Report of the Committee on Sheep and Goats
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Vice Chair: Maggie Highland, WA

Scott Bender, AZ; Deborah Brennan, MS; John Clifford, DC; Walter Cook, TX; Stephen Crawford, NH; William Edmiston, TX; Chester Gipson, MD;; Joseph Huff, CO; Paul Jones, AL; Don Knowles, WA; Eileen Kuhlmann, MN; James Leafstedt, SD; Mary Lis, CT; Jim Logan, WY; Linda Logan, TX; David Marshall, NC; Chuck Massengill, MO; Cheryl Miller, IN; Ronald Miller, PA; Jeffrey Nelson, IA; Kris Petrini, MN; Suelee Robbe-Austerman, IA; Paul Rodgers, WV; Joan Dean Rowe, CA; Mo Salman, CO; David Scarfe, IL; Diane Sutton, MD; Stephen White, WA; Margaret Wild, CO; Ellen Mary Wilson, NM; William Wilson, KS; Nora Wineland, MO;
David Winters, TX; Cindy Wolf, MN.

The Committee met on October 27, 2015 at the Rhode Island Convention Center in Providence, Rhode Island from 1:00 pm to 5:45 pm. There were 10 members and 25 guests present. *Chairman Amy Hendrickson introduced herself and Maggie Highland as new chair and vice chair. resolutions, introductions, or other notices and announcements.*]

Presentations & Reports

Research Update - The Arthropod-Borne Animal Diseases Unit

D. Scott McVey, Research Leader and Veterinary Medical Officer, Arthropod-Borne Animal Diseases Research Unit,

The Arthropod Borne Animal Diseases Research Unit's (ABADRU) research mission is to solve major endemic, emerging, and exotic arthropod-borne disease problems in livestock. The Unit completed the move to Manhattan, KS in 2010 and now the ABADRU is well established at the Center for Grain and Animal Health Research (CGAHR). All ABADRU research falls under the ARS National Research Programs: NP103 and Animal Health and NP104, Veterinary, Medical, and Urban Entomology. The areas of research range from vector biology to virus-host interactions.

The potential introduction of Rift Valley fever (RVF) virus (RVFV) is the most significant arthropod-borne animal disease threat to U.S. livestock. A number of challenges exist for the control and prevention of RVF in the areas of disease surveillance, diagnostics, vaccines and vector control. Understanding the epidemiological factors affecting disease outbreak and the inter-epizootic maintenance of RVFV is necessary for the development of appropriate countermeasures strategies. This includes the ability to detect and characterize emergent viruses. Outcomes of current research will potentially identify determinants of RVFV infection, pathogenesis and maintenance in mammalian and insect vector hosts. Information derived from these studies will also support vaccine development. Vaccine formulations will be developed to improve immunogenicity, onset of immunity and stability to provide better response to outbreaks and prevent RVFV epizootics. The Unit also has a similar, collaborative program investigating Schmallenburg Virus.

The viruses that cause bluetongue (BT) and epizootic hemorrhagic disease (EHD) are of concern to livestock producers in North America because of 1) the emergence of new serotypes, 2) increased reports of spillover and clinical disease in cattle, and 3) increased spread and adaptation to new geographical areas. Current projects in ABADRU include virus genotyping of more recent isolates, virus transmission and related pathogenesis, development of fluorescent microsphere assays for detection of virus-specific antibody and RNA, EHDV infection and transmission of whitetail deer, vector genetics, vector proteomics, vector transcriptomics, vector ecology/biology and vector control.

USDA in collaboration with DOI organized a gap analysis workshop composed of international experts on Orbiviruses. The workshop participants met at the Arthropod-Borne Animal Diseases Research Unit in Manhattan, Kansas, May 14–16, 2013, to assess the available scientific information and countermeasures to effectively control and mitigate the impact of an outbreak of an emerging Orbivirus with epizootic potential, with special emphasis given to bluetongue virus (BTV) and epizootic hemorrhagic disease virus (EHDV).

Orbiviruses, Bluetongue and Epizootic Hemmorhagic Disease: Gap Analysis Workshop Report. 2013. U.S. Department of Agriculture, Agricultural Research Service, Washington, DC.
http://go.usa.gov/BJ5F

The work has been published in *Vector-Borne and Zoonotic Diseases*.

Q Fever Update

Don Knowles, DVM, PhD, DACVP, Research Leader, ADRU-ARS-USDA, Pullman, Washington

Coxiella burnetti is an obligate intracellular bacterium that causes disease in ruminants and humans. This zoonotic infection can cause flu-like symptoms in humans and abortion in ruminants and humans. The recent outbreaks of abortion caused by *C. burnetti* in small ruminants in the Netherlands and the United States have rekindled discussions concerning methods to control transmission. The primary problem related to *C. burnetti* is its environmental stability. Therefore, methods need to be found to decrease transmission from small ruminants into the environment. Future collaborative research will focus on genetic and immunological approaches to decrease small ruminant shedding of *C. burnetti*.

Eliminating the Effects of Footrot on Sheep Flocks in the Northeastern United States

Brzozowski1, R., Settlemire T.[2], Parker C.[3], Lichtenwalner A.[1,4], White S.[5], Cockett N.[6]
1. University of Maine Cooperative Extension, 2. Bowdoin College Depts. of Biology and Chemistry*, 3. Ohio Agricultural Research and Development Center*, 4. University of Maine School of Food and Agriculture, 5. USDA ARS Pullman WA, 6. Dept. of Animal, Dairy and Veterinary Sciences, Utah State University (*Emeritus)

Foot rot in sheep was studied over a four-year period in an effort to eliminate the disease from flocks and in anticipation of identifying genetic resistance to the disease. The popularity of organic production methods and the trend toward reduction of antibiotic use in livestock informed this study, which did not include antibiotic treatment of sheep. The research team developed a 4-week protocol for implementation in flocks having signs and symptoms of lameness. The team and participating farmers actively implemented this protocol which included inspection, trimming, evaluation, segregation of sheep groups and weekly foot bathing with zinc sulfate. Twenty-two sheep farms in the northeast participated in this applied research project by providing their sheep for evaluation via farm visits. These farmers worked alongside the research team in handling the sheep, trimming feet and recording scores. Nearly 1,300 sheep were handled and evaluated over the life of the project. In addition to the protocol, blood was sampled from each sheep and sent with individual foot scores to the Agricultural Research Service laboratory in Pullman, Washington for analysis in an effort to possibly identify a genetic marker for sheep showing resistance and/or susceptibility to the disease. Participating farmers were surveyed each year to determine foot health conditions in their respective flocks. Results from an end-of-project survey of these farms in December 2014 showed that the protocol was effective in 61% of the flocks. Willingness to cull "chronic" cases (animals with severely deformed hooves) was critical to success of the program. Preliminary evaluation of a subset of the sampled sheep suggests that susceptibility to foot rot may be genetically controlled. Initial genotyping was completed on approximately 240 animals using an Ovine SNP50 marker set that includes over 50,000+ single nucleotide markers. The results appear promising for additional genotyping and further genetic analysis. A more refined analysis is needed to determine a possible marker. Over the life of the project, materials were developed and presentations made to equip producers with knowledge and skills in addressing foot rot in their flocks. An on-line template for producers to design their own written biosecurity plan for disease prevention was initiated. To date, over 150 farms have used this tool. Informational items from the project web site have been used as a means to educate producers as well as agriculture service providers. Since its establishment, the project web site has received over 17,000 page views. The video on how to trim the feet of sheep received over 70,000 views. In addition, a 2-session webinar series on small ruminant foot health reached 36 and 33 individuals in the live broadcasts. Records for the archived webinar sessions show that Session 1 was viewed over 900 times and Session 2 was viewed over 250 times. While genetic screening may become a useful tool, hands-on management and education will continue to be important for producers seeking help with this widespread disease. This project showed that even without the use of antibiotics, careful managers can eliminate footrot by following a strict protocol of treatment and culling of chronic cases.

Full presentation provided at end of report.

Insight into mechanisms regulating immune responses to Haemonchus contortus infection of sheep

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Haemonchus contortus causes sever production losses in small ruminants and has become more problematic resulting from development of resistance to multipole drugs used to treat these infections. One approach to the problem is utilizing sheep that are resistant to gastrointestinal nematode (GIN) parasitism. One such breed is the St. Croix that we have shown to reduce fecal egg count to zero by five weeks after a primary infection and no detectable egg count during a challenge infection. Alternatively Suffolk sheep generated high fecl egg count during both infection. The difference between the breeds lies in their ability to generate immune responses to invading larvae. Data generated in my lab has demonstrated that St. Croix sheep have greater cellular infiltrate, lymph node hypertrophy and cytokine production associated with a dramatic decrease in larvae compared to Suffolk sheep. There is a 5 day delay in lymph node hypertrophy and a 7 day delay in production of the cytokine IL-4 in Suffolk sheep. These data have been further validated through in vitro studies that support the conclusion that early host immune responses prevent larval development to adult stages causing pathology observed by H. contortus infection. Failure of Suffolk sheep to mount immune responses to the infective larval stage permits adult establishment thus pathology observed in these sheep. To maintain productivity of economically-relevant breeds of sheep that utilize forage resources, exploration of immunomodulation of those breeds while grazing will be necessary.

Predictive Models in Policy and Decision Making

Mark C. Thurmond DVM, PhD , Professor Emeritus, Veterinary Infectious Disease Epidemiology, University of California, Davis

This paper offers brief descriptions of modeling, as applied generally in risk assessments (RA) and specifically in some USDA RAs driving livestock policy. Issues are reviewed that relate to applications of science, model logic, validation, and representation of facts. Recommendations are offered for ways to prevent bad science in modeling and RA, and specifically, what can be done to ensure 'best available science' will be applied to studies from which livestock policies and decisions are formulated.

Risk Assessment

Definition:

Risk assessment is a framework for processes used to obtain estimates of a projected magnitude and probability (risk) of an event (such as a disease outbreak) in some time period. Data, assumptions, and models that consider known causes of the event and possible mitigation steps are used to estimate predicted values for risk. If validated, results may be applied by governments to develop policy or to make decisions, as a means of managing or controlling risk. A RA offers a framework for viewing well-established information and data, and '---does not create new knowledge or information.'.[1] It is critical to understand that 'Risk assessment cannot be expected to compensate for lack of knowledge'[1], and to do so would be to operate under false pretenses.

Standards for RA --- the National Academies of Science:

In 1983, the National Academies of Science published guidelines for the science of RA, referred to as the 'Redbook'.[2] The report was motivated by stakeholder concerns that government policy considerations would distort '---scientific interpretations in risk assessments and [stakeholders] seek new institutional safeguards against such distortion.'.[1] It has been admonished further that '---risk managers should not attempt to alter those [Redbook] standards in specific cases simply to ensure that some pre-determined management objective is more easily achievable.'.[1] Different assumptions can be applied that manipulate and distort model results, which '--- lead to quite different predictions of risk.', and, thus, have '--- provided the opportunity for case-by-case manipulations of risk assessment results to achieve predetermined risk management objectives ---.'.[1]

Risk Assessment Steps:

The standard steps applied in a RA include hazard identification/assessment, dose response assessment, exposure assessment, and risk characterization. Hazard identification involves building a solid science-based case for the targeted agent, including specific information about the agent, clinical studies, mechanisms of action of the agent, and extensive evidence on the agent as the cause of the problem (ie. disease). This step must provide solid scientific evidence for the hazard under question, and for all factors, including the agent of interest, known to contribute causally to the disease. Dose response assessment addresses how changes in the agent change risk of disease. Exposure assessment

documents the population(s) under consideration, exposures in those populations, how individuals come into contact with the agent, sources of the agent, and mechanisms for transmission. This step also provides data about the doses observed in typical exposures, and temporal aspects of exposure and transmission.[1,2]

General Comments on Modeling:

Modeling has been used for decades to help understand biological processes and to predict what events might unfold following some alteration of factors that drive or influence those processes. Models often are used to provide a framework for thinking about relationship and dynamics of natural phenomena. With greater computational power offered by computers, modeling also has helped to gain insight into complex disease processes and to clarify voids or weak links in our understanding of those diseases. Providing good quality data are available, computer generated models can offer a means to dis-entangle our view of causal pathways for complex and dynamic biological systems that operate through multiple and interacting factors. Use of models to predict future weather events is one classical example of predictive modeling.

Since the 1960's, models have been used to help policy and decision makers develop plans and projections that become the basis for government action. Engineering has had a long-standing history of modeling structural designs, engineering, and materials to project the most likely areas and times for failure. Risk assessment typically can involve use of models to identify and evaluate risks, the various components of risk, and the importance of each component in overall hazard assessment, where hazard is defined as the general threat being faced. A RA of salmonella in poultry, for example, could examine elements of carcass processing to identify the most likely (most probable) points for contamination by salmonella. The ultimate objective in an RA is to obtain validated estimates of probabilities that can be used to manage or mitigate the risks, in this example to reduce the hazard of salmonellosis in humans. There are many varied applications of models and modeling being used to establish or influence livestock policy and decisions. For example, models are used in estimating diagnostic test accuracies, projecting the risk of importing foot-and-mouth disease (FMD) virus into the US, comparing hypothetical vaccination schemes for FMD in the face of an epidemic, and predicting transmission of pneumonia from domestic sheep to bighorn sheep, to mention a few. A basic rule-of-thumb, however, is that all models are wrong, but some models are less wrong than others.

Models used in these applications can be quite varied, ranging from statistical models (models applying mathematical theory of statistics), with generally well-developed theoretical underpinnings, to *ad hoc* mathematical models designed and crafted for the specific application, perhaps without much theoretical foundation and relying instead on fundamental biological knowledge and hypothetical pathways. Some models can be extraordinarily complex, such as those used in weather prediction that may consider a myriad of information involving data on ocean temperatures, jet stream location and speed, humidity, wind speed and direction, currents, etc. In contrast, models for diagnostic test accuracy may be comparatively simple.

Hazard assessment modeling:

In the hazard assessment step, an abundance of science should be presented such that solid, unambiguous documentation exists that the 'agent' causes the problem (ie disease) being considered.[1,2] Thus, prerequisites for any risk assessment are convincing data and studies anchoring cause-and-effect (the agent causes the disease in question). There are many approaches and experimental study designs that can be employed to address the question whether an agent or substance causes a disease. The National Academies, however, singles out one general type of evidence that must be included before cause-and-effect can be claimed in stating: 'Well conducted epidemiologic studies that show a positive association between the agent and the disease are accepted as the most convincing evidence about --- disease.'.[2]

Epidemiologic studies involve the examination of multiple hypothesized factors as 'causes' of a disease. These studies utilize data and models that provide statistical evidence for factors *most likely* to affect disease occurrence, given the conditions of the study. The models will depend on the study design undertaken and on the specific type of data to be collected. Regardless of the approach, a general conceptual model must first be developed to layout a reasonably exhaustive list of potential (hypothesized) factors that could be causally connected to the disease. There should be scientific foundation for selection of the variables, where sound evidence can be found in the literature or highly

compelling logic exists. As an example, consider an epidemiologic model for pneumonia in bighorn sheep, where emphasis here is on illustrating conceptualization and transition of an initial 'simple' model to more formal models, rather than correctness of the model.

Conceptual model of pneumonia in bighorn sheep might be:

First iteration (very general):

Pneumonia (clinical occurrence) = weather + stress + parasites+ bacteria + viruses + age + nutrition

More specific:

Pneu = min daily temp + cum snow + stress (qualitative) + worms (y/n) + OPP/CAEV (y/n) + bacteria + years from median herd age + daily Kcal intake.

General statistical model might be:

Prob $(pneu)_t$ =

$Y + X_1 Tmin_t + X_2 Cumsnow_t + X_3 Stress_t + X_4 Worm_t + X_5 Virus_t + X_6 Bacteria_t + X_7 Age_t + X_8 Kcal_t$ + error.

In words, this expression says that the probability of a case of pneumonia occurring by time t (say after lambing) is a function of (is influenced by) some unknown value (intercept-Y) and the hypothesized variables observed at time t. The statistical analysis, in this case an event-history analysis, would indicate which variable(s) contributed in a statistically 'significant' way to the variation observed in the proportion of animals with pneumonia. The X's indicate the strength of an association. This simple model would examine only the 'main' effects of the variables, but not interactions between variables. For example, the effect of cold temperatures might be more likely to predispose to pneumonia if the animal was very young or very old, or if energy intake was low rather than high. Consequently, models can become somewhat complex in order to address real and important biological dynamics. When applied to modeling, the fallacy of *Occam's Razor*, which states that simpler is better, will greatly ease the work of the modeler, in not having to mess with biological detail, while simplifying-away the gist of the biology and important causality being considered. Predicting biological events, such as transmission of disease agents, must consider the complex interacting webs of causation, as well as disease transition states in the pathogenesis of disease. Otherwise, the models only pervert to make-believe exercises. Development of models, therefore, requires careful attention to pathogenesis and natural occurrence of diseases, where clinicians and other disease experts should be involved in design to assure the most biologically plausible models are considered. Typically, epidemiologic models undergo several iterations to ascertain various prediction scenarios and to run diagnostic tests to ensure the models hold up to the underlying mathematical assumptions. Results of a completed model may provide estimates of the effect of variables with significant influence on the outcome (eg. pneumonia), which can be used to obtain estimates of attributable risk and other measures that provide pragmatic metrics for 'how much pneumonia' was due to a factor. A strong temptation is to conclude that results of such a study will apply to future pneumonia cases in that study herd, or worse--- to all pneumonia cases in all herds at all times. This problem relates to fallacies of inductive reasoning and to how we consider validity of the model (see validation below). The only truthful conclusion would be that results relate only to the herd studied for that specific study period when the observations were made. Thus, as the National Academies of Science notes,[2] well designed epidemiologic studies are prerequisite elements in hazard assessment in order make confident claims that a factor is a cause of a disease.

<u>Predictive-type Models:</u>

Generally, predictive models are developed to project 'what if' scenarios where one wishes to obtain some sense for how an outcome might be altered if various exposures or elements of a process change. Risk assessments, and the models developed for RA, do not create new knowledge and cannot compensate for a lack of knowledge. As an initial step, a conceptual idea or model of the process at hand is crafted to identify elements and mathematical relationships that offer a logical depiction of the biological process of interest. The model indicates the parameters of interest and assumptions of the parameter values.

As an example, suppose we wish to 'predict' the likelihood (probability) that one will become infected with a cold virus after entering a room of people and occupying it for some period of time (t). The process being depicted involves the transmission of an infectious agent, which will be assumed here (for

simplicity) to be transmitted only by physical contact with an infected person. We apply the disease transition states, namely susceptible, resistant, and infected-and-shedding, as well as infectious dose. In addition, we make an assumption about the likelihood of contact (Prob(con)), which we will assume to be 0.2 (20%), meaning that if a person entered the room 10 times, on average, a contact would take place during two of those times. The model assumes, based on known infectious disease concepts, that a transmission event will take place under the following conditions: the person entering the room must be susceptible (not already infected) (probability=Prob(Susc)), and must make contact with an infected person (probability=Prob(Inf)). In turn, that infected person must be shedding the virus (probability=Prob(Shed if inf) at an infectious dose to which the recipient is susceptible (probability=Prob(Dose if inf and shed)).

The probability of transmission in the time period t, Prob($Trans_t$), is the product of these probabilities, indicating all conditions must be met in order for transmission to take place, or
Prob($Trans_t$) =
Prob(Susc) x Prob(Inf) x Prob(Shed if inf) x Prob(Dose if inf and shed) x Prob(Con).

Let the following probabilities (fictitious) be assumed here for the transition states:

Prob(Susc)	= 0.9
Prob(Inf)	= 0.1
Prob(Shed if inf)	= 0.5
Prob(Shed inf dose if shed)	= 0.5

or

Prob($Trans_t$) = 0.9 x 0.1 x 0.5 x 0.5 x 0.2 = **0.0045**. (one in 222).

Thus, in this contrived example, the estimated probability of a person becoming infected with a cold virus after entering a room of people is 0.0045, meaning that one out of 222 such experiences would result in infection with a cold virus. The realistic conditions that must be met are that the room must be occupied, and that no barrier exists to contact; the probability of transmission would be zero if the room were empty or if contact were not possible during the period of occupancy.

This estimate does not represent the probability of acquiring a cold (clinical symptoms), which would represent an additional step, or 0.0045 x Prob(clinical manifestation if infected). For diseases with more complex causes, such as pneumonia, as indicated in the above model, the predictive models for clinical disease following exposure to an infectious agent would require inclusion of all other factors known to cause the disease (eg. weather, stress, nutrition, etc). In this sense, we cannot say that diseases like pneumonia are 'transmissible' because they are caused in part by factors that are themselves not transmissible, such as weather, stress, nutrition, etc. Attempts to model complex diseases, like most respiratory diseases, as transmissible events would illustrate a lack of understanding of disease pathogenesis and epidemiology.

Note that the elements presented here, the logic for the sequence, and assumed probability values used are in part 'made up' or fictitious, for purposes of presentation. Any predictive modeling used in a RA, however, must include strong justification for the logic, elements, and assumed values of parameters, including citation of literature and/or presentation of actual data.[1,2] A RA should address the question: 'how accurate is the prediction model', and thus, a model should not be applied in a RA unless/until it has undergone a validation process to estimate accuracy (see validation below). Recall the rule of thumb; all models are wrong --- some are less wrong.

<u>'Best available science --- and science ethics' in modeling:</u>

One question that must be asked of any RA and model is whether it represents application and practice of 'best available science'. We see that definitions of 'science' have many variations, including pursuit of knowledge and truth, and a highly disciplined practice involving dispassionate search for underlying mechanisms about the world and universe. Good science, whether undertaken by modelers or not, is expected to represent application of well-designed studies, with critical, truthful, and reasoned interpretation of scientific literature, non-fraudulent data and assumptions, and non-fallacious and logical conclusions. Such pursuits must employ a skeptical, but unbiased and non-prejudicial mindset, and a

willingness to change one's mind when data and logic so dictate. What are some signs of bad science, or contradictions to 'best available science'?

A cornerstone anchoring the foundation for any scientific endeavor, including modeling, is the critical analysis of what is already known, or thought to be known. Scientific ethos dictate an honest understanding of the literature, including a faithful and truthful rendering and citation of findings (not mere parroting an author's opinion), as they follow from the methods and as they relate to the broader inferences being addressed. One way counterfeit science and modeling is revealed is by a documented failure to accurately and truthfully support statements of fact. Citations may reveal misinterpretations of the results, reference to results that were obtained using improper methodology or data, or reference merely to an author's opinion or interpretation of results as fact. One who fails to truthfully characterize and cite published findings, regardless of intentions, is guilty of falsifying scientific testimony.

Another sign of bad science is use of fallacious reasoning to support statements of fact. Examples include the fallacy that 'correlation equals causation' and the *post hoc ergo propter hoc* fallacy (meaning 'after this therefore because of this'). The high correlation between smoking and lung cancer was, at one time, believed to mean that coffee consumption caused lung cancer, when in fact the strong correlation between coffee consumption and smoking was responsible for the erroneous conclusion. As an example of the *post hoc ergo propter hoc* fallacy, one would conclude that, if a man developed cancer after being observed riding a bicycle, the bicycle caused the cancer.

Validation of Models:

Validation in the context of modeling refers to the process of estimating accuracy of a model in predicting some event or outcome. Completion of a validation process does not mean a model is valid, correct, or accurate. Generally, a validation process compares the predicted number of outcomes with the number of outcomes observed under natural (real-life) conditions. One needs to know, for example, whether the model predicted 80%, 50%, or 5% of the real events/outcomes. Without validation information, one has no clue as to whether the model has any legitimacy. Weather models offer a unique perspective into validation, whereby the observed data always follow predicted data. Consequently, weather models undergo a continuous validation process to identify parameters and parameter values that need to be 'tweeked' to improve accuracy. The National Academies of Science 'Reference Manual on Scientific Evidence' admonishes that models should hold little legal standing without validation, and that mere publication in a scientific, peer reviewed journal does not mean a study represents any sense of the truth or that findings can be viewed as having any validity.[3]

USDA Payette and Snow Mesa risk assessments and policy-decision:

Payette and Snow Mesa RAs:

The Payette and Snow Mesa RAs undertaken by the USDA Forest Service have been used to develop policy to close domestic sheep allotments in the Payette National Forest and the Snow Mesa region.[4,5] The USDA's presumption underlying the RAs is that contact of a bighorn sheep (BHS) with a domestic sheep will result in 'transmission of respiratory disease' that eventually results in die-offs of BHS. Review of these RAs reveals an absence of key steps, 'best available science', and ethics required in RA and modeling, and in science in general. Some of the more egregious issues revealed in these RAs are described below.

Presenting false testimony:

The Snow Mesa and Payette RAs make several statements of fact claiming domestic sheep transmit disease to BHS, including, for example the statement in the Snow Mesa RA: '--- extirpation [of bighorn sheep herds] due to respiratory diseases, *which can be transmitted by domestic sheep or goats (Besser et al. 2012b, Cassirer et al. 2013)*, appears to be the greatest concern for bighorn sheep population persistence on the Rio Grande National Forest (USDA Forest Service 2010).' There is no scientific foundation for this (italicized) statement based on USDA's citations. Neither publication[6,7] presents any methodology or results for 'transmission of respiratory disease', nor did either examine any domestic sheep as study subjects. Similar false testimonies persist throughout the Payette and Snow Mesa RAs. Such significant misrepresentation of published results is a serious scientific offence and violation of trust, and suggests the papers and literature as a whole were not reviewed by scientists or not read at all --- or findings were surreptitiously misrepresented. These falsifications are even more serious considering they

refer to the core of the USDA hypothesis that domestic sheep are responsible for the decline of BHS. This deception casts a pall over ethics and quality of science applied overall in both of these USDA RAs. False testimony also has been presented using fallacious reasoning. Throughout the RAs the USDA applies the classical *post hoc ergo propter hoc* fallacy to claim causality. The RAs say that because there was an observation of contact between a BHS and a domestic sheep, presumably before a BHS die-off was observed, a subsequent die-off must have been caused by that contact. The USDA also is specious in this argument because it ignores the other logical observations, including contacts observed after a die-off, die-offs with no observed contacts, and contacts with no observed die-offs. Similarly, studies finding that when BHS and domestic sheep are co-mingled in forced and highly stressful close confinement, BHS develop respiratory disease. The USDA has chosen to interpret such a correlation as evidence that contact with domestic sheep causes disease in BHS. These types of non-critical and fallacious thinking have no place in 'currently available science', let alone in an elevated position at the core of a thesis.

Hazard identification/assessment and dose response:
Neither of these RAs met the minimal standards set forth in the Redbook for risk assessment, nor did they address or meet the indispensable, or *sine qua non*, requirement that 'Well conducted epidemiologic studies that show a positive association between the agent and the disease are accepted as the most convincing evidence about ---disease.'.[2] No attention was given to the causality of pneumonia, including the multitude of factors that can contribute to predisposition and onset (eg. weather, nutrition, stress, etc.). Both RAs misrepresented disease processes and epidemiology of pneumonia by referring to transmission of pneumonia or respiratory disease, which itself is not transmissible, at noted above. No presentations were offered for dose response of the agent, mechanisms of action, mechanisms of disease, or exposures. In fact, the Snow Mesa RA made no mention of the agent the USDA had in mind as a cause of bighorn sheep pneumonia die-offs. In the Payette RA, *Mannheimia haemolytica* was offered as a putative agent, which the USDA noted is a commensal bacteria in many animal species. The RA did not address whether the agent was a *necessary* or a *sufficient* cause of pneumonia.[8] *M. haemolytica* is not a necessary cause of pneumonia because other agents also can cause pneumonia, and it is not a sufficient cause of pneumonia because not all animals with *M. haemolytica* have pneumonia. So, what causal relationship, if any, is there? In short, one must wonder how the USDA reconciles establishing a policy to prohibit contact between domestic sheep and BHS that is based on an inadequate RA that failed to provide a legitimate (without false testimony) case for cause-and-effect for an agent, a mechanism for exposure to domestic sheep, or an agent dose response, to mention a few omissions of vital information. Why were standards for 'best available science' in conducting a RA, known for more than three decades,[2] not applied in these USDA RAs? When such critical data cannot be obtained to achieve the minimal information necessary, ethics and good conscience science dictate that risk assessment efforts '---must be forsaken'.[1]

Model for probability of contact and subsequent disease:
The Payette RA[4] created a 'model' for the probability that a bighorn sheep would acquire pneumonia after crossing into a domestic sheep allotment. The model disregarded the transition state elements described above (eg. fictitious cold virus example), and instead assumed only one parameter, namely the probability of contact and acquisition of pneumonia from a domestic sheep. The USDA assumed this probability ranged from 5% to 100%, meaning the probability of contact was assumed to be 100% (transmission could not occur without contact). The RA failed to consider the natural biological process of transmission, or to provide any valid foundation for the assumptions. It is quite easy, as noted above, to apply different assumptions in order to manipulate and distort model results, which '--- lead to quite different predictions of risk.', and, thus, have '--- provided the opportunity for case-by-case manipulations of risk assessment results to achieve predetermined risk management objectives ---.'.[1]
Not only do the high probability values indicate an ignorance of natural disease transition states, an assumed probability of contact of 1 (100%) also is nonsensical in that one would not expect the probability of contact to be the same, let alone 100%, if domestic sheep were 10 feet from the allotment line and a BHS as if they were two miles from the line. Worse yet, the USDA assumed that contact and transmission would take place even if the allotment were not occupied by any domestic sheep whatsoever, which obviously is impossible. The Snow Mesa RA[5] was no better; it merely characterized the probabilities into low, medium, and high risk categories, again without any supporting data. In other

words, using the analogous cold virus model above, the erroneous logic applied by the USDA would have one believe a person could become infected with a cold virus, which would require contact with and infected person, after entering an empty room. As aptly stated elsewhere, the models appear to involve '---the alleged distortion of science by government risk assessors, to ensure that risk managers received the answers they wanted, so that they could decide to regulate, or not to regulate, depending upon how they perceived the social, economic, and political pressures under which they were operating.'.[1]

Model for a bighorn sheep contacting an allotment:

In both RAs, the USDA assumed that BHS movement away from their home range and into a domestic sheep allotment would follow a model created for foraying BHS.[9] The model used data from captured, radio-collared BHS in Hells Canyon to estimate the extent of typical forays. The methods violated basic scientific standards by using data from animals (in Hells Canyon) that were not representative of animals in the Payette or Snow Mesa herds, as well as by using data that were not representative of foraying animals with normal foraying behavior or foraying inducements. A bias, akin to a Hawthorne effect,[10] was introduced whereby procedures imposed by the capture and radio-collaring would alter the outcome observed. That is, the traumatic and fear-inducing process by which BHS were trapped, captured, sedated, examined, and radio-collared could induce a change in foraying behavior. In an effort to flee their home range area, where they experienced the noxious event, they may well have forayed farther and more frequently, compared with non-radio-collared (normal) BHS. Because there were no 'control groups' in this study, as 'best available science' would dictate, one cannot truthfully claim or assume that the traumatic process of collaring did not itself explain the foraying data reportedly observed and modeled. Thus, a simple explanation for the foray results is that the act of being radio-collared induced an escape behavior resulting in frequent and long-distance forays, and the data would have no relevance to foray behavior of non-collared, normal BHS.

Validation:

None of the models or analytical methodologies used in the Payette or Snow Mesa RAs were validated. Consequently, there are no estimates of accuracy in projections of BHS home ranges, foraying proportions or distances, or of contact and transmission probabilities, all of which were critical elements the USDA used to develop its policy to close domestic sheep allotments. Application of non-validated models not only does not square with expectations for the 'best science available', it also runs counter to explicit admonitions of the National Academies of Science, namely that models must undergo validation ---- and peer-reviewed publication does not constitute validation.[3] These non-validated models build one upon another, further compounding falsehoods and corrupting truth, and, as a consequence, render baseless any policy emerging from these RAs.

In summary, legitimate and serious concern is raised about the poor quality of science revealed in the USDA's RAs and in their models. The issues include presenting false testimony about causes of respiratory disease in BHS, fallacious reasoning applied to interpretation of publications, and failure to provide minimal hazards identification or requisite vital information on causation. In addition, the RAs applied anomalous and nonsensical models to predict contact and disease. It is difficult to imagine why the USDA would undertake such flawed science, which only serves to present an appearance of deception or hoax. One would hope and expect that the USDA should set an example for good science, as is done through the Agricultural Research Service, in promoting and exemplifying rigorous and honest pursuits of truth, rather than contriving an impersonation of science aimed at justifying its policy agenda, namely exclusion of domestic sheep from government lands.

<u>Other failures of government modeling and policy:</u>

One of the most disastrous failures of modeling, as applied to policy and decision making, was use of a contrived model to dictate FMD eradication policy of the UK in 2001.[11] The model assumed incorrectly that a quarter of transmission was 'local', and thus predicted that culling contiguous herds near an infected herd would stop the epidemic. Convinced administrators directed that millions of uninfected livestock be killed, believing their 'science-based' policy would quickly put to end the epidemic and save millions. It did not happen. Instead, perhaps millions of uninfected livestock were destroyed.[12] In a related issue, USDA claimed its risk assessment showed it was safe to import beef from Argentina and Brazil, both of which have FMD. Congress, however, found the risk assessment inadequate and disallowed budgeting until the Secretary of Agriculture '---conducts an updated comprehensive risk

evaluation of importing beef produced in Argentina and Brazil---.'.[13] Just recently, an appeals court over-ruled an Environmental Protection Agency RA of the pesticide, in failing to show necessary and substantial data that the pesticide was not toxic to honey bee colonies and 'The [EPA] did not adequately study the pesticide sulfoxaflor'.[14] As a likely prelude to requesting funds to control ebola in Africa, a Centers for Disease Control model predicted 1.4 million deaths by last January 2015, which clearly failed the real-world validation test.[15] A DHS/USDA-sponsored RA projecting spread of FMD virus from the proposed NBAF lab in Kansas was reviewed by the National Academies of Science, which concluded the RA '---was inadequate due to flawed methods and assumptions which potentially underestimated the risk of an accidental FMD release from the NBAF in Manhattan, Kansas.'.[6] Considerable concern has been expressed for the abuse of these non-validated prediction models, referred to as 'the Emperor's new clothes',[17] particularly when basic disease biology has been ignored.

What can be done to ensure 'best available science' has been applied in livestock policy and decisions?
Suggestions for actions that collectively would help ensure that best available science has been and will be applied to livestock policy and decisions include:

1. AVMA and university graduate programs should require curricula to include instruction in research ethics and critical thinking.
2. USDA and DHS (and other agencies) should require research staff to complete an on-line course in research ethics and critical thinking.
3. Before commencing work, USDA, DHS, and other agencies should be required to submit for outside, independent scientific review and acceptance the proposed project plan for risk assessment or other studies that could be used in livestock policy and decisions.
4. Before implementing policy or decisions, USDA, DHS, and other agencies should be required to submit for outside, independent scientific review and acceptance the completed project.
5. Employees of USDA, DHS, and affiliated agencies should not be allowed to serve as reviewers for scientific journals or other review boards considering USDA or DHS-funded projects or of other projects that could form a basis for livestock policy or decisions.
6. USAHA should pass resolutions strongly recommending the above requirements.
7. USAHA should create a standing committee to address and review science-based government policy and decisions.
8. Congressional action on recommendations should be encouraged.

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Bacterial Pneumonia in Sheep, The Domestic –Bighorn Sheep Interface, and Research at ADRU
M. A. Highland, DVM, DACVP, PhD candidate, PhD Veterinary Training Program USDA-ARS ADRU, Veterinary Microbiology and Pathology – Washington State University, Pullman, WA
See full presentation attached.

USSES Research Update (on behalf of Bret Taylor, Acting Research Leader, U.S. Sheep Experiment Station, Dubois, ID) and ADRU ARS USDA Collaborative Research Overview
Presented by M. A. Highland, DVM, DACVP, PhD candidate, PhD Veterinary Training Program USDA-ARS ADRU, Veterinary Microbiology and Pathology – Washington State University, Pullman, WA
See full presentation attached.

Determining Seroprevalence of *Brucellaovis*in U.S. Sheep Flocks
Presented by M.A. Highland, on behalf of Kerry Sondgeroth, DVM, PhD, Assistant Professor/Veterinary Bacteriologist, University of Wyoming/WY State Vet Lab.
See full presentation attached.

Committee Business:

One resolution was discussed in regard to concerns over the need for established criteria in government agencies for evaluating research that is used to support animal health policy decisions. Resolution was moved by Paul Rodgers and seconded by Jim Logan. After extensive discussion the question was called. The committee voted in favor of the resolution with one nay vote. The individual voting against the resolution informed the other members that he is supportive of the concept but is concerned about the possible unintended consequences of the request as set forth.

There had been a request to place discussion of Veterinary Feed Directive but it was decided that the matters of concern had been addressed elsewhere during the full meeting.

With no further business to be discussed, the Committee adjourned at 5:45 pm.