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UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF WASHINGTON

WILDEARTH GUARDIANS, and WESTERN WATERSHEDS PROJECT,

Plaintiffs,

v.

KRISTIN BAIL, Okanogan-Wenatchee National Forest, Forest Supervisor, and U.S. FOREST SERVICE,

Defendants,

S. MARTINEZ LIVESTOCK, a Washington Corporation,

Defendant-Intervenor.

No. 2:20-cv-00440-RMP

DECLARATION OF DR. THOMAS BESSER IN SUPPORT OF PLAINTIFFS' MOTION FOR PRELIMINARY INJUNCTION

I, Thomas Besser, with full knowledge of the penalties of perjury, declare as follows:

1. I have been a Professor and infectious disease researcher in the Department of Veterinary Microbiology and Pathology, College of Veterinary Medicine, and the Washington Animal Disease Diagnostic Laboratory at Washington State University, Pullman, Washington since 1986. Upon my retirement in September 2019, I became Professor Emeritus.

2. I have been asked by Plaintiffs in this litigation to provide my expert opinion on disease transmission from domestic sheep to bighorn sheep and scientific issues relevant for making management decisions where conflicts exist.

I. Professional Qualifications and Experience.

3. I have over thirty-five years of experience researching the epidemiology and ecology of infectious diseases caused by pathogens across host species. My CV is attached as Exhibit A.

4. I hold a Bachelor of Science degree in Zoology from the University of Wisconsin (1973), a Doctor of Veterinary Medicine degree from the University of Minnesota (1981), and a PhD in Veterinary Science from Washington State University (1986).

5. I have held appointments in the Washington Animal Disease Diagnostic Laboratory throughout my career at WSU: Bacteriology Section Supervisor (1986-2000, during which time I founded the Combined Diagnostic

Microbiology / Infectious Diseases PhD program), Director of Laboratory Services (2000-2004), Molecular Diagnostics Supervisor (2004-2008). Since 2008 I provided consultation on issues related to public health/food safety and wildlife diseases. I was Director of the WSU College of Veterinary Medicine's Immunology and Infectious Diseases graduate studies program (2013-2019). I was an adjunct professor in the Paul G. Allen School for Global Animal Health. In 2016, I was appointed to serve as the WSU/WSF Rocky Crate Endowed Chair in wild sheep disease research, which I held until my retirement.

6. I am a member of the American College of Veterinary Microbiologists and served on their Board of Governors from 2009 to 2012. I am also a member of the American Society for Microbiology, and served as a member of its Applied and Environmental Microbiology Editorial Board from 2008 to 2010.

7. I have received honors for my work from the University of Minnesota, Washington State University, the Washington State Academy of Sciences, and the American Association for the Advancement of Science, and I was a Fulbright Research Fellow at the University of Edinburgh in 2000-2001.

8. I have been conducting research in the fields of veterinary immunology, epidemiology, and microbiology since 1982. One of my primary research focus areas since 2005 has been epidemic pneumonia in bighorn sheep,

including the cause, prevention, and management of this disease through laboratory and field-based microbiological and epidemiological studies.

9. Specifically, my wild sheep research has focused on clarifying the cause of bighorn sheep epizootic pneumonia in order to improve the prevention and management of this disease. In 2009, I joined the Bighorn Sheep Disease Research Consortium, an association of experts studying many aspects of bighorn sheep pneumonia, including transmission, immunity, microbial etiology, connectivity and habitat modeling, pneumonia disease dynamics, and chronic carriage. As a member of this consortium and in my roles as a research microbiologist in the Department of Veterinary Microbiology and Pathology and a diagnostician at the Washington Animal Disease Diagnostic Laboratory, I studied the role of *M. ovipneumoniae* in causing the pneumonia outbreaks that follow its introduction into previously healthy bighorn sheep populations, the persistence of this agent (and the disease it causes) in bighorn sheep populations after it is introduced, and methods to clear the agent from infected bighorn sheep populations to improve their health and productivity. I have also worked on the health and productivity costs of *M. ovipneumoniae* in domestic sheep reservoir, and developed methods to eliminate this pathogen from domestic sheep operations.

10. I served as principal investigator of numerous individual and collaborative research projects looking at disease transmission to bighorn sheep

totaling over $700,000 since 2013. The funding sources for these projects included the U.S. Forest Service, the Idaho Wildlife Disease Research Oversight Committee, the Idaho Department of Fish and Game, the Washington Department of Fish and Wildlife, the Hells Canyon Initiative of Washington, Oregon, and Idaho, and both the national and numerous state chapters of the Wild Sheep Foundation.

11. I have published 361 research papers and 11 book chapters on various infectious disease, immunology, and diagnostic methods topics, including 25 papers on the diagnosis, pathophysiology, epidemiology, management, and control of bighorn sheep pneumonia. The bighorn sheep pneumonia papers where I served as first or senior author included[1]:

Association of Mycoplasma ovipneumoniae *infection with population limiting respiratory disease in free-ranging Rocky Mountain Bighorn Sheep* (Ovis canadensis canadensis) (2008).

Causes of Pneumonia Epizootics among Bighorn Sheep, Western United States, 2008-2010 (2012).

Survival of Bighorn Sheep Commingled with Domestic Sheep in the Absence of Mycoplasma ovipneumoniae (2012).

Bighorn sheep pneumonia: Sorting out the cause of a polymicrobial disease (2013).

Epizootic pneumonia of bighorn sheep following experimental exposure to Mycoplasma ovipneumoniae (2014).

[1] The full citations for these papers are found in my CV, attached to this declaration.

Immunogenicity of a Mycoplasma ovipneumoniae *bacteria for domestic sheep* (Ovis aries) (2014).

Comparison of two bacterial transport media for culture of tonsillar swab from bighorn sheep and mountain goats (2016).

Concordance in Diagnostic Testing for Respiratory Pathogens of Bighorn Sheep (2016).

Exposure of bighorn sheep to domestic goats colonized with Mycoplasma ovipneumoniae *induces sub-lethal pneumonia*. (2017).

Evidence for strain-specific immunity to pneumonia in bighorn sheep (2017).

Pneumonia in bighorn sheep: Risk and resilience. (2018).

A pilot study of the effects of Mycoplasma ovipneumoniae *exposure on domestic lamb growth and performance* (2019).

Risk factors and productivity losses associated with Mycoplasma ovipneumoniae *infection in United States domestic sheep operations* (2019).

Comparison of three methods of enumeration for Mycoplasma ovipneumoniae (2019).

Genetic structure of Mycoplasma ovipneumoniae *informs pathogen spillover dynamics between domestic and wild Caprinae in the western United States* (2019).

12. In addition, I contributed to other papers on bighorn sheep pneumonia as a co-author, which included:

Mycoplasma ovipneumoniae *can predispose bighorn sheep to fatal* Mannheimia haemolytica *pneumonia* (2010).

Use of exposure history to identify patterns of immunity to pneumonia in bighorn sheep (Ovis canadensis) (2013).

Pneumonia in Bighorn Sheep: Testing the Super-Spreader Hypothesis. (2015).

Age-specific infectious period shapes dynamics of pneumonia in bighorn sheep. (2017).

Detection of Mycoplasma ovipneumoniae *in Pneumonic Mountain Goat (*Oreamnos americanus*) Kids*. (2018).

Epidemic growth rates and host movement patterns shape management performance for pathogen spillover at the wildlife-livestock interface (2019).

Removal of chronic Mycoplasma ovipneumoniae *carrier ewes eliminates pneumonia in a bighorn sheep population.* (2020).

Restoration of a bighorn sheep population impeded by Mycoplasma ovipneumoniae *exposure.* (2020).

Previously Unrecognized Exposure of Desert Bighorn Sheep (Ovis canadensis nelsoni) to Mycoplasma ovipneumoniae *in the California Mojave Desert.* (2021).

13. I have given numerous presentations on bighorn sheep pneumonia and *Mycoplasma ovipneumoniae* at conferences, institutions of higher education, symposiums, colloquiums, workshops, and consortia both nationally and internationally. These include addresses presented at the following:

University of California (Davis CA, 2007).

Research Symposium: "Respiratory Disease in Wild and Domestic Sheep" hosted by the Association of State Wildlife Agencies (Salt Lake City, 2008).

Idaho collaborative working group on bighorn – domestic sheep (Boise, 2009).

WA/OR/ID Bighorn Sheep Disease Consortium (various, 2010, 2016, 2019).

Association for Veterinary Epidemiology and Preventive Medicine (Chicago, 2011).

Northern Wild Sheep and Goat Council (various, 2012, 2016, 2018, 2020).

Wild Sheep Working Group of the Western Association of Fish & Wildlife Agencies (Reno, 2013, 2015, 2019, 2020).

Wild Sheep Society of British Columbia (Cranbrook, 2013).

The Wildlife Society symposium on wildlife diseases (Charlotte, 2015).

Wallowa Whitman National Forest (Pendleton, 2015).

Okanagan Wenatchee National Forest (Ellensburg, 2016).

Oregon State University College of Veterinary Medicine (Corvallis, 2016).

Challis Sheep Producers (Challis, 2016, 2019).

University of Idaho Sheep Center Industry Advisory Board (Moscow, 2016).

Desert Bighorn Council (Borrego Springs, 2017).

American Society for Microbiology (New Orleans, 2017).

Midwest Wild Sheep Foundation chapter meeting (Minneapolis, 2017).

Wild Sheep Foundation Chapters and Affiliates (2017, 2018).

Ellensburg Sheep Producers (Ellensburg, 2018).

Desert Tortoise Council (Las Vegas, 2018).

Colorado Collaborative (Denver, 2019).

Montana WSF/DS collaborative meeting (Helena MT, February 2017).

Future Farmers of America state meetings in Washington and Idaho (Pullman WA and Moscow ID, 2018).

Hells Canyon area sheep and goat producers (Asotin, 2019).

NAPGA/WSF meeting on the Shoshone National Forest pack goat access issue (Spokane, August 2018).

14. I have also provided scientific opinions to the U.S. Forest Service in its development of management plans to reduce the risk of disease transmission from domestic sheep to bighorn sheep. These included a 2009 presentation to the agency that discussed transmission of pneumonia from domestic sheep to bighorn sheep, and a 2010 letter to the Payette National Forest explaining recent scientific research showing transmission of respiratory pathogens from domestic sheep to bighorn sheep.

II. Relevant Scientific Studies on Bighorn Sheep Disease.

15. My work on bighorn sheep pneumonia first addressed the question: What infection(s) cause bighorn sheep pneumonia outbreaks? Earlier work had identified many candidate pathogens that were found in bighorn sheep with pneumonia but had not clarified which infections caused the disease. My work targeting the cause of bighorn pneumonia resulted in two key papers.

16. First, in the paper, "Causes of pneumonia epizootics among bighorn sheep, Western United States, 2008-2010", we showed that the bacterium *Mycoplasma ovipneumoniae* was detected in nearly all bighorn sheep tested from eight different herds affected by pneumonia outbreaks but not found in the bighorn

sheep from the control healthy herds studied. No other pathogens had similarly high infection rates in pneumonic herds. Furthermore, our work identified single genetic strain types of *M. ovipneumoniae* involved within each outbreak. Many bacterial species exhibit minor changes in their DNA sequence as they evolve, and *M. ovipneumoniae* is more variable than most. This high variability provides a tool to researchers studying *M. ovipneumoniae* infections, because when it is directly transmitted from animal to animal, there is insufficient time for it to evolve and the DNA strain types match exactly, whereas when acquired from different sources differences in DNA sequences are expected. Therefore, the single strain types detected within outbreaks provide direct evidence that *M. ovipneumoniae* had spread directly from animal to animal as expected for the 'primary' pathogen driving the disease; none of the other candidate bacterial pathogen species showed this definite pattern of animal-to-animal spread.

17. Second, in the paper "Bighorn sheep pneumonia: sorting out the cause of a polymicrobial disease", we compared all the major candidate bighorn sheep pneumonia pathogens for how well they fit widely accepted epidemiological 'causal criteria', a powerful method to clarify complex questions of disease causality. The data summarized in this paper provided very strong support for *M. ovipneumoniae* as the causal agent of bighorn sheep epizootic pneumonia, while providing weak or no support for a causal role for all other candidate pathogens.

We now know that *M. ovipneumoniae* is the pathogen that triggers fatal bighorn sheep epizootic pneumonia outbreaks across the range of the species.

18. The genetic (DNA sequence-based) strain typing method described earlier also provided an explanation for a very important pattern that occurs repeatedly in bighorn sheep pneumonia: When previously healthy bighorn sheep herds first experience a pneumonia outbreak, the disease typically affects all age classes from lambs through adult animals. After these 'all-ages outbreaks' have waned, the surviving adults appear to recover to good health and have relatively normal life expectancies. However, for a period of years to decades afterwards, a different pattern of disease emerges, in which most or all lambs annually develop pneumonia, a pattern referred to as 'lamb pneumonia'.

19. Strain typing revealed that lamb pneumonia cases were infected by the same *M. ovipneumoniae* strain that had been introduced to trigger the earlier all-ages outbreak: A subset of the surviving, apparently healthy ewes continue to carry the outbreak strain in their noses and these chronic nasal carrier ewes serve as a source of the infection to lambs each year. The lamb infection quickly spreads to all lambs in the population, often triggering near 100% mortality before they reach 6 months of age. Herds affected by the lamb pneumonia pattern stagnate or decline in population numbers due to the lack of recruitment (sufficient lambs surviving the first year of life to replace normal adult death rates).

20. Another important aspect of bighorn sheep pneumonia is the spread of disease across metapopulations (groups of populations that are largely independent, but that are connected by occasional animal movements). A metapopulation structure generally adds greatly to the resilience of species like bighorn sheep, since local adverse events affecting one population are countered by animal movements from the other populations within the metapopulation. However, diseases like bighorn sheep pneumonia illustrate the downside of the metapopulation structure: if the animal moving between populations happens to be a chronic carrier of *M. ovipneumoniae,* it can trigger additional outbreaks across the metapopulation.

21. This pattern was clearly documented in the Hells Canyon metapopulation across the borders of Idaho, Washington, and Oregon: after the initial introduction of *M. ovipneumoniae* into northern Hells Canyon in 1995, 14 other populations in the metapopulation experienced disease from the same *M. ovipneumoniae* strain type between 1996 and 2013, due to movements of infected bighorn sheep through the canyon. From this and other examples, it is now clear that introduction of *M. ovipneumoniae* into one bighorn sheep population can trigger disease events in many other populations across long time periods.

22. Once the key role of *M. ovipneumoniae* in bighorn sheep pneumonia was clarified, the logical next question to address was: What is the source of *M.*

ovipneumoniae that infect bighorn sheep to trigger pneumonia outbreaks? We had previously shown that healthy bighorn herds (that is, those with no evidence of pneumonia) were free of this pathogen, based on both highly sensitive nasal swab PCR tests and on blood tests that indicated lack of previous exposure or infection. When some of those healthy, *M. ovipneumoniae*-negative herds subsequently experienced fatal pneumonia outbreaks, *M. ovipneumoniae* were invariably present, showing that the herd had recently become infected.

23. Using genetic strain typing of *M. ovipneumoniae*, we were able to demonstrate that some of those outbreaks were caused by *M. ovipneumoniae* known to be carried by other bighorn sheep herds within the metapopulation, and we concluded that those outbreaks resulted from infections carried by infected bighorn sheep from the source infected herd. However, most new bighorn sheep pneumonia outbreaks were caused by *M. ovipneumoniae* strains that were not carried by any bighorn herds within the region, and we concluded that these outbreaks resulted from contacts with other infected animal sources.

24. The most frequent and widespread *M. ovipneumoniae* infected animal sources are domestic sheep and domestic goats. *M. ovipneumoniae* was first discovered and characterized in domestic sheep, and subsequently it has been recognized as infecting and causing respiratory disease in both sheep and goats globally. *M. ovipneumoniae* is extremely common in domestic sheep: the USDA

National Animal Health Monitoring Service Sheep 2011 project, a national survey of domestic sheep operations, detected *M. ovipneumoniae* infections in approximately 90% of domestic sheep operations sampled, including in all operations larger than 500 head involved in the study.

25. We investigated the ability of *M. ovipneumoniae* carried by domestic sheep to cause bighorn sheep disease in the paper "Epizootic pneumonia of bighorn sheep following experimental exposure to *Mycoplasma ovipneumoniae"*, where we showed that *M. ovipneumoniae* carried by apparently healthy domestic sheep can infect and induce severe pneumonia in previously healthy bighorn sheep. These experiments also showed that domestic and bighorn sheep in proximity interacted readily, for example at feedbunks, water supplies, and bedding areas, including nose-to-nose contacts, such that direct contact transmission would be possible.

26. While *M. ovipneumoniae* has recently been reported in species other than sheep and goats (Caprinae), neither its ability to persist in these hosts for long periods of time, nor the ability of these non-Caprinae hosts to transmit the pathogen to bighorn sheep has been demonstrated, and the low carriage prevalence and the low genetic diversity of *M. ovipneumoniae* in non-Caprinae hosts are not consistent with them representing a separate reservoir for bighorn sheep infection. Together, these data show that domestic sheep, if present on the landscape

occupied by bighorn sheep, carry *M. ovipneumoniae* that represents a large risk for bighorn sheep infection and resulting pneumonia outbreaks. Domestic goats also pose a definite risk to bighorn sheep due to their *M. ovipneumoniae* reservoir status, although limited current data shows that goat sources tend to cause less severe and less persistent bighorn disease. In contrast, non-Caprinae species have not yet been shown to present any risk of transmitting *M. ovipneumoniae* to bighorn sheep.

27. *M. ovipneumoniae* infections from different sources can be identified and distinguished based on variation within their DNA sequences, as mentioned earlier. The DNA sequence-based molecular strain typing method (Multi-Locus Strain Typing, MLST) we developed for *M. ovipneumoniae* can be used to track spread of specific strains within and between bighorn sheep populations, and to identify potential sources of strains responsible for new bighorn pneumonia epizootics.

28. This method was first published in the paper entitled "Evidence for strain-specific immunity to pneumonia in bighorn sheep", which determined "…that introduction of a new genotype (strain) of *M. ovipneumoniae* into a chronically infected bighorn sheep population in the Hells Canyon region of Washington and Oregon was accompanied by adult morbidity (100%) and pneumonia-induced mortality (33%) similar to that reported in epizootics

following exposure of naïve bighorn sheep. This suggests an immune mismatch occurred that led to ineffective cross-strain protection." In this example, more than ten years after a severe pneumonia outbreak, the Black Butte bighorn herd exhibited the common 'lamb pneumonia' pattern where the ewes that survived the outbreak had recovered to apparent good health and a normal life expectancy, but included chronic nasal carriers of the original outbreak strain that when transmitted to lambs resulted in annual high lamb pneumonia mortality.

29. In this paper we documented introduction of a new *M. ovipneumoniae* strain that triggered a dramatic change in the pattern of disease: all adult ewes developed signs of pneumonia (morbidity) and 30% died (mortality). The lambs again experienced a fatal pneumonia outbreak, primarily triggered by lung infections with the newly introduced *M. ovipneumoniae* strain. The finding of lack of cross-strain immunity has since been repeated elsewhere, confirming that the limited immunity that bighorn sheep may develop to a strain of *M. ovipneumoniae* with which they have been infected for years fails to consistently protect them from genetically novel strains that they may encounter.

30. The MLST method was subsequently used for a much larger study of *M. ovipneumoniae* strain types that was published in the paper entitled "Genetic structure of Mycoplasma ovipneumoniae informs pathogen spillover dynamics between domestic and wild Caprinae in the western United States". That paper

concluded: "The genetic data identify domestic sheep as an infection reservoir with multiple and ongoing spillovers to bighorn sheep. Domestic goats are also a source of infection to bighorn sheep, but dynamics of spillover appear to differ from domestic sheep. Strain-sharing across bighorn sheep populations and between wild hosts suggests that, following spillover, pathogen persistence and host movements also contribute to pathogen spread. The ability for *M. ovipneumoniae* to persist and maintain virulence in the absence of spillover is unclear." In this context, 'spillover' refers to infections acquired by a susceptible host species (here, bighorn sheep) from an infected source of a different species (here, domestic sheep or goats).

31. The domestic sheep sampled in this study carried a very large diversity of *M. ovipneumoniae* strains, whereas the infected bighorn sheep herds typically carried only one or two strains. This diversity of strains within domestic sheep likely adds considerably to the risk posed by domestic sheep-bighorn sheep contacts due to the high likelihood that a genetic strain to which the bighorn sheep are not immune will be encountered. A final interesting finding from this study was that strains of *M. ovipneumoniae* carried by domestic sheep were clearly distinguishable from strains carried by domestic goats, indicating that the strain type analysis could indicate the likely source host of this pathogen.

III. Scientific Explanation of Risks to Bighorn Sheep from Domestic Sheep.

32. Bighorn sheep pneumonia occurs when *M. ovipneumoniae* infection is acquired by a susceptible bighorn sheep host following direct or close contact with another animal infected with viable *M. ovipneumoniae*. *M. ovipneumoniae* is unable to survive in the environment for more than a few minutes, so to become infected a susceptible animal must either directly contact the infected host (nose-to-nose contact) or be contacted by respiratory droplets within seconds after they are shed by the infected host (for example, immediately after the host coughs or sneezes).

33. Pathogen transmission is not visible, and its detection requires use of specific diagnostic tests; newly infected animals don't exhibit signs of the disease until after the incubation period. For *M. ovipneumoniae*, transmission is detectable by realtime PCR tests as soon as 24 hours after infection, but its relatively long incubation period means that a week or more must elapse before the newly infected animal begins exhibiting disease signs (nasal discharge, coughing, etc.). Death from pneumonia may occur from a few weeks to several months after pathogen transmission.

34. The delays between infection and disease onset, and between disease onset and death, make it impossible to identify the specific moment of transmission or the specific animals involved in a *M. ovipneumoniae* transmission event.

However, the MLST strain typing method that we have developed for *M. ovipneumoniae* offers an alternative method to determine the source when this pathogen is newly introduced into bighorn sheep, since finding that MLST strain in a host within or near the bighorn sheep range strongly suggests that the strain was transmitted from that source.

35. For example, given a new pneumonia outbreak in which *M. ovipneumoniae* is detected, the strain can be compared to *M. ovipneumoniae* strains found in potential sources in the region. If no regional bighorn herds carry the strain, then the source must be an infected individual of some other animal host. Potential source animals/herds can then be systematically screened for the outbreak strain. Using these methods, we have successfully identified the sources of *M. ovipneumoniae* strains that were transmitted from domestic sheep and domestic goats, under range conditions, that resulted in bighorn sheep pneumonia outbreaks. This approach offers good potential to clarify the pathogen sources that cause bighorn sheep pneumonia outbreaks; however, it is laborious, time-consuming, and expensive, and dependent on the willingness of the owners of potential pathogen sources to permit sampling and typing of the strains of *M. ovipneumoniae* carried by their animals.

36. *M. ovipneumoniae* induces disease indirectly; the infection damages the muco-ciliary clearance defense mechanisms that normally clears small numbers

of inhaled bacteria from the mouth and throat. In the presence of *M. ovipneumoniae* infection, these inhaled bacteria are not cleared but instead infect and multiply in the lung tissues and airways, and it is these multiple secondary infections that cause severe disease or death. The severity and course of the disease within an individual infected bighorn sheep result from the extent, diversity, and virulence of the secondary infections that occur, and these vary from animal to animal.

37. As discussed above, bighorn sheep that do not die during a pneumonia outbreak return to apparent good health over the subsequent months but may carry *M. ovipneumoniae* for long periods in their nasal passages. In some herds these persistently shedding individuals serve as a source of infection to lambs born in subsequent years, triggering annual pneumonia outbreaks that may kill a high percentage of the lambs for years or even decades after the initial outbreak. It is now clear that such recurrent lamb pneumonia outbreaks may threaten extirpation of the affected bighorn sheep herds, as the herds are unable to produce enough offspring to recover from the disease event. In addition, carriers of *M. ovipneumoniae* may infect other bighorn herds during movements within metapopulations.

38. After decades of research to clarify this complex disease, the risk posed by pneumonia outbreaks to bighorn sheep populations is now recognized to

flow directly from the infection of the bighorn sheep population with *M. ovipneumoniae* from another animal host. Such infections are relatively infrequent, as a series of uncommon events must occur for pathogen spillover to occur. However, if sources of infection are prevalent within or near the bighorn home ranges, the risk of infection is cumulative across years. When I began studying bighorn sheep pneumonia, the three bighorn herds in the Yakima WA area (Umtanum, Tieton, and Cleman Mountain) were all free of *M. ovipneumoniae* and lacked evidence of significant respiratory disease. Since then, Umtanum was infected with *M. ovipneumoniae* in 2009, Tieton in 2013, and Cleman Mountain in 2020.

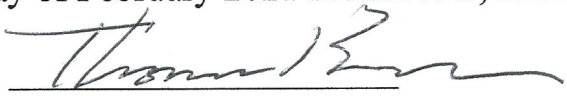
39. These outbreaks differed in their severity, and each was associated with a different genetic strain type of *M. ovipneumoniae*. All three of the outbreak-associated strain types belonged to the group of strain types typically detected in domestic sheep, rather than domestic goats. None of the *M. ovipneumoniae* genetic strain types associated with these outbreaks had previously been detected in any bighorn sheep population anywhere in western North America, ruling out bighorn sheep to bighorn sheep transmission as the source of infection of these herds. Together, these three outbreaks indicate a high regional risk of spillover *M. ovipneumoniae* transmission to bighorn sheep, suggesting that increased efforts to

remove sources of this pathogen will be required to permit recovery of these regional bighorn sheep populations.

40. In conclusion, over the last decade the science around pneumonia infections of bighorn sheep has evolved significantly. It has established that *M. ovipneumoniae* is the agent that causes respiratory disease episodes in bighorn populations, and that many strains of *M. ovipneumoniae* exist such that infection with one strain will not prevent future infection by another strain. Domestic sheep carry many strains of *M. ovipneumoniae* and together with domestic goats are the most likely source of transmission to bighorn sheep. Because of the potential for a significant die-off within an infected bighorn herd, spread of disease to other herds, and long-lasting impacts on lamb recruitment within infected populations, the consequences of *M. ovipneumoniae* transmission from a domestic sheep to a bighorn sheep are severe. Such consequences are evident in the three Yakima area bighorn herds, which all have experienced infection of *M. ovipneumoniae* from domestic sheep and could experience further disease impacts from future transmission from domestic sheep or goats within or near their home ranges.

Pursuant to 28 U.S.C. § 1746, I declare under penalty of perjury that the foregoing is true and correct.

Signed this 26th day of February 2021 in Moscow, Idaho.



Thomas Besser