after the testing procedure. This animal had gross lung lesions and mesenteric lymph node involvement. Histopathology results were suggestive; however, bacterial culture attempts were not successful. At the time of tuberculosis testing, the elk were also brucellosis tested. Two elk were classified as reactors.

Herd “H”. Herd Owner “H” is a dealer in native exotic wild animals. In addition, he maintains a herd of buffalo, elk and deer. When he became aware of the problem in herd “O”, he arranged slaughter of five adult cows. One cow elk slaughtered had gross lesions of tuberculosis in the mediastinal lymph nodes. M. bovis was subsequently isolated from tissues submitted to NVSL. Herd “H” was tested as outlined for herd “O”, Twenty reactors were disclosed. Fifteen were negative to the test. One animal that was classified as a reactor had an extensive draining abscess on the right side of the rib region. Exudate material submitted to NVSL resulted in another M. bovis isolate. Herd owner “H” believes that the infection probably entered his herd in the fall of 1978 from a purchase made at a zoo in Iowa. During the past two years he had an unexplained death loss. The buffalo herd on the same premise was tested with negative results. One herd of domestic cattle was also tested. One animal was classified as suspect. Upon slaughter no gross lesions were observed. This herd will remain under surveillance and further testing will be
completed. Animals were also tested for brucellosis with approximately 90\% classified as reactors.

Herd owner "J". Herd owner "J" (North Dakota) purchased 17 elk from herd owner "H" in summer of 1981. Five animals died of unexplained causes within 2-3 months. A test on this herd revealed 13 reactors of 15 tested. Similar test procedures were used as previously described. One animal with a minute response was autopsied. One small gross lung lesion was observed. At this time laboratory confirmation of tuberculosis has not been made.

Herd Owner "L". Herd "L" (North Dakota) purchased one bull calf April of 1979 from herd owner "H". This animal, plus thirteen others was tuberculosis tested in accordance with previous described procedures. All fourteen animals were negative. No clinical signs have been observed in this herd.

Herd "R". Seven bull calves were sold to herd "R" in South Dakota during the fall of 1979 from herd "H". Field investigation of herd "R" indicated that the animals involved were immediately shipped to herd "F" in Wyoming. Herd "R" maintains a privately owned animal exhibit that is open to the public. Elk at this exhibit have been tuberculosis tested with negative results.

Herd "F". Herd "F" located in northern Wyoming purchased bull elk sold by herd "H" to herd "R". Herd "F" maintains a ranch of 20,000 acres that is completely fenced to maintain elk. This ranch has approximately 150 elk and 400 cattle. Tuberculosis test on the cattle and elk were negative. Montana tested several elk herds that had received elk from this ranch. All elk herds tested in Montana were negative.

Circumstantial evidence indicates that herd "H" was the probable source of this tuberculosis outbreak. Effort was made to determine the source of herd "H" infection. Movements are illustrated in Figure 1. Nine herds located in seven states were investigated. Several herds were tested and found to be negative. Herd "H" had bought animals from many sources including zoos. One theory that has circumstantial support is that the infection entered herd "H" from a zoo purchase in 1978. This animal died of apparent clinical signs of tuberculosis a few months later but no effort was made to determine the cause of death. At this time the source of infection in herd "H" had not been determined.

Three persons associated with the South Dakota herds converted from negative to positive status on a tuberculosis test. The attending veterinarian associated with herd "O", herd owner "H", and a rendering plant worker were persons involved.

This outbreak in South Dakota resulted in extensive legal problems that is still continuing. Herd "O" was retested in August 1982, 46 reactors were destroyed and ten animals were classified as negative and will be subjected to retest. Herd "H" was retested with 30 of 31 animals being classified as reactors. All elk were destroyed. Five deer were tested with
two reactors. All deer were destroyed. Both herds during the period of
first and second testing continued to experience death losses of ap-
proximately 20%.

Herd "J" in North Dakota experienced heavy death losses in the
winter of 1981 and 1982. Eight animals were destroyed in February of
1982.

Some important conclusions from this tuberculosis outbreak is as
follows:

1. As the bovine tuberculosis program reaches its final conclusion, con-
sideration must be given to handling of reservoirs of tuberculosis in
exotic animal collections. Many are maintained in agricultural con-
ditions and represent a potential source of tuberculosis to livestock
populations.

2. In many instances, State and Federal regulations are not adequately
or nonexistent to properly handle outbreaks of tuberculosis in exotic
animal collections.

3. All tuberculosis problems in animals present a public health hazard
to humans. This incident re-emphasizes the need for adequate
safeguards in handling tuberculosis infected animals.

4. There is a need for information in regard to tuberculosis testing
techniques and methods of diagnosis of tuberculosis infection in
exotic animals. This outbreak presented an unusual opportunity to
evaluate such procedures. This opportunity was loss due to absence
of funds and supporting regulations.

FIGURE #1

INFECTED HERDS
 Negro EXPOSED HERDS
 Possible source Herds, status undetermined
EXPERIMENTAL INFECTION OF SWINE WITH MYCOBACTERIUM AVIUM SEROTYPE 4: LESIONS AND TRANSMISSION

H. M. Acland and R. H. Whitlock
University of Pennsylvania
School of Veterinary Medicine
New Bolton Center
Kennett Square, PA 19348

Tuberculosis in swine is a significant cause of financial loss to meat packers and pork producers in the United States because the presence of mycobacterial lymphadenitis necessitates the trimming, cooking or condemnation of carcases.\textsuperscript{1} Mycobacterium avium serotypes 4 and 8 have recently been recovered from herds with a high incidence of lesions.\textsuperscript{2,3} Serotype 4 is considered important in Pennsylvania.

Often the source of \textit{M. avium} is unknown, even though in some outbreaks there has been strong evidence that deep litter,\textsuperscript{4} soil\textsuperscript{5} and contaminated bedding of sawdust or woodshavings\textsuperscript{6} are involved. \textit{M. avium} has been recovered from bedding material.\textsuperscript{7} Congenital transmission has been reported.\textsuperscript{8} Excretion of \textit{M. avium} serotype 6\textsuperscript{9} and serotypes 1 and 2 and an untyped isolate\textsuperscript{10} in the feces of inoculated pigs has been documented. In studies with small numbers of contact animals using a serotype of porcine origin\textsuperscript{11} and serotype 8\textsuperscript{12} transmission of tuberculosis from inoculated to contact animals occurred.

The experimental infection of groups of pigs at 4, 8, 12, 16 or 20 weeks of age with serotype 4 showed that pigs infected at 8 weeks of age were the most likely to have gross lesions that would cause condemnation at slaughter.\textsuperscript{13} Transmission to uninoculated in contact animals was minimal. In this paper the gross lesions are further analyzed and the histological lesions are reported.

MATERIALS AND METHODS

As described previously\textsuperscript{14} swine were housed in a modular building with fiberglass-plastic slat flooring. Each group of 5-8 pigs was kept in a pen with solid 3' high walls. Groups of pigs were inoculated at 4, 8, 12, 16 and 20 weeks of age with 1.5 ml (30mg/ml) of \textit{Mycobacterium avium} serotype 4 prepared by Dr. Charles O. Thoen, Iowa State University. For each age group there was a similar group of uninoculated pigs that was held in a separate pen to prevent the acquisition of organisms from the inoculum shed in the feces of the treated pigs. After 4 weeks the 2 groups were mixed and divided between the 2 pens. One exception to this protocol (caused by an insufficiency of inoculum) was 1 uninoculated, 12-week-old pig being held with the inoculated animals from the onset. The pigs were slaughtered when they attained market weight, at about 210 lbs. This was at 126-134, 116-120, 83-84, 72-76 and 42-43 days after inoculation for the 4, 8, 12, 16 and 20 week groups respectively.
Tissues collected for histological examination were tonsil, soft palate, lung (right apical lobe), thymus, spleen, liver, esophagus, lymph nodes (mandibular, parotid, suprpharyngeal, posterior superficial cervical, left bronchial, gastric, hepatic, anterior mesenteric, posterior mesenteric, external iliac), stomach (esophageal and fundic), duodenum (cranial flexure), anterior jejunum (junction of 1st and 2nd third), posterior jejunum (junction of 2nd and 3rd third), ileum (4 cm from cecum), ileocecal valve, mid-cecum, apex of spiral colon, rectum (30 cm from anus). Adrenal gland and kidney were collected if available after the commercial slaughtering process. Specimens from the gastrointestinal tract were fixed in Bouin’s fluid and other specimens were fixed in 10% buffered formalin. Tissues were embedded in paraffin and stained with hematoxylin and eosin.

RESULTS

Gross lesions. Lesions found in the 65 pigs were summarized in Table 1. Miliary caseous and calcareous foci, caseous and calcareous foci up to 0.5 cm in diameter and sometimes larger were present, with and without enlargement, in the anterior part of the mesenteric chain and the posterior part of the mesenteric chain in most pigs inoculated at 4 weeks and 8 weeks of age and in a smaller proportion of the pigs inoculated at 12 and 16 weeks of age, but not in any of the pigs inoculated at 20 weeks of age. The mandibular lymph nodes had similar lesions in only 1 age group of pigs, those inoculated at 8 weeks. Two pigs were affected. Lesions of this type were not present in any other location. Enlargement with no other change was recorded in the bronchial lymph node of 1 contact pig in the 8 week age group, 1 pig inoculated at 12 weeks and in 3 pigs each in the 16 week inoculated and contact groups. Similar enlargement of both the anterior and posterior parts of the mesenteric chain was present in 1 pig from the 8 week contact group (not the pig with bronchial lymph node enlargement) and in 2 pigs from the group inoculated at 20 weeks of age. One pig in the group inoculated at 12 weeks (not the pig with the bronchial lymph node lesion) had an enlarged hepatic lymph node.

Grey and red consolidation of the anterior and ventral lung lobes, considered typical of enzootic pneumonia, was present to a mild or moderate extent in 26 pigs—2, 1, 2, 5 and 2 respectively in the pigs inoculated at 4, 8, 12, 16 and 20 weeks and 5, 1, 1, 5 and 2 of the corresponding contact groups. All the pigs with enlarged bronchial lymph nodes were involved. Mild atrophic rhinitis was present in a few pigs, but the nasal cavity was not examined in all pigs. Ulceration of the gastric squamous mucosa occurred in several pigs. No other gross lesions were observed.

Histological lesions. All the inoculated animals had tuberculous granulomas in at least one site. Two in-contact animals had lesions. The main locations of the inflammation observed histologically were the tonsils, mandibular and parotid lymph nodes, mesenteric chain of lymph nodes and the mucosa and submucosa of the small intestine and ileo-cecal
valve. Results are summarized in Table 2. Other sites where tuberculous lesions were present were suprpharyngeal lymph node (1 pig inoculated at 8 weeks) and hepatic lymph node (1 pig inoculated at 8 weeks and 1 at 20 weeks). No tuberculous lesions were seen in any pig in the soft palate, lung, bronchial lymph node, spleen, liver, esophagus, stomach, duodenum, cecum, colon, rectum, thymus, pancreas, adrenal, kidney, gastric lymph node, posterior superficial cervical lymph node, or external iliac lymph node. All the lymph nodes that had grossly visible caseation and calcification were histologically tuberculous (Table 1). None of the lymph nodes that on gross examination were only enlarged had TB lesions histologically, except for the mesenteric lymph nodes of two pigs inoculated at 20 weeks of age, in which the tuberculous lymphadenitis was moderately severe. The majority of the enlarged lymph nodes were either hyperplastic or had mild acute to subacute diffuse lymphadenitis.

Histologically, the tuberculous granulomatous inflammation had a range of appearances. In the animals inoculated at 20 weeks it tended to be discrete or diffuse aggregations of epithelioid macrophages, while in those inoculated at 16 weeks there was a mixture of epithelioid cells and multinucleate giant cells. In those animals inoculated at 12, 8 and 4 weeks the amount of caseation and calcification and fibrous encapsulation progressively increased and the number of giant cells dramatically decreased. In the contact animal placed with the pigs inoculated at 12 weeks the granulomas in the ileum and ileo-cecal valve were minimal and composed of aggregates of epithelioid and giant cells in the Peyer's patch. The other affected in-contact pig was with the group inoculated at 4 weeks, and had a moderately severe diffuse infiltration of the posterior mesenteric lymph node with epithelioid cells and giant cells.

All the lungs that grossly had lesions of enzootic pneumonia had histological changes typical of the disease, with the addition that in the 4 week age group 2 of the inoculated animals and 3 of the contact animals had as well multiple small discrete granulomas in which large, elongate, pale basophilic foreign bodies were present.

DISCUSSION

The impression gained from the gross examination of the carcases of the inoculated and in-contact animals that swine tuberculosis caused by Mycobacterium avium serotype 4 is a disease of low contagiousness is still a valid one after the completion of the histological examination of the specimens. The 1 in-contact animal thought, because of enlargement of mesenteric lymph nodes, to have tuberculosis did not, but microscopic lesions were found in 2 other in-contact animals that had no gross lesions. It would be extremely unwise to regard lymph nodes with only enlargement to be non-tuberculous in view of the histological TB lesions found in the 2 such lymph nodes from animals inoculated at 20 weeks.

Experimental infection of swine with serotypes 1 and 2 and an unknown serotype suggested that mycobacteria in the feces for the first 10 days were from the inoculum, while those excreted between 20 and 69
days were from the lesions in the tonsils and intestines. The incorporation of a time interval of 4 weeks between inoculation of pigs and mixing with contact animals in the current experiment was chosen in the hope of creating a situation closer to that in the field. The finding of lesions in the intestinal walls in the 3 groups of pigs with shorter time intervals between inoculation and killing suggests that these lesions are present for a finite time after inoculation and then resolve. It would be reasonable to assume that organisms are shed into the gut lumen from these lesions and excreted in the feces. Small amounts of feces did accumulate in the pens. Culturing of the feces that were collected from each animal at intervals through the experiment will give information on the size of the inoculum that would have been available to contacts. Another experiment in progress, involving the killing of pigs at intervals after inoculation at a single age will give a better picture of the progression of gut lesions.

Tuberculosis in swine caused by Mycobacterium avium serotype 4 appears to be an almost non-contagious disease under the conditions of this experiment. In field outbreaks it would be worthwhile to seek a common source of organisms, rather than to consider pig-to-pig transmission an important factor.

SUMMARY

Groups of 5-8 pigs were either orally inoculated with 45 mg of Mycobacterium avium serotype 4 or, from 4 weeks after inoculation, held in contact with inoculated pigs. Pigs were inoculated at 4, 8, 12, 16 and 20 weeks of age, and killed when they reached approximately 210 lbs. weight. Caseo-calcareous foci were present in the mesenteric lymph nodes of most of the pigs inoculated at 4 and 8 weeks, some of the pigs inoculated at 12 and 16 weeks and none of those inoculated at 20 weeks. Similar foci were present in the mandibular lymph nodes of only the pigs inoculated at 8 weeks of age. Histologically, all the inoculated animals had tuberculous granulomatous lymphadenitis in at least one site, most frequently the mesenteric lymph nodes. Many of those in the 3 age groups of later inoculation had lesions in the intestinal tract and many of those in the group inoculated at 20 weeks had tuberculous tonsilitis. Two in-contact animals had microscopic lesions. One held with the group inoculated at 12 weeks had very mild lesions in the intestine, and 1 with the group inoculated at 4 weeks had a moderately severe lesion in a mesenteric lymph node. In inoculated animals a range in characteristics of the microscopic lesions that changed with age or time between inoculation and killing could be seen. As tuberculosis caused by Mycobacterium avium serotype 4 appears to be a disease of low contagiousness, in field outbreaks attention should be paid to a possible common source of infection rather than to pig-to-pig transmission.

Acknowledgement.

This project was supported by The Pennsylvania Fair Fund, Grant #ME-44, Commonwealth of Pennsylvania.
Table 1. Numbers of pigs with gross and histological lesions in lymph nodes

<table>
<thead>
<tr>
<th>Age (weeks)</th>
<th>Treatment</th>
<th>Number of pigs</th>
<th>Lymph Nodes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mandibular</td>
<td>Anterior mesenteric</td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>C</td>
</tr>
<tr>
<td>4</td>
<td>Inoculated</td>
<td>7</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>8</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>Inoculated</td>
<td>7</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>7</td>
<td>-</td>
</tr>
<tr>
<td>12</td>
<td>Inoculated</td>
<td>5</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>7</td>
<td>-</td>
</tr>
<tr>
<td>16</td>
<td>Inoculated</td>
<td>6*</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>6</td>
<td>-</td>
</tr>
<tr>
<td>20</td>
<td>Inoculated</td>
<td>6</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>6</td>
<td>-</td>
</tr>
</tbody>
</table>

(s) = Number of specimens with a gross lesion that histologically had granulomatous lymphadenitis.
E = Enlargement alone.
C = Caseation and/or calcification, with or without enlargement.
* = 5 pigs examined completely, 1 pig only head and iliac lymph nodes examined.

Table 2. Numbers of pigs with microscopically visible tuberculosis lesions

<table>
<thead>
<tr>
<th>Age in weeks</th>
<th>Treatment</th>
<th>Number of pigs</th>
<th>Site</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mandibular</td>
<td>Anterior mesenteric</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lymph node</td>
<td>lymph node</td>
</tr>
<tr>
<td>4</td>
<td>Inoculated</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>Inoculated</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>12</td>
<td>Inoculated</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>7</td>
<td>0*</td>
</tr>
<tr>
<td>16</td>
<td>Inoculated</td>
<td>6</td>
<td>1*</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>20</td>
<td>Inoculated</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Contact</td>
<td>6</td>
<td>0</td>
</tr>
</tbody>
</table>

* = Only 6 tonsils examined.
* = Only 5 tonsils examined.
$ = 6 pigs inoculated, viscera of only 5 examined.
REFERENCES


In Fiscal Year 1982, tuberculous infected cattle herds were detected geographically in four corners of the continental United States. These herds were located in California, a dairy cattle importing State; Washington, a State which has not experienced an infection outbreak since 1964; Florida, a State that imports a great volume of dairy cattle; and an isolated outbreak in New York, an Accredited-Free State. In addition to the four States noted, infected herds were found in southern Louisiana. Tuberculosis has been endemic in southern Louisiana for many years. Bovine tuberculosis shows once again that it is no respector of State status or geographic location.

In Fiscal Year 1982, there was a significant increase in the number of regular-kill tissue submissions. This increase was higher than any tissue submissions received for a single year in the last 10 years. The number of case investigations closed was 78. This was the largest case load since 1978 and was due to 75 submissions from feedlot cattle. Thirty-eight out of the 75 feedlot cases, or 51 percent, have been documented as imported feeder steers. The figure actually would be much greater if all available information was considered.

Depopulation of infected and exposed herds was carried out to the limit of available Federal indemnity funds. At the close of FY 1982, there are two confirmed *M. bovis* herds and one exposed herd still remaining under quarantine.

Figure 1—In FY 1982, two additional States became Tuberculosis Accredited-Free. These two States were Virginia, declared Accredited-Free on July 1, 1982, and Nebraska, declared Accredited-Free on October 1, 1982. This brings the total number of States Accredited-Free to 22. These States are: Arizona, Colorado, Connecticut, Maine, Maryland, Michigan, Minnesota, Montana, Nebraska, New Hampshire, New Jersey, New Mexico, New York, North Carolina, North Dakota, Rhode Island, South Carolina, Utah, Vermont, Virginia, Wisconsin, and Wyoming, plus the Virgin Islands. Enactment of dealer laws in several other States has prompted requests for Accredited-Free status in FY 1983. There are eight States that have not experienced an *M. bovis* infected herd in over 5 years.

Figure 2—The location of 11 tuberculous herds is illustrated in Figure 2. There was one infected herd each in Washington, California, New York, and Florida. There were three infected and three exposed herds in Louisiana and one exposed herd in Georgia. In FY 1982, there were a total of seven infected and four exposed herds.

Figure 3—Herds were located in the following manner. Traceback
from regular-kill slaughter animals accounted for four infected herds. *M. bovis* was isolated initially in two cases from the suspicious bovine tissue submitted. In the third case, *M. bovis* was isolated from thoracic lesions of a slaughter pig and investigation led to the cattle herd. In the fourth case, *M. bovis* was not isolated from slaughter submission but from reactors when the source herd was tested. One herd was located by routine area testing and retest of its suspects by the comparative cervical (c-c) test.

The c-c test reactors demonstrated lesions positive on guinea pig isolation and biochemical typing. An exposed herd was located through tracing of offspring from an infected dairy into a second State. The exposed heifers were depopulated after extensive investigations. Five exposed and infected herds were located through retest of quarantined herds.

Figure 4—Epidemiologic tracing detected five infected and exposed herds. Four of the five herds were located through traceback from slaughter and one as a result of tracing from an infected herd in Florida. All other tuberculin testing detected six herds (one infected herd in California and five out of the six infected and exposed herds in Louisiana). It might be debated that the five herds in Louisiana were detected through epidemiological tracing since one of the infected herds contained cattle from the same source that infected the herd located through a 6-35 investigation. However, since these herds had been under test for a period of time as high risk herds, it was decided to classify them as quarantined herds and include them in all other tuberculin testing.

Figure 5—Fiscal Year 1982 did not duplicate FY 1981 when all detected herds were depopulated. Eight of the 11, or 73 percent, of infected and exposed herds were depopulated and indemnity funds were totally utilized. Lack of funds was not the sole reason for three herds remaining under quarantine at the close of FY 1982.

Figure 6—This graph illustrates the proportion of tuberculous herds depopulated and their locations. A large dairy herd in California remains under quarantine. Retest of this herd has given a low rate of response to tuberculin testing. The animals have shown little gross pathology and there has not been another *M. bovis* isolation. The source of infection appears to have been from a recent introduction with limited transmission within the herd. Two herds in Louisiana (one infected and one exposed) were not depopulated because of owners' reluctance and financial considerations that were important to the owners. These herds are under quarantine and testing. Depopulation will be considered in FY 1983.

Figure 7—The presence or absence of identification devices and their submission with suspicious regular-kill lesions continues to play a major part in successful case investigations. Of 75 unidentified specimens, 2 percent were successfully closed, whereas with identification the success rate was 66 percent.
Figure 8—A line graph shows the suspicious tissues submitted from regular-kill slaughter animals to National Veterinary Services Laboratories (NVSL) for histopathology and bacteriology in FY 1982. Compared with the same information for FY 1981, there was an increase of 106 tissue specimens. There was a 2 percent increase in adult animals sampled and a 2 percent increase in adult animals with identification. The percent of identification devices collected from adult animals to submit with tuberculosis sample did not change. This may be because it is necessary to retrieve all identification devices from MCI blood collection stations in order to send these ID’s with the tuberculosis specimen. The submission of all identification devices provides information far beyond just their numbers. They are used to verify information on farms, in the packing plants, and at auction markets. Backtags provide information to the epidemiologist on the color and breed of the animal.

Figure 9—Tuberculosis traceback investigation cases closed in FY 1982 were 2,022. This is somewhat larger than the number of cases submitted which is illustrated in the preceding graph as 1,895. This was due to some cases that were pending from last year and were carried over. Seventy-eight investigations were closed and written epidemiological reports were submitted. This was discussed at the onset of the status report and represents an increase this year in total submissions and closing of feedlot cases.

In conclusion, there were a total of 11 tuberculous herds in FY 1982, of which eight were depopulated, as compared to 13 herds located and depopulated in FY 1981. Herds not depopulated and released from tuberculosis quarantine and monitored through annual high-risk herd testing should be tested on schedule. The testing veterinarian should be alert to the fact that some of these herds are very likely to contain residual infection. This occurrence has been demonstrated in both Fiscal Year 1981 and Fiscal Year 1982.
Tuberculosis Eradication

Bovine Tuberculosis Area Status

September 30, 1982

Accredited Free States (22) plus Virgin Islands

Modified Accredited Areas (28) plus Puerto Rico

* No M. Bovis for Over 5 Years (8)

Tuberculosis Eradication

Location of 11 Tuberculous Herds

FY 1982
Tuberculosis Eradication
Methods of Locating 11 Tuberculous Herds Initially Detected during FY-82

- Area Testing (1)
- Traceback of Regular Kill Slaughter Animals (4)
- Retest of Quarantined Herd (5)
- Tracing Exposed Cattle from Affected Herds (1)

Tuberculosis Eradication
Detecting Herds with TB Infection:
1972 through 1982

![Graph showing number of herds detected each year from 1972 to 1982.](image-url)
Tuberculosis Eradication

Herds Found vs. Herds Depopulated

FY 1972-82

<table>
<thead>
<tr>
<th>Year</th>
<th>Herds Found</th>
<th>Herds Depopulated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1972</td>
<td>52</td>
<td>21</td>
</tr>
<tr>
<td>1973</td>
<td>38</td>
<td>17</td>
</tr>
<tr>
<td>1974</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>1975</td>
<td>47</td>
<td>29</td>
</tr>
<tr>
<td>1976</td>
<td>52</td>
<td>37</td>
</tr>
<tr>
<td>1977</td>
<td>29</td>
<td>17</td>
</tr>
<tr>
<td>1978</td>
<td>29</td>
<td>19</td>
</tr>
<tr>
<td>1979</td>
<td>28</td>
<td>17</td>
</tr>
<tr>
<td>1980</td>
<td>17</td>
<td>6</td>
</tr>
<tr>
<td>1981</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>1982</td>
<td>11</td>
<td>8</td>
</tr>
</tbody>
</table>

Tuberculosis Eradication

Proportion of Tuberculous Herds Depopulated

FY 1982

[Map showing proportion of tuberculous herds depopulated]
Tuberculosis Eradication

Traceback of 78 Tuberculous Cases (Regular Kill Animals) FY 1982

- 75 Unidentified
  - 98% Unsuccessful
  - 2% Successful
- 3 Identified
  - 34% Unsuccessful
  - 66% Successful

Tuberculosis Eradication

Number of 6-35’s Submitted FY 82 (Federal Establishments)

- Number of 6-35’s Submitted
- Number of Adults with Identification
- Number of Adult Animals
- Number of ID Devices Submitted

Tuberculosis Eradication

Tuberculosis Traceback Investigations
(Regular Kill)

FY 1982 (Cases Closed)

<table>
<thead>
<tr>
<th>Fiscal Year</th>
<th>Cases not tuberculosis</th>
<th>Cases of tuberculosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1973</td>
<td>1044</td>
<td>234</td>
</tr>
<tr>
<td>1974</td>
<td>1042</td>
<td>195</td>
</tr>
<tr>
<td>1975</td>
<td>1682</td>
<td>195</td>
</tr>
<tr>
<td>1976</td>
<td>1628</td>
<td>167</td>
</tr>
<tr>
<td>1977</td>
<td>1574</td>
<td>58</td>
</tr>
<tr>
<td>1978</td>
<td>1955</td>
<td>65</td>
</tr>
<tr>
<td>1979</td>
<td>1741</td>
<td>62</td>
</tr>
<tr>
<td>1980</td>
<td>1849</td>
<td>52</td>
</tr>
<tr>
<td>1981</td>
<td>1894</td>
<td>53</td>
</tr>
<tr>
<td>1982</td>
<td>2022</td>
<td>78</td>
</tr>
</tbody>
</table>
TUBERCULOSIS IN FEEDLOT ANIMALS

Mitchell A. Essey*
William L. Searles**

While the numbers of positive tuberculosis cases found on "regular kill" each year since 1978 has remained remarkably constant (range 52-78; average 62), the percentage of cases traced to feedlots has risen steadily. Table I depicts the cases completed by fiscal year. It shows a rise in the percentage of "feedlot" cases from 32% in 1978 to a precipitous 96% in fiscal 1982. Upon considering bovine tuberculosis in U.S. feedlots, it becomes clear that the problem is double pronged. Investigations indicate that most tuberculosis positive feedlot cases either traced back directly (or indirectly) to Mexico, or had been associated with cattle of Mexico origin.

A substantial number of "feedlot" cases are detected in packing houses located in California and Texas. Since 1978, California investigated fifty-nine positive cases. This represented 19% of the U.S. total of 311 cases completed during this period (Table II). Of this total, only one case was an adult animal. All others (93.3%) were traced to feedlots. Of the fifty-eight cases traced to feedlots, all but three were of Mexico origin, or were associated with Mexico imports. Fully 100% of 40 cases initiated or investigated by Arizona during this same period were feedlot cases. Almost all were traceable to Mexico. The data for Texas during this period was essentially the same except that the percentage of feedlot cases was somewhat lower (about 80%).

Historically, the number of lesioned cases detected is low in each affected lot. Usually a single lesioned animal is reported. Occasionally, the occurrence of lesioned cases per lot is substantially higher. Three lots slaughtered in Kansas and Nebraska during 1975-76 had from six to ten lesioned cases each. Those lots were of Holstein steers that traced back directly to Mexico. Outbreaks in small (2-300 head) family-operated feedlots have yielded as high as 60% lesioned cases in domestic cattle, but such cases are rare.

A recent case affecting major feedlots involved a group of 1222 steers assembled in Mexico that entered the United States in November 1979.

---

*Mitchell A. Essey
Regional Epidemiologist
Reno, Nevada

**William L. Searles
Regional Epidemiologist
Austin, Texas

They were pastured in at least two locations in Texas. About June 1981 the group was dispersed and moved to four feedlots; one each in Texas, Oklahoma, New Mexico and Arizona. The first three feedlots yielded twenty-three lesioned cases (2.8%) in 826 animals of this group slaughtered during 1981. The Arizona feedlot had received 350 head described as the "poor doers" of the group. By April 1982, when the slaughter of this lot was completed, twenty-two lesioned cases (6.3%) were found. It was this lot that was largely responsible for the significant increase in "regular kill" tuberculosis cases detected in 1982 (13 slaughter lots with 45 carcasses affected); and that focused attention on the feedlot problem.

Epidemiological reports of feedlot cases have revealed certain areas of risk of variable degree. In the case of Mexico cattle, the usual time span from U.S. entry to slaughter is over 2 years. For most of that period the animals are run on one or more pastures. Epidemiology in most cases is incomplete or absent, especially with regard to adjacent herds that may have become exposed. In one case, an affected group was run on the same ranch with a large breeding herd. The two groups were reportedly "separated" by natural and man-made barriers.

Occasionally, a lesioned case of Mexico origin had been used as a roping steer. The degree of risk here is difficult to access but one can certainly see here the possibility of occasional contact with domestic livestock.

Epidemiological investigations of the feedlot itself are seldom complete and usually terminate with the statement that "all animals in the affected lot have been sent to slaughter." Rarely is there real assurance that no exposure had occurred of "two way" cattle. It can be assumed, but is not commonly documented, that subjects from an affected lot had passed through the "hospital pen" and were later sorted into a different lot that more closely matched in weight. Lesioned cases have traced back to "sick pens" where no effort was made to determine the disposition of other animals exposed in the sick pen during that period.

Recommendations for Handling Outbreaks in Feedlots:

1. The UM&R for Bovine Tuberculosis Eradication states in *Procedures in Affected Herds*, that "cattle in feedlots known to be exposed to tuberculous cattle shall be quarantined and shipped under permit directly to slaughter." The USAHA Committee for Tuberculosis proposed this year (1982) that "feedlots, or portions of feedlots, that contained tuberculous and tuberculosis exposed cattle shall be vacated after movement to slaughter, cleaned and disinfected." When possible, such pens should be left vacant after cleaning and disinfection for a period of 30 days.

2. The discovery of multiple cases from a feedlot should be followed by an immediate attempt to recover identification devices at slaughter from any other animals from that lot that may be found to be lesioned. Positive hide identification is of paramount importance in such animals
because a brand, if present, may be the only type of meaningful identification available.

3. More complete information is needed on each case, with special emphasis on (1) possible pasture contacts prior to entry into the feedlot, and (2) movements within the feedlot with particular regard to possible contacts with cattle destined to other than slaughter.

4. Develop National "high risk" name list in cases involving domestic cattle. Experience has shown that feedlot tracebacks, beyond dealers, to producers, can generate dozens of names of consignors. Names appearing frequently should be investigated.

Recommendations regarding cattle imported from Mexico.

1. Present requirements for U.S. entry have not stemmed the movement of tuberculosis affected steers. The best solution would seem to be the requirement for a post-entry tuberculin test (30-60 days). Should evidence of tuberculosis be disclosed by such test, the group should be handled as an affected herd with all restrictions that apply to domestic herds found infected.

2. The ultimate solution to this problem lies in the eradication of bovine tuberculosis from the country of Mexico. It would seem appropriate at this time for the U.S. to offer resources and technical assistance toward that end for the everlasting mutual benefit of both countries.

SUMMARY

Epidemiologic investigation of tuberculous bovine cases found on "regular kill" in the United States show that the percentage of cases traced to feedlots has risen steadily from 1978 through 1982. Feedlot sources comprised 96% of the seventy-eight slaughter traceback cases completed in 1982. Investigations in Texas, California and Arizona, where most feedlot cases are initiated, provide evidence that most lesioned cases are of Mexico origin. The exposure potential of domestic cattle is discussed, and recommendations are offered for the management of tuberculosis in feedlots and for the conduct of such investigations. Recommendations are also made that pertain to the importation of cattle from Mexico.
Table I

CALIFORNIA POSITIVE "REGULAR KILL" TUBERCULOSIS CASES

<table>
<thead>
<tr>
<th>FY</th>
<th>CASES</th>
<th>% of U.S. TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>U.S.</td>
<td>CA</td>
</tr>
<tr>
<td>78</td>
<td>66</td>
<td>6</td>
</tr>
<tr>
<td>79</td>
<td>62</td>
<td>17</td>
</tr>
<tr>
<td>80</td>
<td>52</td>
<td>10</td>
</tr>
<tr>
<td>81</td>
<td>53</td>
<td>17</td>
</tr>
<tr>
<td>82</td>
<td>78</td>
<td>9</td>
</tr>
<tr>
<td>TOTAL</td>
<td>311</td>
<td>59</td>
</tr>
</tbody>
</table>

Table II.
REPORT OF THE COMMITTEE ON TUBERCULOSIS
AND JOHNE'S DISEASE

Chairman: J. M. Dick, Harrisburg, PA
Vice Chairman: V. P. LaBranche, Boston, MA

J. A. Acree, FL; Don Agresti, CA; L. R. Barnes, IN; C. E. Boyd, SC; M. A. Essey, CA; J. G. Flint, MN; G. H. Frye, MD; H. C. Hairston, MD; G. F. Hoffsis, OH; R. L. Hosker, MD; D. E. Hughes, SD; Sarah B. Hurley, WI; C. A. Lamb, CA; L. L. Larson, WI; A. R. McLaughlin, WI; R. S. Merkal, IA; D. J. Meyers, Canada; H. E. Nadler, NY; W. J. Owen, IA; M. S. Silberman, GA; D. H. Smith, OR; P. O. Smith, CA; G. R. Snyder, VA; P. L. Spencer, IL; Roy Strange, GA; C. D. Stumpff, KS; C. O. Thoen, IA; E. J. Wilson, MD.

Meeting Hyatt Hotel—Tuesday and Wednesday, 1:30 p.m.-5:30 p.m. Suite 6A

The committee met in the afternoon of November 10 and November 11, 1982. Eighteen committee members and 16 guests were in attendance for all or a portion of the meetings.

The following reports presented and discussed will be published in the proceedings.

1. Swine mycobacteriosis—Dr. Helen Acland—University of Pennsylvania School of Veterinary Medicine
2. Decontamination, Media and Culture Methods for Mycobacterium Para-tuberculosis—Dr. Richard Merkal—NADC, Ames, Iowa
3. Dr. Ralph Hosker, APHIS-VS, gave a review of the present TB program status and a case report of the M&M Dairy of Florida
4. A case report on the Peter Finney herd of N.Y. was given by Dr. Harold McCoy—APHIS-VS
5. Tuberculosis in Elk—Epidemiology—Dr. Charles Stumpff APHIS-VS
6. Tuberculosis in Feedlots—Dr. M. A. Essey—APHIS-VS

A report was given by Dr. G. H. Frye on Swine Mycobacteriosis as follows:

There were no formal meetings of the subcommittee during the year. The subcommittee did, however, follow the progress of the Food Safety Inspection Services (FSIS) proposal to modify the time-temperature cooking requirements for tuberculosis swine carcasses.

These changes were based on Dr. Richard Merkal's work in this area and had the endorsement of the tuberculosis Committee.

The proposed amendments to the FSIS regulations would have allowed passed for cooking carcasses to be used in a wider variety of meat products without any compromise with the wholesomeness of the product. Unfortunately, the proposal received uninformed and emotional
criticism in the press prior to publication and was withdrawn and is no longer under consideration.

Two members of the subcommittee (Drs. Snyder and Frye) attended a meeting on mycobacteriosis at the Center for Disease Control (CDC) in Atlanta, Georgia in June. This meeting was proposed by Dr. John Brown, University of Georgia, to consider research that would verify or disprove the appropriateness of current meat inspection requirements on the disposition of tuberculosis swine carcasses. Attending were representatives from LCI, FSIS, Veterinary Services, CDC and the University of Georgia. There was general agreement that the risk to human health from swine with mycobacteriosis was extremely low or none at all. If research could show this, then CDC could reassess the position it took in 1970 (In the absence of information either way, it had assumed there was a risk). This research could result in a change in the Meat Inspection Regulations adopted in 1972 that were based on CDC's decision.

Losses from swine mycobacteriosis are due to the Meat Inspection regulations and not to the effect of the disease on the animal itself, therefore, the committee recommends that research should be encouraged with the active participation of CDC to receive an unbiased evaluation to determine if these regulations are justified from the Public Health standpoint.

In executive session, the following subcommittee reports were presented and discussed:

A. The subcommittee report on Johne's Disease was given by Dr. A. R. McLaughlin

Four members of the subcommittee met on Tuesday, November 8, 1982

1. The subcommittee has developed a sample information and education packet for those states interested in developing a control program and it consists of the following:
   a) A three page field manual for a Johne's disease program.
   b) A three page Johne's disease calfhood vaccination program agreement.
   c) Information sheet for practitioner and training course certification.
   d) Color photo of veterinarians finger that was injected with the vaccine.
   e) Seventeen guidelines for Johne's disease eradication.
   f) Order sheet for vaccine, tags and supplies.
   g) Form letter to go to practitioner assigning him a permit number for vaccine and supplies.
   h) Johne's disease calfhood vaccination report form (original and two copies)
Dr. McLaughlin moved that the subcommittee approve and accept the packet as a suggested packet to be made available to all State Animal Health Officials interested in establishing a Johne's Disease Control Program.

The motion was seconded by Dr. Hoffsis.

Motion carried.

2. The subcommittee received inquiry from Select Sires and the American Guernsey Cattle Club:

The paramount question is the effect of the vaccine on the overall incident of Johne's disease.

Will vaccinations make the identification of positive or carrier animals more difficult than it already is and hence increase the potential for dissemination of Johne's disease through sale of registered cattle and semen for artificial insemination?

Dr. McLaughlin moved that the subcommittee reply to the inquiry as follows: There is no evidence available that indicates that vaccination will increase infection or transmission of the disease; however, the subcommittee is concerned that vaccination could confuse the interpretation of other diagnostic tests for Johne's disease and tuberculosis.

The motion was seconded by Dr. Hoffsis.

Motion carried.

3. Reconsideration of establishing Guidelines for According Private Laboratories to do fecal culturing for M. paratuberculosis:

After discussion the subcommittee recommended that this item be referred to Veterinary Services and that the idea be encouraged that private laboratories become accredited to conduct fecal culturing using the same accepted procedures as state laboratories.

4. Establishment of a standard technique for conducting the intradermal Johnin test for export:

Dr. McLaughlin moved that the subcommittee recommend the following:
Johne's disease intradermal testing for international export will be 0.1 ml of Johnin intradermally in the Caudal fold and read at 72 hours unless the importing country or buyer requests or requires a different test.

The motion was seconded by Dr. Hoffsis.

Motion carried.

5. The subcommittee decided that the full committee should discuss and rule on a revision or deletion of the Parts 71 and 80 of the CFR relating to paratuberculosis.

The full committee action on this proposal resulted in no change at
this time of these parts (71 and 80) of the Code of Federal Regulations.

A motion to accept the subcommittee report was made by Dr. Paul Spencer, seconded by Dr. P. L. Smith.

The motion carried.

B. The subcommittee report on the Uniform Methods and Rules by Dr. Ralph Hosker, APHIS-VS.

The following changes of the UM&R were proposed

Part I — Definitions

Caudal Fold Tuberculin Test — To the present definition add the sentence:

Animals or herds of unknown status will not be subjected to retest at intervals of less than 60 days.

A motion was made by Dr. P. L. Smith and seconded by Dr. V. P. LaBranche to accept this change. The motion was passed.

Part II — Recommended Procedures

J. Procedures in affected herds — Following the sentence, "Cattle in feed lots known to be exposed to tuberculosis cattle shall be quarantined and shipped under permit directly to slaughter," add the sentence: "Feed-lots or portions of feedlots that have contained affected and exposed cattle shall be vacated, cleaned, and disinfected following the removal of such cattle to slaughter."

A motion was made by Dr. M. A. Essey, to accept this change, seconded by Dr. D. H. Smith. The motion was carried.

Part III — Herd and Herd Status Plan

A. Accredited Herd Plan for Cattle:

3(a) Accreditation and reaccreditation. Amend the present sentence which reads, "Qualified herds may be issued a certificate by local State and Federal officials, to read: Qualified herds shall be issued a certificate by the local State and Federal officials and other appropriate information to emphasize the significance of the herd accreditation plan."

A motion to accept the change was made by Dr. Spencer and seconded by Dr. McLaughlan. The motion carried.

3(b) At the end of the present paragraph add the sentence: "To assure adherence to tuberculosis testing procedures and practices, each accredited herd shall be monitored at least once every three years by a State or Federal regulatory veterinarian."

Dr. McLaughlan moved to reject this portion of part III, seconded by Dr. Essey. Motion carried.

Under part III A. Accredited Herd Plan for Cattle, add the following
new title and paragraph. 4. Post mortem Surveillance for Tuberculosis—To qualify for reaccreditation, a herd owner shall furnish evidence showing that 5 percent of the adult herd has been subjected to a post mortem examination each year. Such evidence shall provide documentation of the sale of cull animals to regular slaughter, post mortem certification by an accredited veterinarian of on farm death losses, or the combination of the two.

A motion to accept this recommendation was made by Dr. L. R. Burnes, was seconded by Dr. LaBranche. The motion was defeated.

A discussion was held regarding the permanent individual identification of feeder cattle imported from Mexico to support our domestic bovine tuberculosis eradication program. Present import requirements seem adequate. However, we are concerned about native cattle being exposed to cattle from Mexico that may have been exposed to tuberculosis infected cattle prior to entry. There have been confirmed reports of tuberculosis lesions appearing in Mexico cattle.

A subcommittee, chaired by Dr. Essey, was formed to check on import requirements and to verify the extent of the problem. Iowa State University has been contracted by VS to develop an identification method to use on foreign imports.

A resolution was presented to the committee by the North Central USAHA as follows: "Whereas, the entire United States has been declared a modified accredited area for tuberculosis and whereas, the unnecessary expenses of tuberculosis testing for all primary moves of cattle interstate seems excessive and whereas, a minimum number of infected and exposed herds have been uncovered by the movement testing procedure—the North Central United States Animal Health Association strongly recommends that state regulatory officials consider to support the premise that tuberculin testing (M bovis) for primary interstate movement of cattle from herds of origin be abandoned and that tuberculosis in cattle be classified as an emergency disease by APHIS-VS-U.S.D.A."

Dr. John Atwell APHIS-VS and Dr. Ralph Hosker, APHIS-VS discussed the criteria of the disease and whether or not it fit the category of an emergency disease.

Dr. Spencer moved to accept the resolution, seconded by Dr. D. Smith. The motion was defeated.

A motion was made by Dr. D. H. Smith to discontinue the requirement for tuberculin testing of goats for interstate or intrastate movement except for accreditation and export. The motion was seconded by Dr. Spencer. After some discussion, the motion passed.

The committee strongly recommends continued funding by APHIS-VS to achieve eradication of tuberculosis, to support continued research for the development of better test procedures for the detection of tuberculosis and paratuberculosis and to provide adequate indemnity for
reactors and herd depopulation. A resolution has been submitted relative to the decrease in indemnity funding for the 1983 budget.

Dr. Barnes reported on a court case involving a herd owners lawsuit against a veterinarian for alleged misreading of the tuberculin test. The accredited herd was depopulated following a negative herd test when reactors and lesion animals were discovered at a later date. The suit charged the veterinarian with improper reading of the test because he used a glove and the owner claimed the time in reading was inadequate. The veterinarian after several years of litigation was found not guilty by jury trial.

Meeting adjourned 5:00 PM, November 10, 1982.
INTRODUCTION

Malignant catarrhal fever (MCF) is a sporadic, highly fatal viral disease of cattle and several species of wild ruminants, characterized by fever, leukopenia, profuse nasal discharge, ophthalmitis, corneal opacity, lymphadenopathy, and mucosal erosions of the upper digestive tract. Microscopically, a multifocal vasculitis and lymphoreticular proliferation are consistent pathognomonic features of MCF (18, 33, 37, 40, 68).

While episodes of MCF in cattle in the United States, Europe and Australia are usually sporadic with low morbidity, a few outbreaks of epidemic proportion have been reported in the United States (14, 21, 29, 36).

In East Africa's Masai-lands, the seasonal occurrence of MCF usually parallels the calving period of wildebeest and has been reported to be responsible for approximately 7% annual mortality of cattle in this area (26, 28).

While Masai herdsmen recognized an association between calving wildebeest and the occurrence of MCF in their cattle for many decades, it was not until 1960 that the etiologic agent was identified as a cell-associated herpesvirus by Plowright, who isolated the virus (MCFV) from a blue wildebeest, Connochaetes taurinus (38, 40).

Malignant catarrhal fever also occurs sporadically in domestic cattle in the United States, Europe, Asia and Australia. It has been epidemiologically associated with affected cattle having had contact with domestic sheep, especially at the time of lambing. The etiologic agent for this so-called "sheep-associated" MCF has not been clearly established (14, 36). A recent report by Rossiter (48) provides serologic evidence that domestic sheep may be infected with a virus related to the wildebeest-origin MCFV. He detected antibodies to MCFV, strain WC II by indirect immunofluorescence in 162 of 167 sheep sera obtained from five United Kingdom, one Austrian, on Australian, and two Kenyan flocks. To date, however, a virus responsible for "sheep-associated" MCF has not been isolated from sheep, or affected cattle except in one reported instance (14).

Domestic rabbits and hares are experimentally susceptible to MCFV (alcelaphine strain) infection and respond with clinical signs and pathologic lesions similar to those seen in ruminants (3, 12, 19). Natural infection of lagomorphs has, however, not been documented.
Various wild ruminant species are also susceptible to the alcelaphine herpes-MCFV (designated by some as bovine herpesvirus-3; 1, 2, 4, 6-8, 11, 16, 22, 26-28, 31, 32, 40, 42, 45, 51, 53, 56, 58, 60, 61, 63, 66-69). Species reported to have been affected by MCFV are listed in Table 3.

The first reported instances of MCF in captive wild ruminants in the USA occurred in white-tailed deer at a zoo in New Jersey and Connecticut in 1971 and 1972 (64). Diagnosis was based upon pathognomonic histopathologic lesions.

MCF was diagnosed in three greater kudus (*Tragelaphus strepsiceros*) at the St. Louis Zoo in February, 1972 (2). These were among nine kudus housed in the same building with a white-tailed gnu (wildebeest; *Connochaetes gnu*) which gave birth on November 22, 1971.

In September, 1974, a Javan banteng (*Bos javanicus javanicus*) died of MCF at the San Diego Zoo (SDZ). Subsequently in 1976 MCF was diagnosed in an Indian gaur (*Bos gaurus*) and two more banteng at the SDZ, and one banteng at the San Diego Wild Animal Park. Sixteen additional MCF cases occurred at the Wild Animal Park (WAP) between July, 1976 and January 1979 involving: banteng, barasingha deer, Pere David’s deer, Arabian oryx, sika deer, Eld’s deer and blackbuck. No virus isolations were made from these cases and the diagnosis was based on clinical signs and pathognomonic lesions (16).

In October, 1979 and January, 1980 MCF was diagnosed in an Indian gaur and greater kudu at the Oklahoma City Zoo (4). Herpesviruses were isolated from these animals and serial experimental transmission of MCFV to domestic cattle and white-tailed deer was accomplished with blood from the gaur and greater kudu. Serologic, morphologic and cytopathic characteristics of the virus isolated from gaur and kudu indicated that each isolate was related to the wildebeest-derived strain of MCFV (4, 5, 64, 68).

In October, 1981, we initiated an investigation of the history of MCF at the San Diego Zoo and Wild Animal Park and began to attempt virus isolations from clinical MCF cases and from asymptomatic wildebeest, topi and hartebeest (all alcelaphine antelope). The chronologic history of MCF cases at the Zoo and Park is shown and summarized in Table 1. Virus isolation results are tabulated in Table 2.

A total of 50 cases have occurred between 1974 and October 1, 1982. Forty cases were at the WAP and 10 at the Zoo, and involved 15 species. Four of these were African species which do not share habitat in nature with alcelaphine antelope species. Eleven species involved were Asian in origin. Viruses identified as MCFV, alcelaphine strain, by their characteristic CPE, indirect immunofluorescence and virus neutralization have been isolated in our laboratory from four clinical cases of MCF, and from one wildebeest carrier calf (Table 2).

Since initiating our studies on MCF, we have learned that many other zoos and wild animals parks in the U.S., Europe, Africa, Asia, and
Australia have been experiencing sporadic cases of MCF in captive wild ruminants. It appears that the incidence of this disease in captive zoo animals has either increased in recent years, or is being more readily diagnosed and differentiated from other similar diseases such as bovine viral diarrhea-mucosal disease, bluetongue and epizootic hemorrhagic disease. MCF has become a serious problem resulting not only in the loss of many highly endangered wild ruminant species but continues as an important threat to their successful captive propagation and maintenance. This is a matter of grave concern for those interested in the preservation of endangered species.

However, there is an even greater concern with regard to MCF; the potential hazard for the domestic cattle industry. Although MCF has generally occurred as sporadic cases among cattle, occasional large scale outbreaks (epizootics) have been reported (14, 21, 29, 36). Because of its potential for impact on domestic livestock, there is an urgent need for a better understanding of the epidemiology of MCF. Circumstantial evidence has suggested that captive wild ruminants might have been a potential source of MCFV which resulted in a series of MCF cases in a dairy located in the vicinity of a wild animal park (21, 29). In this instance a total of 15 cumulative sporadic cases occurred during the winters of 1974 to 1975 and 1975 to 1976 (21). To our knowledge, no additional cases have occurred on this dairy since.

Serious episodes of MCF in domestic cattle in the U.S. have, however, been more frequently associated with contact of involved cattle with domestic sheep (14, 36). The relationship of sheep-associated MCF to alcelaphine herpesvirus-caused MCF, implied by the report of Rossiter (48), and the report by Hamdy et al. of MCF in a Minnesota dairy in 1977 (14), must be more thoroughly studied and defined. A bovine herpesvirus isolated from sheep (62) needs further examination to ascertain its possible relationship to MCFV of alcelaphine origin.

Important questions which must be answered in regard to the epidemiology of MCF include:

1. What other species of animals are reservoirs of MCFV and capable of shedding infectious virus?
   a. All Alcelaphinae? All Caprinae and Ovinae?
   b. Other ruminants?
   c. Non-ruminants (e.g., Lagomorpha)?

2. Are all animals with MCF serum antibodies carriers of MCFV? Are they all capable of shedding infectious virus, or are some, like cattle, dead end hosts which apparently do not shed cell-free (transmissible) virus?

3. Do arthropod or avian species act as vectors and/or reservoirs?

4. Is the, as yet unidentified, sheep-associated MCF agent a variant herpesvirus related to the alcelaphine herpesviruses (bovine her-
pesivirus-3), an entirely different agent; or is this a disease syndrome with multiple etiologic agents?

5. What are the stability or survival capabilities of alcelaphine MCFV under various environmental conditions? Will standard disinfection procedures eliminate MCFV in a contaminated environment?

6. What is the relationship biochemically and antigenically of alcelaphine MCFV to other herpesviruses, especially of cattle? To what extent will they crossreact in serologic procedures?

7. To what extent is there biochemical and genetic variability among alcelaphine MCFV strains?

8. What are the modes of transmission of MCFV?

9. Since wildebeest, topi and hartebeest have been documented as carriers of MCF, are other members of this group also (i.e., blesbok, bontebok, Hunter's antelope, etc.)?

How to prevent the transmission of MCFV from domestic sheep and wild ruminant carrier-reservoirs to domestic cattle, very valuable and endangered captive and free-living wild ruminants is a problem confronting us now. Sound approaches to the control of the MCF hazard must be based upon the best knowledge available, and not be characterized by over-reaction or inaction.

Based upon our current knowledge on MCF, the following policies are suggested to reduce the risk of spread of MCF among zoo animals, to domestic cattle and free-living wildlife:

1. Regulation by state or federal animal health authorities of the movement from or to any zoos of antelope of the subfamily Alcelaphinae (i.e., wildebeest, topi, hartebeest).

2. Restrict the issuance of permits for movement of and placement of alcelaphine antelope to USDA approved zoos and parks. Do not allow animals of this subfamily to be acquired by private individuals, ranches, game farms or any similar facilities where they might expose domestic cattle, sheep or goats or indigenous wildlife such as deer, elk, bison, etc.

3. Only allow movement of non-pregnant alcelaphine antelope over four months of age, since current information indicates that only wildebeest less than four months old and near term pregnant females will shed cell-free (infectious) MCFV.

4. Institute a voluntary MCF antibody testing program for all captive wild ruminants intended for movement from any zoos or parks where MCF has occurred or alcelaphine antelope are present. Inform prospective recipients of such animals of test results so that they may make a decision on whether or not to take delivery, or determine where the animals will be located if accepted.

5. Premises receiving wild ruminants from other facilities where
alcelaphine antelope are held should quarantine new arrivals for 60 days. They should determine their serologic status with respect to MCF antibody upon arrival and at the completion of 60 days in quarantine.

6. While there is no evidence at present that non-alcelaphine antelope, deer, bovine or wild sheep and goat species seropositive for MCF antibodies can transmit infectious MCFV to other susceptible ruminants, it is recommended that any ruminants seropositive for MCF antibodies be segregated from seronegative animals. Hygienic and sanitary precautions should be followed in handling such seropositive animals to preclude possible indirect exposure of susceptible animals.

RESEARCH IN PROGRESS AND FUTURE RESEARCH OBJECTIVES

The need for research on MCF to answer some of the foregoing important questions is obvious. The research program on MCF at the San Diego Zoo has become one of our major commitments.

As mentioned previously, retrospective study has been made of all cases of MCF which have occurred at the San Diego Zoo and Wild Animal Park. Findings are summarized in Table 1.

Since initiating attempts to isolate MCFV from clinical cases and from alcelaphine antelopes, i.e., wildebeest (gnu), topi, and hartebeest, we have made five isolations of MCFV (Tables 1 and 2): One nilgai antelope, two sika deer, and one axis deer (all young animals with clinical MCF); and from one white-tailed gnu calf with which the three deer above were directly in contact in the same holding facility. The identity of each MCFV isolate has been confirmed by virus neutralization and the indirect immunofluorescence test (IIF) using anti-MCFV (WC II) hyperimmune bovine serum supplied to us by the Plum Island Animal Disease Center. Each of the five MCFV isolates is being serially passaged in fetal aoudad kidney (FAK) cells to attempt to increase the titer of cell-free virus. It is interesting that in the two above animals showing the most severe clinical signs and gross lesions (one nilgai and one axis deer) a non-cytopathogenic bovine viral diarrhea virus (BVDV) was also present and isolated along with MCFV. This suggests that BVDV coinfection exalts the pathogenicity of MCFV.

We have completed duplicate tests to determine if sera containing antibodies to bovine herpesviruses 1, 2 and 4 will react with MCFV antigens in the indirect immunofluorescence test as currently conducted at the National Veterinary Services Laboratory. Results are shown in Table 4. Crossreactivity occurred at 1:20 and 1:40 dilutions among all four bovine herpesvirus antisera. It is apparent therefore, that the test as currently conducted, i.e., test sera diluted 1:20 for the IIF test, will result in false positive reactions for MCF antibody. It is therefore suggested
that the test should be conducted at dilutions of 1:20 and 1:100 to provide a more specific assessment of MCF antibody.

The virus neutralization test does not show a similar crossreactivity among the four bovine herpesviruses, based upon preliminary studies conducted in our laboratory. This test would therefore be the test of choice to determine if animals are seropositive for MCF antibodies.

We have set the following research objectives for our continuing program on MCF:

1. To ascertain and develop the most reliable, rapid and specific laboratory procedures to diagnose MCF, confirm virus identity, and identify MCF seropositive animals (potential reservoir/carryer). (Study currently in progress.)

2. To determine the prevalence and species distribution of MCF antibodies among captive wild ruminants in U.S. zoos and in domestic sheep. (Study currently in progress.)

3. To compare alcelaphine herpesvirus strains with each other and with other ruminant herpesviruses by molecular and immunologic methods. (Study currently in progress.)

4. To determine the ecology, modes of transmission (including arthropod), and environmental survival characteristics of alcelaphine MCFV.

5. To determine if animals seropositive for MCF will recrudesce latent virus upon simulated stress.

6. To develop a safe and efficacious inactivated vaccine against MCF for use in animals at risk of exposure.

It is our hope that agencies and individual donors will recognize the urgency of the above described research needs and objectives such that necessary funding for this work may be realized.
<table>
<thead>
<tr>
<th>Date</th>
<th>Path#</th>
<th>Age</th>
<th>Specie(s)</th>
<th>I.D.</th>
<th>Sex</th>
<th>Diagnosis*</th>
<th>Zoo or WAP</th>
<th>Origin</th>
</tr>
</thead>
<tbody>
<tr>
<td>09/07/74</td>
<td>08921</td>
<td>0/3</td>
<td>Javan banteng <em>(Bos javanicus)</em></td>
<td>&quot;Buttercup&quot;</td>
<td>F</td>
<td>MCF-Clin,Path</td>
<td>ZOO</td>
<td>Born @ SDZ</td>
</tr>
<tr>
<td>07/25/76</td>
<td>10890</td>
<td>0/3</td>
<td>Javan banteng <em>(Bos javanicus)</em></td>
<td>176179</td>
<td>F</td>
<td>MCF-Clin,Path</td>
<td>ZOO</td>
<td>Born @ SDZ</td>
</tr>
<tr>
<td>07/26/76</td>
<td>10893</td>
<td>5/0</td>
<td>Indian gaur <em>(Bos gaurus)</em></td>
<td>173020</td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>ZOO</td>
<td>Born @ OK City Zoo 07/01/71. Rec'd SDZ 02/19/73</td>
</tr>
<tr>
<td>08/02/76</td>
<td>10911</td>
<td>11/0</td>
<td>Javan banteng <em>(Bos javanicus)</em></td>
<td></td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>ZOO</td>
<td>Born @ Phila. Zoo</td>
</tr>
<tr>
<td>01/10/77</td>
<td>11392</td>
<td>6/0</td>
<td>Javan banteng <em>(Bos javanicus)</em></td>
<td>171013</td>
<td>F</td>
<td>MCF-Clin,Path</td>
<td>ZOO</td>
<td>Born @ SDZ 01/31/71</td>
</tr>
<tr>
<td>02/18/77</td>
<td>11471</td>
<td>1/6</td>
<td>Javan banteng <em>(Bos javanicus)</em></td>
<td>175143</td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>ZOO</td>
<td>Born @ SDZ 06/10/75</td>
</tr>
<tr>
<td>10/21/77</td>
<td>12280</td>
<td>0/3</td>
<td>Barasingha <em>(Cervus duvauceli)</em></td>
<td>&quot;Boysen&quot;</td>
<td>F</td>
<td>MCF-Clin,Path</td>
<td>WAP (ACC)</td>
<td>Born WAP 07/14/77</td>
</tr>
<tr>
<td>11/03/77</td>
<td>12321</td>
<td>0/3</td>
<td>Barasingha <em>(Cervus duvauceli)</em></td>
<td>WAP 04</td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>WAP (ACC)</td>
<td>Born WAP 07/08/77</td>
</tr>
<tr>
<td>11/16/77</td>
<td>12360</td>
<td>0/5</td>
<td>Formosan sika deer <em>(Cervus nippon)</em></td>
<td>WAP 12</td>
<td>F</td>
<td>MCF-Clin</td>
<td>WAP (ACC)</td>
<td>06/19/77</td>
</tr>
<tr>
<td>01/17/78</td>
<td>12595</td>
<td>0/4</td>
<td>Barasingha <em>(Cervus duvauceli)</em></td>
<td>WAP 08</td>
<td>M</td>
<td>Inoc 2 100 ml</td>
<td>WAP (ACC)</td>
<td>Born WAP 08/09/77</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>blood from sika (WAP 12); MCF, Path 11/16/77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Date</td>
<td>Path#</td>
<td>Age yr/mo</td>
<td>Species</td>
<td>I.D.</td>
<td>Sex</td>
<td>Diagnosis-*</td>
<td>ZOO or WAP</td>
<td>Origin</td>
</tr>
<tr>
<td>----------</td>
<td>---------</td>
<td>-----------</td>
<td>----------------------------------------------</td>
<td>--------</td>
<td>-----</td>
<td>-----------------</td>
<td>------------</td>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>09/07/78</td>
<td>13312</td>
<td>3/5</td>
<td>Slender-horned gazella (<em>Gazella leptoceros</em>)</td>
<td>WAP 21</td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>WAP</td>
<td>Born WAP 04/07/75</td>
</tr>
<tr>
<td>11/16/78</td>
<td>13483</td>
<td>3/8</td>
<td>Javan banteng (<em>Bos javanicus</em>)</td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>WAP</td>
<td>Born SDZ 03/12/75; Rec'd at WAP 07/27/75</td>
<td></td>
</tr>
<tr>
<td>12/26/78</td>
<td>13585</td>
<td>6/0</td>
<td>Javan banteng (<em>Bos javanicus</em>)</td>
<td>172003</td>
<td>F</td>
<td>MCF-Clin,Path</td>
<td>ZOO</td>
<td>Born SDZ 11/72</td>
</tr>
<tr>
<td>01/02/79</td>
<td>13598</td>
<td>1/2</td>
<td>Burmese thamin (<em>Elvi's deer; Cervus eldi thamin</em>)</td>
<td>WAP 03</td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>WAP</td>
<td>Born WAP 10/14/77</td>
</tr>
<tr>
<td>01/09/79</td>
<td>13626</td>
<td>1/9</td>
<td>Pere David's deer (<em>Elaphurus davidianus</em>)</td>
<td>WAP 04</td>
<td>F</td>
<td>Unident. RNA virus + MFC-Path, Clin</td>
<td>WAP</td>
<td>Rec'd from Front Royal, VA 05/78</td>
</tr>
<tr>
<td>01/09/79</td>
<td>13622</td>
<td>5/2</td>
<td>Javan banteng (<em>Bos javanicus</em>)</td>
<td>SDZ 7</td>
<td>F</td>
<td>MCF-Clin,Path</td>
<td>WAP</td>
<td>Born SDZ 11/19/73</td>
</tr>
<tr>
<td>01/24/79</td>
<td>13668</td>
<td>8/6</td>
<td>Arabian oryx (<em>Oryx leucoryx</em>)</td>
<td>WAP 13</td>
<td>F</td>
<td>MCF-Path, (?)</td>
<td>WAP</td>
<td>Born Phoenix Zoo 06/21/70</td>
</tr>
<tr>
<td>11/15/79</td>
<td>14761</td>
<td>1/5</td>
<td>Blackbuck (<em>Antilope cervicapra</em>)</td>
<td>F</td>
<td>MCF-Path</td>
<td>WAP</td>
<td>Born WAP 06/22/78</td>
<td></td>
</tr>
<tr>
<td>11/19/79</td>
<td>14774</td>
<td>3/6</td>
<td>Blackbuck (<em>Antilope cervicapra</em>)</td>
<td>WAP 09</td>
<td>F</td>
<td>MCF-Path, (?)</td>
<td>WAP</td>
<td>Born WAP 06/24/76</td>
</tr>
<tr>
<td>12/04/79</td>
<td>14820</td>
<td>6/8</td>
<td>Luzon sambar deer (<em>Cervus mariannus</em>)</td>
<td>WAP 348</td>
<td>F</td>
<td>MCF-Path</td>
<td>WAP</td>
<td>Born WAP 03/16/73</td>
</tr>
<tr>
<td>05/12/80</td>
<td>15242</td>
<td>Adult</td>
<td>Pere David's deer (<em>Elaphurus davidianus</em>)</td>
<td>WAP 10</td>
<td>M</td>
<td>MCF-Clin, Path (?)</td>
<td>WAP</td>
<td>Rec'd WAP 03/28/80</td>
</tr>
<tr>
<td>05/13/80</td>
<td>15249</td>
<td>1/0</td>
<td>Pere David's deer (<em>Elaphurus davidianus</em>)</td>
<td>WAP 05</td>
<td>M</td>
<td>MCF-Clin, Path (?)</td>
<td>WAP</td>
<td>Born WAP 05/20/79</td>
</tr>
<tr>
<td>Date</td>
<td>Path#</td>
<td>Age yr/mo</td>
<td>Species</td>
<td>I.D.</td>
<td>Sex</td>
<td>Diagnosis*</td>
<td>ZOO or WAP</td>
<td>Origin</td>
</tr>
<tr>
<td>----------</td>
<td>-------</td>
<td>-----------</td>
<td>-----------------------------------</td>
<td>------</td>
<td>-----</td>
<td>------------</td>
<td>-------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>05/17/80</td>
<td>15264</td>
<td>7/0</td>
<td>Javan banteng (Bos javanicus)</td>
<td>WAP 01</td>
<td>M</td>
<td>MCF-Clin, Path</td>
<td>WAP (As Swp)</td>
<td>Born WAP 04/27/73</td>
</tr>
<tr>
<td>05/18/80</td>
<td>15275</td>
<td>2/0</td>
<td>Pere David's deer (Elaphurus davidianus)</td>
<td>WAP 17</td>
<td>F</td>
<td>MCF-Clin, Path</td>
<td>WAP (As Swp)</td>
<td>Born 04/13/78 Rec'd WAP 09/01/78</td>
</tr>
<tr>
<td>05/21/80</td>
<td>15292</td>
<td>2/0</td>
<td>Pere David's deer (Elaphurus davidianus)</td>
<td>WAP 02</td>
<td>F</td>
<td>MCF-Clin, Path</td>
<td>WAP (As Swp)</td>
<td>Born WAP 04/08/78</td>
</tr>
<tr>
<td>06/01/80</td>
<td>15336</td>
<td>4/11</td>
<td>Javan banteng (Bos javanicus)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Born St. Louis Zoo 07/02/75. Rec'd WAP 03/04/78</td>
</tr>
<tr>
<td>06/10/80</td>
<td>15371</td>
<td>6/0</td>
<td>Pere David's deer (Elaphurus davidianus)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Born WAP 05/02/74</td>
</tr>
<tr>
<td>06/14/80</td>
<td>15391</td>
<td>1/3</td>
<td>Javan banteng (Bos javanicus)</td>
<td>WAP 03</td>
<td>M</td>
<td>MCF-Clin, Path</td>
<td>WAP (As Swp)</td>
<td>Born WAP 03/29/79</td>
</tr>
<tr>
<td>07/25/80</td>
<td>15557</td>
<td>&gt;10yr</td>
<td>Pere David's deer (Elaphurus davidianus)</td>
<td>WAP 03</td>
<td>F</td>
<td>MCF-Clin, Path</td>
<td>WAP (As Swp)</td>
<td>Rec'd WAP 09/17/70</td>
</tr>
<tr>
<td>08/07/80</td>
<td>15553</td>
<td>3/2</td>
<td>Pere David's deer (Elaphurus davidianus)</td>
<td>18R 20L</td>
<td>F</td>
<td>MCF-Clin, Path</td>
<td>WAP (As Swp)</td>
<td>Rec'd from NZP 05/04/78 breeding loan</td>
</tr>
<tr>
<td>08/16/80</td>
<td>15560</td>
<td>4/2</td>
<td>Pere David's deer (Elaphurus davidianus)</td>
<td>L13 R15</td>
<td>F</td>
<td>MCF-Clin, Path</td>
<td>WAP (As Swp)</td>
<td>Born WAP 06/11/77</td>
</tr>
<tr>
<td>08/30/80</td>
<td>15644</td>
<td>4/0</td>
<td>Pere David's deer (Elaphurus davidianus)</td>
<td>WAP 01</td>
<td>F</td>
<td>MCF-Clin, Path</td>
<td>WAP (As Swp)</td>
<td>Born WAP 05/17/76</td>
</tr>
<tr>
<td>Date</td>
<td>Path#</td>
<td>Age yr/mo</td>
<td>Species</td>
<td>I.D.</td>
<td>Sex</td>
<td>Diagnosis*</td>
<td>ZOO or WAP</td>
<td>Origin</td>
</tr>
<tr>
<td>----------</td>
<td>-------</td>
<td>-----------</td>
<td>----------------------------------------------</td>
<td>---------</td>
<td>-----</td>
<td>------------</td>
<td>------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>10/16/80</td>
<td>15764</td>
<td>6/0</td>
<td>Bongo (Taurotragus eurycerus isaaci)</td>
<td>176185</td>
<td>M</td>
<td>MCF-Path (?)</td>
<td>ZOO</td>
<td>Rec'd from Kenya 06/24/76</td>
</tr>
<tr>
<td>10/29/80</td>
<td>15802</td>
<td>5/0</td>
<td>Eld's deer (Cervus eldi thamin)</td>
<td>WAP 11</td>
<td>M</td>
<td>MCF-Path (?)</td>
<td>WAP (So Afr)</td>
<td>Born WAP 10/16/75</td>
</tr>
<tr>
<td>11/07/80</td>
<td>15828</td>
<td>0/11</td>
<td>Bongo (Taurotragus eurycerus isaaci)</td>
<td>179338</td>
<td>F</td>
<td>MCF-Path (?)</td>
<td>ZOO (CZ)</td>
<td>Born SHZ 11/24/79</td>
</tr>
<tr>
<td>11/30/80</td>
<td>15879</td>
<td>1/4</td>
<td>Formosan sika deer (Cervus nippon)</td>
<td>WAP 187</td>
<td>F</td>
<td>MCF-Clin,Path</td>
<td>WAP (Afr Sup)</td>
<td>Born WAP 7/29/79</td>
</tr>
<tr>
<td>10/04/81</td>
<td>17106</td>
<td>0/2</td>
<td>Axis deer (Cervus axis)</td>
<td>WAP 38</td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>WAP (ACC)</td>
<td>Born WAP 7/14/81</td>
</tr>
<tr>
<td>10/05/81</td>
<td>17108</td>
<td>0/2</td>
<td>Barasingha (Cervus duvauceli)</td>
<td>WAP 61</td>
<td>F</td>
<td>MCF-Clin,Path</td>
<td>WAP (An Pins)</td>
<td>Born WAP 7/17/81</td>
</tr>
<tr>
<td>12/07/81</td>
<td>17303</td>
<td>Juv.</td>
<td>Nilgai (Boselaphus tragocamelus)</td>
<td>WAP 34</td>
<td>M</td>
<td>MCF-Clin,Path VI</td>
<td>WAP (ACC)</td>
<td>Burn WAP</td>
</tr>
<tr>
<td>12/09/81</td>
<td>17308</td>
<td>0/5</td>
<td>Barasingha (Cervus duvauceli)</td>
<td>WAP 34</td>
<td>M</td>
<td>MCF-Clin,Path</td>
<td>WAP (ACC)</td>
<td>Burn WAP 7/14/81</td>
</tr>
<tr>
<td>02/15/82</td>
<td>17509</td>
<td>2/0</td>
<td>Zulu suni (Nasotragus moschatus zulencia)</td>
<td>D2025</td>
<td>M</td>
<td>MCF-Path</td>
<td>ZOO</td>
<td>Bn. Dallas 1/1/80</td>
</tr>
<tr>
<td>05/13/82</td>
<td>17875</td>
<td>2/8</td>
<td>Nilgiri tahr (Hemitragus hylocrius)</td>
<td>WAP 08</td>
<td>M</td>
<td>MCF-Path (?)</td>
<td>WAP (Afr Sup)</td>
<td>Born WAP 9/1/79</td>
</tr>
<tr>
<td>07/30/82</td>
<td></td>
<td>0/1</td>
<td>Formosan sika deer (Cervus nippon)</td>
<td>WAP 43</td>
<td>M</td>
<td>MCF-VI, Clin</td>
<td>WAP (ACC)</td>
<td>Burn WAP</td>
</tr>
<tr>
<td>08/11/82</td>
<td>18371</td>
<td>2 wks</td>
<td>Axis deer (Cervus axis)</td>
<td>WAP 231</td>
<td>F</td>
<td>MCF-Clin,Path VI</td>
<td>WAP (ACC)</td>
<td>born WAP 7/25/82</td>
</tr>
<tr>
<td>08/11/82</td>
<td>18372</td>
<td>0/1</td>
<td>Barasingha (Cervus duvauceli)</td>
<td>WAP 50</td>
<td>M</td>
<td>MCF-VI, Clin, Path</td>
<td>WAP (ACC)</td>
<td>Burn WAP 7/10/82</td>
</tr>
<tr>
<td>Date</td>
<td>Path#</td>
<td>Age</td>
<td>Species</td>
<td>I.D.</td>
<td>Sex</td>
<td>Diagnosis-#</td>
<td>ZOO or WAP</td>
<td>Origin</td>
</tr>
<tr>
<td>------------</td>
<td>--------</td>
<td>-----</td>
<td>--------------------------</td>
<td>------</td>
<td>-----</td>
<td>-------------</td>
<td>------------</td>
<td>-------------</td>
</tr>
<tr>
<td>08/11/82</td>
<td>1837W</td>
<td>0/1</td>
<td>Formosan sika deer (Cervus nippon)</td>
<td>WAP 44</td>
<td>F</td>
<td>MCF-VI, Clin, Path</td>
<td>WAP (ACC)</td>
<td>Born WAP</td>
</tr>
<tr>
<td>08/22/82</td>
<td>18412</td>
<td>3/8</td>
<td>Indian gaur (Bos gaurus)</td>
<td>WAP 05</td>
<td>M</td>
<td>MCF-Path</td>
<td>WAP (As Swp)</td>
<td>Born WAP</td>
</tr>
<tr>
<td>08/22/82</td>
<td>18415</td>
<td>0/4</td>
<td>Axis deer (Cervus axis)</td>
<td>WAP 219</td>
<td>F</td>
<td>MCF-VI, Clin, Path</td>
<td>WAP (ACC)</td>
<td>Born WAP</td>
</tr>
<tr>
<td>09/27/82</td>
<td>18539</td>
<td>0/5</td>
<td>Axis deer (Cervus axis)</td>
<td>WAP 220</td>
<td>F</td>
<td>MCF-Clin, Path</td>
<td>WAP (ACC)</td>
<td>Born WAP</td>
</tr>
<tr>
<td>10/01/82</td>
<td>18558</td>
<td>0/5</td>
<td>Axis deer (Cervus axis)</td>
<td>WAP 222</td>
<td>F</td>
<td>MCF-Clin</td>
<td>WAP (ACC)</td>
<td>Born WAP</td>
</tr>
</tbody>
</table>

**SUMMARY - Total by Species**

<table>
<thead>
<tr>
<th>Species</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>11 Banteng</td>
<td></td>
</tr>
<tr>
<td>2 Guur</td>
<td></td>
</tr>
<tr>
<td>6 Harasingha</td>
<td></td>
</tr>
<tr>
<td>1 Slender-horned gazelle</td>
<td></td>
</tr>
<tr>
<td>1 Nilgiri tahr</td>
<td></td>
</tr>
<tr>
<td>4 Formosan sika deer</td>
<td></td>
</tr>
<tr>
<td>2 Eld's deer</td>
<td></td>
</tr>
<tr>
<td>10 Pere David's deer</td>
<td></td>
</tr>
<tr>
<td>1 Arabian oryx</td>
<td></td>
</tr>
<tr>
<td>1 Zulu sunit</td>
<td></td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>50</td>
</tr>
</tbody>
</table>

**Totals by Location**

<table>
<thead>
<tr>
<th>Location</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>San Diego Zoo</td>
<td>1</td>
</tr>
<tr>
<td>WAP (As Swp)</td>
<td>19</td>
</tr>
<tr>
<td>WAP (As Plns)</td>
<td>1</td>
</tr>
<tr>
<td>WAP (ACC)</td>
<td>16</td>
</tr>
<tr>
<td>WAP (So Afr)</td>
<td>1</td>
</tr>
<tr>
<td>WAP (unspecified)</td>
<td>3</td>
</tr>
</tbody>
</table>

**Totals by Year**

<table>
<thead>
<tr>
<th>Year</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>1974</td>
<td>1</td>
</tr>
<tr>
<td>1975</td>
<td>0</td>
</tr>
<tr>
<td>1976</td>
<td>3</td>
</tr>
<tr>
<td>1977</td>
<td>5</td>
</tr>
<tr>
<td>1978</td>
<td>4</td>
</tr>
<tr>
<td>1979</td>
<td>7</td>
</tr>
<tr>
<td>1980</td>
<td>16</td>
</tr>
<tr>
<td>1981</td>
<td>4</td>
</tr>
<tr>
<td>1982</td>
<td>10</td>
</tr>
</tbody>
</table>

**Abbreviations**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Meaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACC</td>
<td>Animal Care Center</td>
</tr>
<tr>
<td>As Swp</td>
<td>Asian Swamp</td>
</tr>
<tr>
<td>As Plns</td>
<td>Asian Plains</td>
</tr>
<tr>
<td>So Afr</td>
<td>South Africa</td>
</tr>
<tr>
<td>WAP</td>
<td>Wild Animal Park</td>
</tr>
<tr>
<td>Accession No.</td>
<td>Date</td>
</tr>
<tr>
<td>--------------</td>
<td>------------</td>
</tr>
<tr>
<td>DX 211-81</td>
<td>Dec., 1981</td>
</tr>
<tr>
<td>DX 11-82</td>
<td>June, 1982</td>
</tr>
<tr>
<td>DX 16-82</td>
<td>July, 1982</td>
</tr>
<tr>
<td>DX 19-82</td>
<td>July, 1982</td>
</tr>
<tr>
<td>DX 21-82</td>
<td>Aug., 1982</td>
</tr>
</tbody>
</table>

* Isolations made in fetal auodad kidney cells, identified as MCFV by cytopathology, indirect immunofluorescence, and virus neutralization with MCFV-WCII strain antiserum provided by the Plum Island Animal Disease Center.

**BVDV (noncytopathogenic) also isolated.

**Key to Abbreviations:**

- ACC = Animal Care Center
- AsPlns = Asian Plains
- BC = Buffy coat leukocytes
- ENS = Eye and nasal secretion pool
- Fec = feces
- NS = Nasal secretion
- WAP = San Diego Wild Animal Park
Table 3 - Summary of Clinical Episodes of Malignant Catarrhal Fever in Captive and Free-living Wild Ruminant Species

<table>
<thead>
<tr>
<th>Species</th>
<th>Geographic Location</th>
<th>(C) Captive or (F) Free-living</th>
<th>Wildebeest Associated?</th>
<th>Sheep Associated?</th>
<th>Year</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>American bison</td>
<td>South Dakota USA</td>
<td>F</td>
<td>-</td>
<td>+</td>
<td>1976</td>
<td>(50)</td>
</tr>
<tr>
<td>Arabian oryx</td>
<td>San Diego CA (WAP) USA</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1979</td>
<td>(U)</td>
</tr>
<tr>
<td>Axis deer (Cervus axis)</td>
<td>Texas USA</td>
<td>F</td>
<td>-</td>
<td>+</td>
<td>1968,1969,1972 (6,7)</td>
<td></td>
</tr>
<tr>
<td>Banteng (Bos javanicus)</td>
<td>Bali (Bos javanicus)</td>
<td>C</td>
<td>-</td>
<td>+</td>
<td>1974–1982 (41)</td>
<td></td>
</tr>
<tr>
<td>Blesbok (Damaliscus dorcas)</td>
<td>Johannesburg, SO. AFR.</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1964</td>
<td>(8)</td>
</tr>
<tr>
<td>Blackbuck (Antilope cervicapra)</td>
<td>San Diego CA (WAP) USA</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1979</td>
<td>(U)*</td>
</tr>
<tr>
<td>Bongo (Taurotragus oryxerus isaaci)</td>
<td>San Diego CA (Zoo) USA</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1980</td>
<td>(U)</td>
</tr>
<tr>
<td>Bushbuck (Tragelaphus scriptus)</td>
<td>Johannesburg, SO. AFR.</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1964</td>
<td>(8)</td>
</tr>
<tr>
<td>Cape buffalo (Syncerus caffer)</td>
<td>London, England UK</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1979</td>
<td>(1)</td>
</tr>
<tr>
<td>Congo buffalo</td>
<td>Catskill NY USA</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1965</td>
<td>(31)</td>
</tr>
<tr>
<td>Duiker (Species not indicated)</td>
<td>Johannesburg, SO. AFR.</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1964</td>
<td>(8)</td>
</tr>
<tr>
<td>Eld's deer (Cervus eldi thamin)</td>
<td>San Diego CA (WAP) USA</td>
<td>C</td>
<td>+</td>
<td>-</td>
<td>1979,1980 (50)</td>
<td></td>
</tr>
</tbody>
</table>

*Unpublished
<table>
<thead>
<tr>
<th>Species</th>
<th>Location</th>
<th>First Year</th>
<th>Last Year</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wisent or European bison (Bison bonasus)</td>
<td>Europe</td>
<td>1977</td>
<td>1979</td>
<td>(59)</td>
</tr>
<tr>
<td></td>
<td>London, England UK</td>
<td></td>
<td></td>
<td>(1)</td>
</tr>
<tr>
<td>Greater kudu (Tragelaphus strepsiceros)</td>
<td>St. Louis MO USA</td>
<td>1972</td>
<td>1974</td>
<td>(2)</td>
</tr>
<tr>
<td></td>
<td>Johannesburg, SO. AFR.</td>
<td>1964</td>
<td>1970</td>
<td>(8)</td>
</tr>
<tr>
<td></td>
<td>Oklahoma City OK USA</td>
<td>1979,1980</td>
<td></td>
<td>(4,5,67)</td>
</tr>
<tr>
<td>Hartebeest, red (Alcelaphus sp.)</td>
<td>Johannesburg, SO. AFR.</td>
<td>1964</td>
<td>1970</td>
<td>(8)</td>
</tr>
<tr>
<td>Indian gaur (Boa gaurus)</td>
<td>Oklahoma City OK USA</td>
<td>1979</td>
<td>1982</td>
<td>(16)</td>
</tr>
<tr>
<td></td>
<td>San Diego CA (Zoo, WAP) USA</td>
<td>1964,1982</td>
<td></td>
<td>(U)*</td>
</tr>
<tr>
<td></td>
<td>Kansas City KS USA</td>
<td>?</td>
<td></td>
<td>(U)</td>
</tr>
<tr>
<td>Lechwe (Kobus lechwe)</td>
<td>Johannesburg, SO. AFR.</td>
<td>1964</td>
<td></td>
<td>(8)</td>
</tr>
<tr>
<td>Llama (Llama quanicoe glama)</td>
<td>Johannesburg, SO. AFR.</td>
<td>1964</td>
<td></td>
<td>(8)</td>
</tr>
<tr>
<td>Mule deer (Odocoileus hemionus)</td>
<td>Colorado USA</td>
<td>1975</td>
<td></td>
<td>(34,44)</td>
</tr>
<tr>
<td>Nilgai (Boselaphus tragocamelus)</td>
<td>Albuquerque NM USA</td>
<td>1970</td>
<td></td>
<td>(U)</td>
</tr>
<tr>
<td></td>
<td>London, England UK</td>
<td>1979</td>
<td></td>
<td>(1)</td>
</tr>
<tr>
<td></td>
<td>San Diego CA (WAP) USA</td>
<td>1981</td>
<td></td>
<td>(U)</td>
</tr>
<tr>
<td></td>
<td>Johannesburg, SO. AFR.</td>
<td>1964</td>
<td></td>
<td>(8)</td>
</tr>
<tr>
<td>Pere David's deer (Elaphurus davidianus)</td>
<td>London, England UK</td>
<td>1957,1959,1979</td>
<td>(1,60)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>San Diego CA (WAP) USA</td>
<td>1980,1982</td>
<td></td>
<td>(U)</td>
</tr>
<tr>
<td></td>
<td>Chicago IL USA</td>
<td>1981</td>
<td></td>
<td>(U)</td>
</tr>
<tr>
<td>Red deer (Cervus elaphus)</td>
<td>England UK</td>
<td>1976</td>
<td></td>
<td>(44)</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>1976</td>
<td></td>
<td>(44)</td>
</tr>
<tr>
<td>Rusa deer (Cervus timorensis)</td>
<td>Australia</td>
<td>1982</td>
<td></td>
<td>(62)</td>
</tr>
<tr>
<td>Sambar deer (Cervus mariannus)</td>
<td>Johannesburg, SO. AFR.</td>
<td>1964</td>
<td></td>
<td>(8)</td>
</tr>
<tr>
<td></td>
<td>San Diego CA (WAP) USA</td>
<td>1979</td>
<td></td>
<td>(U)</td>
</tr>
<tr>
<td>Sika deer (Cervus nippon)</td>
<td>Toronto, ONT. CAN.</td>
<td>1973,1974</td>
<td></td>
<td>(52)</td>
</tr>
<tr>
<td></td>
<td>San Diego CA (WAP) USA</td>
<td>1977,1980,1982</td>
<td>(U)</td>
<td></td>
</tr>
</tbody>
</table>

*Unpublished
Table 4: Test of Specificity of Indirect Immunofluorescence Method for Assaying Malignant Catarrhal Fever Herpesvirus (MCFV) Antibody

<table>
<thead>
<tr>
<th>MCFV Infected FAK* Cells</th>
<th>Uninfected FAK Cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undilute</td>
<td>1:20</td>
</tr>
<tr>
<td><strong>Anti-bovine herpesvirus-1</strong> (IBR)</td>
<td>+</td>
</tr>
<tr>
<td><strong>Anti-bovine herpesvirus-2</strong> (mamillitis)</td>
<td>+</td>
</tr>
<tr>
<td><strong>Anti-bovine herpesvirus-3</strong> (MCF)</td>
<td>+</td>
</tr>
<tr>
<td><strong>Anti-bovine herpesvirus-4</strong> (DN599)</td>
<td>+</td>
</tr>
</tbody>
</table>

*FAK = Fetal aoudad (Barbary sheep) kidney.

*Unpublished
REFERENCES


INVESTIGATIONS OF MALIGNANT CATARRHAL FEVER IN RUMINANTS AT THE OKLAHOMA CITY ZOO

B. M. Baumeister, M.S.*

***

INTRODUCTION

Malignant catarrhal fever (MCF) is a known clinical entity worldwide, with an etiologic agent described in African ruminant species. However, the etiology of MCF in other parts of the world is controversial. The disease has been recognized as a cause of death in domestic cattle, wildlife, and zoo ruminants in North America; however until recently, the etiologic agent had not been isolated in the United States. Following definitive diagnosis of MCF at the Oklahoma City Zoo in 1980, the Zoo began investigating the source of the MCF virus within its collection of ruminants and the extent of exposure of these species by tests for antibodies to MCF virus in sera. This paper presents the results of these serologic tests and the isolation attempts for MCF virus during a 2-year period. Our current protocol for handling movement of zoo ruminants in zoos where MCF is diagnosed and our recommendations for modification of that protocol are also presented.

MCF is an acute generalized disease of most ruminant species and is characterized by a severe inflammation of the upper respiratory and alimentary tracts, lymphadenopathy, high fever and ophthalmia. MCF has long been recognized as a cause of death in cattle in Europe, Africa, and North America. In cattle, MCF has a low morbidity rate but an extremely high mortality rate. Description of two forms of the disease, an African and an American (sheep-associated) form, has added to the complexity in understanding the pathogenesis of MCF. The clinical signs, gross lesions, and histopathologic lesions described are identical in both forms. Differentiation between the two forms clinically or at necropsy is difficult or impossible.

The etiologic agent, a herpesvirus termed Alcelaphine herpesvirus 1,3 has been isolated from wildebeest species, the presumed reservoir host of the virus in Africa. Field observations suggest that sheep may be the reservoir host for the disease in America. Several viral agents have been reported in clinical cases of MCF in domestic ruminants in the United States, however no definite etiologic agent has yet been described. Presently, serologic data indicates one serotype for MCF virus. Serologic
tests currently in use are unable to distinguish between antibodies produced by the MCF-affected animals in the United States or Africa. In this paper MCF shall be considered as one disease entity with certain different clinical manifestations.

In zoos, MCF has been recognized as a disease of ruminant species since 1962. Reports from eight zoos have documented MCF in exotic ruminant species. However, it was not until 1980 that a herpesvirus was isolated from a clinical case of MCF at a North American zoo. In a period from October 1979 to January 1980, the Oklahoma City Zoo experienced deaths, diagnosed as MCF, of 3 gaur, Bos gaurus, and one greater kudu, Tragelaphus strepsiceros. All four animals exhibited clinical signs of mucopurulent nasal discharge, oral ulcerations, corneal opacity, and fever (40.5-41.5°C). Death occurred in all cases within 6 days of onset of symptoms. Gross and histopathologic lesions were characteristic of MCF. Histopathologic lesions of lymphocytic vasculitis/perivasculitis were observed throughout all the body tissues examined.

A herpesvirus was isolated in fetal bovine kidney and thyroid cell cultures from buffy coat cells obtained from 1 gaur and from the greater kudu. Serial transmission of MCF was accomplished by passage of whole blood from the gaur into a domestic heifer, Bos taurus, then blood from the heifer into a bovine calf, and blood from the calf into a white-tailed deer, Odocoileus virginianus. Whole blood from the kudu inoculated into a deer also produced clinical MCF. Viral isolates from the gaur, kudu, heifer, and white-tailed deer each exhibited similar immunologic, morphologic, and cytopathic characteristics as the Alcelaphine herpesvirus 1 of MCF isolated in Africa. In may 1982, an adult female gaur from the same herd died from MCF. Diagnosis was made on the basis of clinical signs and histopathology. No virus was isolated from that case.

In January of 1980, a decision was made to quarantine all ruminants in the collection for 6 months and to serologically test most of them for antibodies to MCF virus. Management of the white-tailed gnu (WTG), Connochaetus gnou herd was altered to avoid possible contact or fomite transmission of the virus, especially during the wildebeest calving season. Following a 6 month quarantine, the zoo then resumed a normal policy with regard to animal sales, but elected not to move any WTG off grounds until further information about MCF was acquired.

Methods & Results: Our investigations were concentrated into three areas: 1) a serologic survey of the zoo's ruminants for antibodies to MCF virus, 2) virus isolation attempts from white blood of day-old WTG and Cape hartebeest, Alcelaphus buselaphus caami, calves, and 3) virus isolation and histopathological exam of euthanatized WTG.

A serologic profile of the ruminant collection was conducted from January 1980 to September 1982. Serum samples from 267 individuals representing 25 ruminant species at the Oklahoma City Zoo were tested by an indirect fluorescent antibody test (IFAT) for antibodies to MCF virus at
INVESTIGATIONS OF MCF 573

the National Veterinary Service Laboratory (NVSL), Ames, Iowa. Additionally, 12 sera from WTG were evaluated by the serum-virus neutralization assay at Plum Island Animal Disease Center, Greensport, N.Y. The results of both serologic surveys are presented in Table 1.

Of 46 WTG tested, 30 (65%) were seropositive for MCF at a 1:20 dilution. Twenty of these WTG were less than two days old when tested; of these 11, (55%) were seropositive for MCF. Four WTG between 2 days and 6 months of age were tested; 3 (75%) were seropositive. Twenty-two juveniles and adults were tested; 16 (73%) were seropositive.

Of 22 Cape hartebeest tested, 14 (64%) were seropositive. Seven of the hartebeest tested were less than two days old; 2 (28%) were seropositive. Six hartebeest between 2 days and 6 months of age were tested; 5 (75%) were seropositive. Nine juveniles and adults were sampled; 7 (88%) were seropositive for MCF. One juvenile harebeest previously seropositive reverted to seronegative.

Of 211 other animals tested at random, 19 (9%) individuals representing seven species were seropositive for antibodies to MCF virus. Two Eur-asian species, muntjac (Muntiacus reevesi) and chamois (Rupicapra rupicapra), had seropositive individuals. Of 72 Eur-asian ruminants tested, 9 (23%) were seropositive. Five non-alcelaphine African species, eland (Taurotragus oryx), sitatunga (Tragelaphus spekei), addax (Addax nasomaculatus), Nubian ibex (Capra ibex nubiana) and Grant's gazelle (Gazella granti roosevelti), had seropositive individuals. Of 186 non-alcelaphine African ruminants tested, 10 (5%) were seropositive for antibodies to MCF virus.

During the 1981 and 1982 calving seasons, 20 WTG and 8 Cape hartebeest calves were caught and bled within 48 hours of birth. Serum was collected and tested for antibodies to MCF virus and 30 to 40 ml whole blood was obtained for virus isolation attempts. Of 11 seropositive WTG calves tested, virus was isolated from one individual and identified morphologically as a herpesvirus. Subsequent inoculation of this herpesvirus into a white-tailed deer produced clinical MCF. Of the 9 WTG calves seronegative or having questionable titers, a herpesvirus, serologically related to MCF virus, was isolated from one calf. No attempts were made to transmit this latter virus to other species. Virus was not isolated from Cape hartebeest calves.

In 1982, the WTG herd had grown to 6 males and 20 females. To prevent young males breeding females and intermale aggression, it was decided to euthanatize all surplus males, and attempt MCF virus isolation from these animals. Five males were immobilized and transported live to the Oklahoma Animal Disease Diagnostic Laboratory, Stillwater, Oklahoma. Three animals were electro-ejaculated to obtain sperm and approximately 500 to 1000 ml whole blood collected from each individual prior to euthanasia. A complete necropsy was performed on each animal. For virus isolation attempts, Buffy coat cells, pooled organ tissues (lung, lymph node, and spleen; liver and kidney) and sperm, which was sonicated to
rupture the membranes on the sperm head, were co-cultivated with fetal bovine kidney and thyroid tissue cultures. The results of these attempts are shown in Table 3 (animals 1, 2, 3, 6 and 7). One of these males was sero-positive for MCF. Three of the seronegative males had histopathologic vasculitis lesions, suggestive of infection by MCF virus. Viral isolation attempts were uniformly negative.

Additionally in 1982, two pregnant female WTG were transported alive to OADDL for euthanasia, necropsy and virus isolation attempts (Table 3, animals 4 and 5). Tissues from both females were negative for viruses. One female was seronegative but had moderate vasculitis lesions present in lung tissue. From the fetuses, WTG kidney and lung cell cultures were established. Viruses were not isolated from these cell cultures of fetal tissues although one culture did exhibit cytopathic effects (microsyncytia) suggestive of an infection by a herpesvirus. Inoculation of a young white-tailed deer with these fetal WTG cells did not produce clinical disease or produce a seroconversion for antibodies to MCF virus.

One orphaned WTG calf was taken to OADDL at age 4 days, (Table 3, animal 8). Virus isolation attempts from antemortem samples of nasal and conjunctival washings were unsuccessful. The animal exhibited no clinical signs, was seronegative for antibodies to MCF virus and, at postmortem, no histopathologic lesions of MCF were found. A herpesvirus which was non-enveloped when seen by electron microscopy was isolated from pooled organ tissues (spleen, lymph node, bone marrow, lung) in fetal bovine kidney cell culture: inoculation of this virus into white-tailed deer produced clinical MCF.15

DISCUSSION

The mode of transmission of MCF remains obscure. In Africa, MCF is associated with the mixing of cattle and wildebeest during the wildebeest calving season.4 MCF virus has been isolated from ocular and nasal secretions from wildebeest calves younger than 3 months; but not in the secretions of wildebeest calves older than 3 months.16 That study appears to implicate young calves as the transmitters of virulent MCF virus. That virus was not cell-associated but was cell-free. Cell-free viruses appear less sensitive to humidity and temperature than cell-associated viruses. This may be critical for transmission in nature.

Of the clinical cases of MCF at the Oklahoma City Zoo, only the case of the greater kudu has a plausible explanation as to the source of the virus as the kudu was housed with a WTG calf. The reservoir and method of transmission of the MCF virus in the outbreak in the gaur, is more difficult to explain. The herd of gaur are housed in the same general location within the zoo as WTG and Cape hartebeest but there has been no direct contact between the gaur and WTG or hartebeest nor does drainage occur from WTG and hartebeest pens into the gaur lot. In the 1979 MCF outbreak, the WTG calves were all less than 3 months of age and all the hartebeest calves were older than 6 months. In 1982, no WTG calves under 6 months of age were present on grounds. The ages of Cape har-
tebeest calves in the collection in May 1982 varied from newborns to 3 months old. The origin of the MCF virus has not been identified for any of the disease outbreaks in the gaur herd.

The number of seropositive WTG in our collection is impressive and clearly indicative of the presence of MCF virus as a latent or persistent infection. The subsequent isolation of virus from WTG calves, although seemingly latent but still virulent, affirms its presence. Furthermore, isolation of herpesvirus from the blood of 2 one-day old WTG calves points to the vertical transmission of virus from dam to offspring as reported by Plowright, and the potential of these young animals as transmitters of the virus.

The presence of virus in two WTG calves seronegative (by IFAT) indicates that not all WTG calves harboring virus contain antibodies to MCF virus. The immaturity of the immune systems in animals 1 to 12 days old could account for the negative serology result. The presence of 6 seronegative juvenile and adult WTG raised in the herd and presumed to have been exposed to MCF virus suggests that some WTG do not react immunologically to the virus. Whether this lack of response to the virus is due to an immaturity of the immune system, failure of recognition (immune tolerance) in those individuals exposed to MCF virus early in utero or due to an immunologically defective virus is unknown. In as much as 4 asymptomatic seronegative WTG (80% of those examined histopathologically) possessed vasculitis lesions described for MCF, it appears that WTG which are seronegative for MCF (by IFAT) cannot be called free of MCF virus. This suggests that although the current serologic test will detect antibodies to MCF virus, it cannot be used as the sole criteria for the ultimate disposition of animals.

Our inability to isolate virus from juvenile or adult WTG suggests that at time of sampling these animals were not viremic or shedding virus. This agrees with other reported observations that transmission of virus appears to occur only during wildebeest calving.

The number of Cape hartebeest seropositive for MCF is also impressive. Although other hartebeest species are reported to harbor herpesviruses, MCF is not found in cattle which graze with calving hartebeest in "natural" situations. The possibility that the hartebeest possess a herpesvirus antigenically similar to, but not, the MCF virus was suggested. The number of attempts at virus isolation from hartebeest in our collection have been few and herpesviruses have never been isolated. The status of the MCF-carrier state in the Cape hartebeest remains open. The numbers of seropositive individuals without virus isolated raises the question of specificity of the IFAT.

The number of individuals of other species with positive serologies for MCF virus in our collection is a concern. Presented with the distance of clinical gaur cases at our zoo in relation to the location of WTG and the lack of adequate information as to transmission of MCF, it does not seem surprising to find the wide distribution of seropositive individuals.
throughout the zoo. The sporadic incidence and wide phylogenetic distribution of seropositive individuals without clinical MCF in those species, however, again raises the question as to whether these animals are truly seropositive for antibodies to MCF virus or if these species harbor other indigenous herpesviruses which cross-react serologically by IFAT with the MCF virus. This question has been raised by recent studies by W. Heuschele.\(^2\) The absence of a seropositive gaur is of interest, as all the sampled gaur were housed with and exposed to the clinical cases. This result argues for the absence of contact transmission of MCF from cattle to cattle as stated by Plowright.\(^1\)

These studies have provided much data; however, they only begin to answer some of the major problems of MCF. The questions which still must be investigated are:

1) Are all wildebeest calves potential carriers of MCF virus whether seropositive or seronegative?

2) Are hartebeest species inapparent carriers of MCF virus?

3) Which non-alcelaphine species can carry and subsequently transmit MCF virus?

Because of the number of unknowns, how then do we handle ruminant stock with respect to MCF and the movement of exotic ruminant species from collections at zoos? Admittedly, losses in zoos to MCF appear to be few. It is possible, however, that MCF has gone unrecognized in many zoos. No major catastrophies have occurred or been recognized in the zoo world or wildlife populations in North America despite years of movement of wildebeest species. This does not, however, diminish the responsibilities to our own collections or to the purchasers of these animals. Nor does it reduce the liability if an MCF outbreak in domestic animals or wildlife is traced back to stock sold from zoos.

We offer now our suggestions in handling this problem. The first step should be an endorsement of continued research on MCF. As this truly represents a profession-wide problem, it would seem that this effort should be supported profession-wide. It should not be dependent on a few institutions to support the cost and bear the results of this research. A primary step would be to support the relaxing of federal restrictions which hinder the study of this disease in laboratories in the U.S. Without access to infectious virus, reagents and MCF-related materials, meaningful research by a sufficient number of investigators cannot be accomplished. Refined techniques for measurement of antibodies, with known specificity, must be established. Methods for the identification of carrier animals, standardization of virus isolation techniques, and epidemiologic work-up of known clinical cases should receive major priorities. Additional serologic surveys of ruminant species located in zoos of North America, regardless of the presence of wildebeest or alcelaphine species in the collections, should be encouraged.

Education must occur simultaneous to and in conjunction with continued research. For zoo veterinarians, curators and directors and state
and federal veterinarians to render decisions regarding control of MCF and disposition of zoo stock they must be aware of what is currently known. The increasing amount of available data makes the education process especially important and challenging. The information should be brought forth immediately by recognized experts.

The most difficult aspect of MCF research and education is that it creates the dilemma, medical and ethical, of how to handle alcelaphine species and seropositive non-alcelaphine species. The question of what is practical, equitable and effective for the identification and disposition of ruminants which might be inapparent carriers of MCF virus is not easily answered. Until the profession can formulate a policy each individual institution and its state authorities must create their own.

The following is the current policy for handling ruminant stock and their movement at the Oklahoma City Zoo. This was devised by the zoo staff and our state and federal veterinarians. It is reported here not as a recommendation, but as an example of an interim handling plan being used, until more data and a formal, hopefully, national, policy is created.

1. No WTG is to be moved off zoo ground, except to diagnostic labs for MCF study.
2. No WTG or Cape hartebeest calf will be hand-raised. If a calf is rejected or cannot be mother-raised, it will be utilized for further research into MCF.
3. Any ruminant to be moved out of the collection will be isolated from other stock and after a period of three to four weeks, a blood sample obtained for MCF serology. If that serum is negative, the animal is free to leave the collection. If the serum is positive, the animal is restricted to grounds until a change in policy is made.

This system is obviously cumbersome, expensive and, probably, condemns some WTG, hartebeest, and seropositive stock unnecessarily to the stigma of MCF. Because of the knowledge gained by two years of research into MCF at our zoo, the authors suggest the following modifications of the above protocol.

1. Adult and juvenile non-pregnant, non-wildebeest/hartebeest ruminant species be allowed to move freely from the collection. That all of these animals be tested for MCF, either prior to or at shipping, and the results of that test be reported to the purchaser and, if seropositive, to the state veterinarian of the state of entry.
2. That non-wildebeest/hartebeest ruminant calves, within the first six months of life, and pregnant females in the last trimester be tested, with results available prior to shipment. If seropositive, these animals are confined to grounds until older than six months or until they have calved and the calf is weaned.
3. Screen the blood of the seropositive non-wildebeest/hartebeest animals for virus by available methods to ascertain the viral status of the host.
4. Allow movement of adult and juvenile non-pregnant wildebeest and hartebeest, sero-tested for MCF, to permanent quarantine entry facilities (PQEF) without restriction. Adult animals unable to breed, i.e. castrated or neutered, may be available to non-PQEF facilities.

5. Continue to restrict movement of wildebeest/hartebeest pregnant 3rd trimester females and calves less than six months of age from zoo grounds.

The above recommendations are but one set of suggestions of how to deal with MCF in zoos. Clearly, in light of the available knowledge our former, "struthioform," attitude toward MCF is no longer appropriate. As much as presenting the complexities of how to cope with MCF, this problem also presents us with the opportunity to develop a model for handling infectious disease problems in our zoo stock. As our medical sophistication and diagnostic techniques become more advanced, our lives and responsibilities also will become more complex. We do not feel the questions that these complexities pose need be, necessarily, all answered individually. Rather, the zoo veterinary profession, possibly via the AAZV, must develop the vehicle for the acquisition and presentation of the answers needed.

Acknowledgement:

We gratefully acknowledge the assistance and cooperation of personnel at the National Veterinary Service Laboratory, Ames, Iowa, and Oklahoma Animal Disease Diagnostic Laboratory, Stillwater, Oklahoma.
TABLE 1. INCIDENCE OF SEROPOSITIVE ANIMALS TESTED FOR MCF ANTIBODIES AT THE OKLAHOMA CITY ZOO FROM 1980 - 1982

<table>
<thead>
<tr>
<th>ANIMAL</th>
<th>NUMBER TESTED</th>
<th>NUMBER POSITIVE</th>
<th>% POSITIVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>WHITE-TAILED GNU</td>
<td>46</td>
<td>30</td>
<td>65.2%</td>
</tr>
<tr>
<td>Connochaetes gnou</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAPE HARTBEEST</td>
<td>22</td>
<td>14</td>
<td>63.6%</td>
</tr>
<tr>
<td>Alcelaphus buselaphus caama</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MUNTJAC</td>
<td>18</td>
<td>5</td>
<td>27.7%</td>
</tr>
<tr>
<td>Muntiacus reevesi</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BARASINGHA</td>
<td>6</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Cervus duvauceli</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PERE DAVID DEER</td>
<td>1</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Elaphurus davidianus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GIRAFFE</td>
<td>2</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Giraffa sp.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AMERICAN BISON</td>
<td>3</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Bison bison</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GAUR</td>
<td>4</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Bos gaurus gaurus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAPE BUFFALO</td>
<td>4</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Syncerus caffer caffer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NILGAI</td>
<td>1</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Boselaphus tragocamelus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ELAND</td>
<td>6</td>
<td>1</td>
<td>16.6%</td>
</tr>
<tr>
<td>Taurotragus oryx</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NYALA</td>
<td>2</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Tragelaphus angasi</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SITATUNGA</td>
<td>17</td>
<td>1</td>
<td>6%</td>
</tr>
<tr>
<td>Tragelaphus spekei</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GREATER KUDU</td>
<td>6</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Tragelaphus strepsiceros</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADDAX</td>
<td>23</td>
<td>4</td>
<td>17.4%</td>
</tr>
<tr>
<td>Addax nasomaculatus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SABLE</td>
<td>15</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Hippotragus niger</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DEFASSA WATERBUCK</td>
<td>18</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Kobus elliptiprymnus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WHITE-EARED KOB</td>
<td>11</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Kobus k. leucotis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GEMSBOK</td>
<td>18</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Oryz gazella gazella</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DAMA GAZELLE</td>
<td>4</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Gazella dama ruficollis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GRANT'S GAZELLE</td>
<td>10</td>
<td>1^b</td>
<td>10.0%</td>
</tr>
<tr>
<td>Gazella granti roosevelti</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MARKHOR</td>
<td>1</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Capra falconeri heptneri</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NUBIAN IBEX</td>
<td>10</td>
<td>3</td>
<td>30.0%</td>
</tr>
<tr>
<td>Capra ibex nubiana</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHAMOIS</td>
<td>12</td>
<td>4</td>
<td>33.3%</td>
</tr>
<tr>
<td>Rupricapra rupricapra</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAIGA</td>
<td>19</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Saiga tatarica</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>279</td>
<td>63</td>
<td>22.6%</td>
</tr>
</tbody>
</table>

a Whole herd sample at same time
b This animal converted to negative at subsequent testing
<table>
<thead>
<tr>
<th>ANIMAL</th>
<th>NUMBER TESTED</th>
<th>SEROLOGY</th>
<th>CULTURE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>NUMBER POSITIVE</td>
<td>NUMBER NEGATIVE</td>
</tr>
<tr>
<td>W. T. GNU</td>
<td>20</td>
<td>11</td>
<td>9</td>
</tr>
<tr>
<td>HARTEBEEST</td>
<td>8</td>
<td>2</td>
<td>6</td>
</tr>
</tbody>
</table>

* Pending results on one individual
<table>
<thead>
<tr>
<th>ANIMAL</th>
<th>AGE</th>
<th>SEX</th>
<th>SEROLOGY</th>
<th>VIRUS ISOLATION</th>
<th>HISTOPATHOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>AD</td>
<td>M</td>
<td>-1:20</td>
<td>Negative</td>
<td>Vasculitis</td>
</tr>
<tr>
<td>2</td>
<td>AD</td>
<td>M</td>
<td>-1:20</td>
<td>Negative</td>
<td>No Vasculitis</td>
</tr>
<tr>
<td>3</td>
<td>AD</td>
<td>M</td>
<td>+1:20</td>
<td>Negative</td>
<td>Slight Non-Specific Vasculitis</td>
</tr>
<tr>
<td>4</td>
<td>AD</td>
<td>F</td>
<td>+1:20</td>
<td>Negative</td>
<td>Slight Non-Specific Vasculitis</td>
</tr>
<tr>
<td>5</td>
<td>AD</td>
<td>F</td>
<td>-1:20</td>
<td>Dam-Neg. Fetus-Un-identified Virus Isolated</td>
<td>Vasculitis-But Not Severe</td>
</tr>
<tr>
<td>6</td>
<td>8 Mo.</td>
<td>M</td>
<td>-1:20</td>
<td>Negative</td>
<td>Vasculitis-But Not Severe</td>
</tr>
<tr>
<td>7</td>
<td>8 Mo.</td>
<td>M</td>
<td>-1:20</td>
<td>Negative</td>
<td>Vasculitis-But Not Severe</td>
</tr>
<tr>
<td>8</td>
<td>12 Days</td>
<td>M</td>
<td>-1:20</td>
<td>Positive</td>
<td>No Vasculitis</td>
</tr>
</tbody>
</table>
REFERENCES


The chairman opened the meeting by presenting the agenda topics to be brought before the committee. Dr. Anthony Castro, Oklahoma Animal Disease Diagnostic Laboratory, Stillwater, Oklahoma, and Dr. Werner Hueschele, Research Department, San Diego Zoo, were introduced to present papers on Malignant Catarrhal Fever (MCF) in certain zoological animals.

As presented by the speakers, important questions which must be answered in regard to the epidemiology of MCF include:

1. What other species of animals are reservoirs of MCFV and capable of shedding infectious virus?
   A. All Alcelaphinae? All Caprinae and Ovinae?
   B. Other ruminants?
   C. Non-ruminants (E.G. Lagomorpha)?

2. Are all animals with MCF serum antibodies carriers of MCFV? Are they all capable of shedding infectious virus, or are some, like cattle, dead end hosts which do not shed cell-free (transmissible) virus?

3. Do arthropod or avian species act as vectors and/or reservoirs?

4. Is the, as yet unidentified, sheep-associated MCF agent a variant herpesvirus related to the alcelaphine herpesviruses (bovine herpesvirus-3)?

5. What are the stability of survival capabilities of alcelaphine MCFV under various environmental conditions?

6. What is the relationship biochemically and antigenically of alcelaphine MCFV to other herpesviruses, especially of cattle? To what extent will they crossreact in serologic procedures?

7. To what extent is there biochemical and genetic variability among alcelaphine MCFV strains?

8. What are the modes of transmission of MCFV?

9. Since wildebeest, topi and hartebeest have been documented as carriers of MCFV, are other members of this group also (i.e., blesbok, bontebok, Hunter’s antelope, etc.)?

How to prevent the transmission of MCFV from domestic sheep and wild ruminant carrier-reservoirs to domestic cattle, very valuable and
endangered captive and free-living wild ruminants is a problem confronting us now. Sound approaches to the control of the MCF hazard must be based upon the best knowledge available, and not be characterized by over-reaction or inaction.

Based upon our current knowledge on MCF, the following policies are suggested to reduce the risk of spread of MCF among zoo animals, to domestic cattle and free-living wildlife:

1. Regulation by state or federal animal health authorities of the movement from or to any zoos of antelope of the subfamily Alcelaphinae (i.e., wildebeest, topi, hartebeest).

2. Restrict the issuance of permits for movement of and placement of alcelaphine antelope to USDA approved zoos and parks. Do not allow animals of this subfamily to be acquired by private individuals, rances, game farms or and similar facilities where they might expose domestic cattle, sheep or goats or indigenous wildlife such as deer, elk, bison, etc.

3. Only allow movement of non-pregnant alcelaphine antelope over four months of age, since current information indicates that only wildebeest less than four months old and near term pregnant females will shed cell-free (infectious) MCFV.

4. Institute a voluntary MCF antibody testing program for all captive wild ruminants intended for movement from any zoos or parks where MCF has occurred or alcelaphine antelope are present. Inform prospective recipients of such animals of test results so that they may make a decision on whether or not to take delivery, or determine where the animals will be located if accepted.

5. Premises receiving wild ruminants from other facilities where alcelaphine antelope are held should quarantine new arrivals for 60 days. They should determine their serologic status with respect to MCF antibody upon arrival and at the completion of 60 days in quarantine.

6. While there is no evidence at present that non-alcelaphine antelope, deer, bovine or wild sheep and goat species seropositive for MCV antibodies can transmit infectious MCFV to other susceptible ruminants, it is recommended that any ruminants seropositive for MCF antibodies be segregated from seronegative animals. Hygienic and sanitary precautions should be followed in handling such seropositive animals to preclude possible indirect exposure of susceptible animals.

Considerable positive discussion was held on the information and recommendations presented. Drs. Castro and Hueschele were commended for their presentations. It was a consensus of the committee members present that both papers be published in the USDA Proceedings as an integral part of the Zoological Animal Committee report.

Three resolutions were presented and passed for consideration by the Resolutions Committee.
The chairman reported that the subcommittee on drafting a model state statute relating to zoological animals would complete the initial draft after the first of the year and that this would be sent to the members for their perusal.

The meeting was adjourned at 4:55 pm.
87th ANNUAL MEETING
October 16–21, 1983
SAHARA HOTEL
Las Vegas, Nevada

88th ANNUAL MEETING
October 21–26, 1984
HYATT REGENCY HOTEL
Fort Worth, Texas

89th ANNUAL MEETING
October 27–November 1, 1985
THE MARC PLAZA HOTEL
Milwaukee, Wisconsin