

Comments on the 'Risk Analysis of Disease Transmission between Domestic Sheep and Bighorn Sheep on the Payette National Forest, 2006'

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Currently the main concern amongst the majority of Wildlife Biologists tasked with the management of Bighorn Sheep (BHS) populations throughout the western US is that of disease transmission from domestic livestock, primarily sheep, to BHS. The perception is that any contact between domestic sheep (DS) and BHS will invariably lead to disease and death in the BHS. For more than a century this legend was perpetuated until in the 1990s a scientific approach was attempted to rule DS in or out as a cause for BHS disease events and die-offs. To date not a single report has been published where disease transmission from DS to BHS was proven to be the cause for morbidity and mortality in BHS in their natural habitat. Both the Sierra Nevada Bighorn Sheep Recovery Plan and the Payette NF Risk Assessment completely ignore this fact. Both plans are almost identical in several sections.

In the literature review section many of the statements are very familiar, but have never been adequately documented. Where are accurate historical accounts to be found on these mass die-offs in the 1800s and early 1900 hundreds? Historical sources about wildlife in the Western US differ greatly in their statements about the abundance of wildlife. The only ever documented mass die-off of wildlife and domestic livestock took place in Southern Africa in 1898 after the introduction of Rinderpest through a shipment of cattle. Sir Arnold Theiler can be credited with documenting the event and diagnosing the cause of this disaster.

In the chapter on 'Effects of Disease in Bighorn Populations' I find another statement which even though completely hypothetical is often touted as a dogma. Against 'common' believe no domestic livestock species has ever been selected for disease resistance. Selection parameters were fertility, muscle mass, milk yield, fiber length and quality etc. Undoubtedly domestic livestock species have adapted to pathogens in their environment, and so have wildlife species. We are all familiar with the concept of natural selection. The mechanism of adaptation of mammalian immune systems should be similar if not identical for livestock and wildlife; particularly in those cases where species are closely phylogenetically related. With regard to this observation it is clearly troubling to read that BHS polymorphonuclear leukocytes are highly susceptible to leukotoxins secreted by *Mannheimia haemolytica* (Silflow & Foreyt, 1994; Liu et al., 2006). Even though the authors do not present conclusive evidence that one ligand (CD18) and its expression level (which was not studied) would result in the observed cellular response *in vitro*, the question arises as to when an organism/species should be considered (partially) genetically immuno-compromised. What are the consequences for BHS conservation if the BHS is indeed genetically immuno-compromised?

The authors of the Payette NF Risk assessment state that the all-age losses of BHS in the late 1800s and early 1900s coincided with the introduction of DS for grazing. The

authors fail to mention that BHS have their own species of Psoroptic mites which they harbor until today, whereas in DS Psoroptic mange has been eradicated. The authors cite several *Pasteurella* pneumonia transmission studies and state that BHS and DS must be kept separated in order to maintain health BHS. The studies quoted used captive BHS. Bighorn Sheep do not adapt well to captivity, which is the reason they do not breed well in captivity and their stress levels should be considered to be elevated throughout captivity (Jack Ryan, USDA-WS, personal communication). One of the most frequently cited studies on disease transmission in most papers (Foreyt et al., 1994) used 5.3×10^8 to 8.6×10^{11} colony forming units to inoculate BHS. Seven of eight inoculated bighorn sheep died from acute pneumonia within 48 hr of inoculation. The infectious dose for the majority of bacterial pathogens lies somewhere in the order of 1×10^1 to 10^4 .

The chapter on 'Management of BHS disease issues' is entirely dedicated to risks associated with BHS/DS interactions. It should be reasonably clear by now that BHS carry a sufficient number of virulent *Pasteurellaceae* and other pathogens in their respiratory tract to be prone to pneumonia in the absence of DS contact. It is obvious that the majority of the populations described in this document have had die-offs which were passed on to adjacent populations; which is to be expected, since a virulent *Pasteurellaceae* will be passed on to susceptible individuals and populations no matter if it was contracted from or evolved in a BHS or a DS. Why does this risk assessment mention nothing about a thorough investigation into the endemic risk of disease transmission within a metapopulation even though several of these disease events are described later in this document? How much longer are wildlife managers willing to ignore the single most important risk factors for BHS population, the endemic disease risk?

In BHS/DS disease transmission studies in Nevada between 2002 and 2004 several hundred *Pasteurella* isolates were cultured from BHS (sick and healthy) and DS. To date more than 200 strains of *Pasteurella multocida* and *trehalosi*, as well as *Mannheimia hemolytica* have been genotyped using Amplified Fragment Length Polymorphism. Genetic diversity is significant in both BHS and DS derived isolates. None of the isolates were shared between BHS and DS (Rink et al, unpublished).

Pasteurella pneumonia in domestic livestock is called 'Shipping fever'. The upper respiratory tract of most domestic and wild ungulates is colonized by *Pasteurella* spp (Ward et al., 1997), under stressful conditions, such as shipping; the pathogen can overwhelm the host's immune system. Drs. ACS Ward and GC Weiser, Caine Veterinary Teaching and Research Center, The Caine *Pasteurella* Research Laboratory, Caldwell, Idaho have published widely on prevalence, phylogenetic diversity, pathogenicity, transmission and identification of *Pasteurellaceae*. Just like in both versions of the Sierra Nevada BHS recovery plan their work has been ignored during the preparation of this risk assessment!

On page 8, the final paragraph in the section on the Hells Canyon metapopulation states: Disease, primarily pneumonia initiated by contact with DS, has been identified as the key factor limiting bighorn restoration in Hells Canyon. Who proved it, how and where is it published? Statements like this are not only false, but the biggest obstacle to a rational, science based approach to BHS management and restoration! Eliminating

domestic sheep grazing will probably have zero impact on the health status of Bighorn Sheep populations in the Western United States.

As to the panelists review of 'risk', it leaves me confused and bewildered. The entire risk assessment is based on one unproven and most likely false assumption, that of inevitable disease transmission on BHS/DS contact. In the 15th and early 16th century many a conclave was held and all attendees, mostly highly educated men, decided that the earth was a disk. This document is oddly reminiscent of reports from these councils.

On a more positive note I have to mention that the approach to investigating the currently ongoing disease outbreak in Hells Canyon (July 2006) is a giant leap in the right direction. The outcome of this investigation will be eagerly awaited and very significant for both the Sheep Industry and Bighorn Sheep restoration.

References:

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