Bighorn sheep (*Ovis canadensis*) diseases: a brief literature review and risk assessment for translocation

SHELLI DUBAY, Arizona Game and Fish Department, 2221 W. Greenway Rd., Phoenix, AZ 85023, USA
HELEN SCHWANTJE, Ministry of Water, Land and Air Protection, PO Box 9338, 2975 Jutland Road, Victoria, British Columbia, V8W 9M1, Canada
JIM DEVOS, Arizona Game and Fish Department, 2221 W. Greenway Rd., Phoenix, AZ 85023, USA
TED MCKINNEY, Arizona Game and Fish Department, 2221 W. Greenway Rd., Phoenix, AZ 85023, USA

Abstract: Prior to European settlement in western North America, bighorn sheep (*Ovis canadensis*) were more widespread and abundant than they are today (Buechner 1960). The species arrived via the Bering land bridge approximately 70-100,000 years before the present (YBP) (Kurten and Anderson 1980) and slowly spread to occupy most mountainous regions of western North America from southern British Columbia and Alberta, Canada to the Cape of Baja California and northern Sierra Madre in Mexico (Brown 1989). Based on fossil records, it is likely that bighorn sheep arrived in the southwestern United States at the end of the Pleistocene era approximately 9-12,000 YBP (Findley et al. 1975). It is clear that bighorn sheep underwent dramatic declines in both occupied area and numbers throughout their range in North America in the 3 decades prior to 1900. The most probable cause of declines in this era was the introduction of domestic sheep with a suite of diseases to which bighorn sheep were naïve (DeForge et al. 1981, Brown 1989, deVos 1989). Subsequent to 1900, bighorn sheep population declines continued due to several causes including habitat fragmentation and degradation, unregulated harvest for trophies and subsistence, and competition with domestic livestock. One strategy to repatriate bighorn sheep populations is translocation of groups from healthy source populations to repopulate vacant historic habitat. Translocation is also used as a management tool to bolster populations that are below demographic objectives. Managers overseeing translocations need to be cognizant of the potential to introduce diseases when moving animals, and their potential impacts on indigenous wildlife or domestic livestock. To facilitate translocations and minimize disease risk, managers need to develop an understanding of diseases that play roles in bighorn sheep demographics, and develop methods to minimize any risk to bighorn sheep, other wildlife, and livestock. This is particularly important when managers move bighorn sheep between jurisdictions and across international boundaries (typically Canada to the U. S., and bi-directional from U. S. – Mexico). In this paper, we review several diseases of livestock and bighorn sheep and propose recommendations for health screening of bighorns to minimize disease risks to animals in the recipient area and to aid in reestablishing healthy bighorn sheep populations.

Key words: bighorn sheep, diseases, risk, translocation, serology

INTRODUCTION

Translocation of an animal and its associated organisms, including bacteria, viruses, and internal or external parasites, can threaten the health of indigenous wild species or domestic livestock. In addition, the effects of stress on the immune system of animals while captured and held, even in short term captivity before release, may increase this risk. However, the risk can be assessed in advance and substantially reduced if timely veterinary precautions

are taken (Woodford 2000). Precautions include a clinical evaluation of the health status of source animals and those at the translocation destination, appropriate health screening procedures, consideration of the legal and veterinary restrictions of wild animals to and from certain geographical areas or populations and, when necessary, pre-release treatment and immunization. The translocated animals as well as the indigenous wildlife in the reception area should undergo health screening. Once a wild animal has been released into the wild it is very rarely possible to recover it or the potential pathogens it may have carried (Woodford 2000).

Several parasites, bacteria, and viruses are reported to cause disease in bighorn sheep (*Ovis canadensis*), and some have been involved in large-scale epizootics in populations in the western United States and Canada (Spraker 1977, deVos et al. 1980, King and Workman 1983, Onderka and Wishart 1988, Schwantje 1988). Singer et al. (2001) used empirical models to predict the effects that disease epizootics and habitat patch size might have on overall viability of bighorn sheep populations. They predicted that populations with 250 or more sheep were able to withstand disease epizootics much better than small populations, but disease could have significant impacts on populations overall. Gross et al. (1997) also investigated the impact of disease on bighorn sheep via population models. They concluded that contiguous patches of habitat were the most important variable when determining the likelihood of extinction for a population. However, diseases influenced extinction rate; large populations occupying large contiguous patches were not insulated from disease-induced extinction. When multiple disease epizootics were added to the model, the likelihood of extinction increased dramatically. Therefore, an important part of bighorn sheep management is to reduce the likelihood of disease epizootics.

Jessup (1985) discussed common livestock diseases that affect bighorn sheep, most of which are commonly present in domestic sheep flocks and some are also found in domestic cattle and goats. There are many reports of single or multiple infectious organisms isolated from bighorn sheep following contact with domestic animals. Numerous accounts document fatal pneumonia epizootics, usually associated with *Pasteurella* (*Mannheimia*) infections after such contact (Monello et al. 2001). Viral and/or bacterial pneumonia and/or scabies mite infestations transmitted to bighorn sheep from domestic sheep have been implicated in epizootics in Colorado, Wyoming, Arizona, New Mexico, Alberta, and British Columbia (Lange et al. 1980, Jessup 1985, Onderka and Wishart 1988, Schwantje 1988, Ward et al. 1997). In addition, bluetongue, contagious ecthyma, and parainfluenza 3 viruses were identified as potential causes of decline in bighorn sheep herds. Clark et al. (1985) found evidence of exposure to parainfluenza-3, *Protostrongylus* sp. lungworm, bluetongue and epizootic hemorrhagic disease viruses, respiratory syncytial virus, bovine viral diarrhea, and contagious ecthyma virus in 18 herds of desert bighorn sheep in California. Evidence for exposure to *Brucella* sp. and *Leptospira* spp. was not found in this study.

Exposure to infectious organisms may not result in obvious mortality, but animals moved from one jurisdiction to another can result in infections of new populations, particularly if these populations are naïve or stressed. The susceptibility of bighorn sheep, which originated in the New World,

to disease agents of domestic livestock from the Old World is high. This is likely because bighorn sheep did not co-evolve with diseases common to domestic sheep and cattle that were selectively bred to survive intensive husbandry and infectious diseases that exist with close contact (Technical Staff, Desert Bighorn Council, 1990). Thus, bighorn sheep are exposed to pathogens to which they are not adapted when domestic animals come in contact with them on rangelands. In addition, Foreyt and Evermann (1988) found that bighorn sheep neutrophils were much less capable of killing bacteria *in vitro*. Bighorn sheep and domestic sheep are closely related through behavior and genetics and have been known to seek each other out on ranges. These facts combine to create a high risk of fatal disease exposure for bighorn sheep when in contact with domestic sheep. This review will discuss diseases of bighorn sheep that should be considered by managers prior to and during translocation programs.

SPECIFIC DISEASE ASSESSMENTS

Contagious ecthyma:

Contagious ecthyma (CE), orf, or sore mouth, is a parapoxvirus infection that can potentially affect many ungulate species. It has been seen in domestic sheep and goats for over 200 years and recognized in bighorn sheep since 1954 (Thorne et al. 1982, L'Heureux et al. 1996). Symptoms include scab forming sores and localized swelling, usually around the mouth, but also around the udder and coronet bands in some animals. Sheep may be affected year round, with increased numbers of cases in young animals in spring and summer, or following mixing of animals, such as during breeding season. Generally, virus enters the skin of the mouth through abrasions caused by mechanical insult, such as thorns on plants or abrasive materials, such as salt blocks. Visible signs of infection are seen approximately 4 days post-inoculation when domestic sheep are experimentally infected with virus (Robinson and Balassu 1981). Bighorn sheep in a national park in Canada were diagnosed with CE near salt used for road de-icing (Blood 1971). Bighorn sheep often concentrate at salt blocks or road surfaces during winter. Infected and uninfected animals use salt blocks concurrently, thereby transferring virus material to the substrates and then to naïve bighorn sheep (Blood 1971). Scab material exposed to the environment can hold viable virus for long periods of time, even years. Infection can occur at sites in the absence of salt sources. In the late 1990s, a group of adult rams with severe CE lesions was observed along a highway in British Columbia where bags of livestock grain had been dumped (H. Schwantje, British Columbia Ministry of Water, Land and Air Protection, unpublished data). Other herds in British Columbia have had small epizootics of mild to moderate CE with no obvious potential sources of infection; mortality was not reported in these cases. In addition, severe CE has been reported in British Columbia bighorn sheep herds in adult survivors or lambs born in the first two or three years following pneumonia epizootics (H. Schwantje, unpublished data). It is thought that once bighorn sheep are infected with CE as lambs, they are afforded some immunity against the virus as adults (Blood 1971, Thorne et al. 1982, King and Workman 1983). Bighorn lambs are usually more seriously affected than adults and sores on the muzzle make nursing painful. Lesions usually disappear within 4 weeks of onset, but occasional deaths due to CE are recorded (Thorne et al. 1982). Samuel et al. (1975) reported

that 2 bighorn sheep in Waterton Lakes National Park, Alberta and 1 mountain goat (*Oreamnos americanus*) from Kootenay National Park, British Columbia were infected with debilitating CE infections. Several others with lesions were found dead, suggesting that CE infection could be fatal. Affected animals were found near artificial sources of salt. L'Heureux et al. (1996) investigated CE infection in lambs in Alberta, Canada and concluded that infected lambs were lighter in mass than uninfected lambs, but disease did not influence lamb survival. Given that serologic exposure to CE does not indicate current viral infection, only previous exposure, the presence of antibodies against CE should not impede translocations of bighorn sheep. To the contrary, clinically normal bighorn sheep with antibodies against CE may be afforded some protection if the herd in the translocation area has active CE in the population. **STATUS – Widespread and posing little risk.**

Bluetongue and epizootic hemorrhagic diseases:

Bluetongue (BTV) and epizootic hemorrhagic disease (EHD) are closely related viral diseases that can impact many free-ranging and domestic ungulates (Thorne et al. 1982). The viruses are transmitted by biting midges of the genus *Culicoides*. Epizootics usually occur near water in the late summer and fall because the midges require water to reproduce. Affected animals can die acutely or demonstrate increased respiration rates, weakness, diarrhea, and hemorrhages in most organs (Thorne et al. 1982). EHD is generally thought to be less pathogenic in bighorn sheep than BTV, but Noon et al. (2002) identified hemorrhagic disease in 2 bighorn sheep carcasses in Arizona, and BTV virus was isolated from 1 animal while EHD was isolated from the other. Hemorrhages were found in several organs including conjunctiva, heart, and rumen in both cases. Bighorn sheep deaths in California and Wyoming have been attributed to BTV, and antibodies against both EHD and BTV have been documented from bighorn sheep in Arizona (Jessup 1985, Heffelfinger et al. 1995). Robinson et al. (1967) found that severe pneumonia debilitated a bighorn sheep ram in Texas. The ram had hemorrhages in the brain as well. The infected lungs were used as an experimental inoculum for 2 domestic sheep and 1 contracted severe pneumonia and died. Both domestic sheep tested positive for antibodies against BTV confirming the diagnosis. Robinson et al. (1967) suggested that contact with domestic sheep could be responsible for bluetongue in the bighorn sheep. Antibodies against BTV and EHD have been detected in many free-ranging species including bighorn sheep with no clinical signs, suggesting that the viruses are enzootic in much of the western United States (Thorne et al. 1982). Bluetongue and EHD are considered reportable foreign animal diseases in Canada. The vector *Culicoides sonorensis* is resident in western Canada, however only sporadic late summer mortality has been reported in wild deer and occasionally bighorns, with no apparent maintenance of the viruses from year to year. Bluetongue serotype 11 or EHD serotype 2 have caused outbreaks in southern Alberta (1962) and in the Okanagan valley of southern British Columbia (1975, 1987, 1988, 1999) (Dulac et al. 1989, Pasick et al. 2001). To ensure that Canada retains its BTV-free international status, the Okanagan valley has special zoning for livestock with a federal surveillance program in place. BTV and EHD are reported commonly in

deer mortality events in the Western and Southwestern United States and can be considered widespread on a seasonal basis (Gaydos et al. 2002). It is likely that exposure to the North American serotypes of BTV and/or EHD may provide animals some immunity and serological evidence does not indicate current disease status, especially if clinical symptoms are not evident at time of capture (Thorne et al. 1982). **STATUS – Widespread, poses health risk in areas where these diseases are absent or to naïve animals being translocated to enzootic area.**

Parainfluenza 3:

Parainfluenza 3 (PI3) is common to domestic sheep and cattle, and free-ranging animals that come in contact with domestics can be exposed to the virus (Jessup 1985). PI3 can cause pneumonia in domestic animals but it is considered to be of low pathogenicity. The virus can be part of the "shipping fever" syndrome where combined infections of other viruses and bacteria invade respiratory tracts of stressed animals and cause severe lung infections and death. Few cases of mortality due solely to PI3 infection have been cited in free-ranging animals, but antibody titers have been described from several sympatric free-ranging species. Zarnke and Erickson (1990) identified antibodies against PI3 in bison (*Bos bison*) in Alaska, and prevalence increased from 0 % in 1975 to nearly 100 % in 1983 to 1988 without clinical disease in the herd. The virus was likely introduced to the bison from cattle that recently grazed adjacent to the bison herd. Free-ranging fallow deer (*Cervus dama*) in Italy have been shown to harbor antibodies against PI3 as well. Clinical signs of infection were not observed and cattle were grazed on the reserve where fallow deer were sampled, suggesting that cattle introduced the virus to wildlife (Giovannini et al. 1988). Sadi et al. (1991) investigated potential causes of high mortality among white-tailed deer (*Odocoileus virginianus*) on Anticosti Island, Quebec in 1985. Sera from white-tailed deer were tested for antibodies against several pathogens, and antibodies against PI3 were found in between 82 and 84 % of the animals sampled over a 3-yr period. Antibodies against bovine herpesvirus-1 increased in the population while the herd was experiencing high mortality, suggesting that herpesvirus was responsible for increased mortality and that PI3 was enzootic in population and contributed little to population declines. Antibodies against PI3 have been detected in bighorn sheep in the western United States (Sandoval et al. 1987) and British Columbia (H. Schwantje, unpublished data). The virus has been isolated from clinically ill bighorn sheep in California (Jessup 1985) and from mortalities during pneumonia epizootics in British Columbia (H. Schwantje, unpublished data). Isolates from the British Columbia mortalities were obtained from lungs affected by multiple organisms. PI3 was also implicated in the pneumonia death of a captive bighorn sheep in Wyoming (Parks et al. 1972). No serologic evidence of exposure to PI3 was found in 20 desert bighorn sheep (*O. c. mexicana*) in Arizona during 2000-2002 (T. McKinney, Arizona Game and Fish Department, unpublished data). In general, PI3 infection alone is considered a minor disease of free-ranging wildlife, with many species being exposed and little evidence of mortality without other pathogens being involved (Zarnke and Erickson 1990). **STATUS – Widespread and believed to pose little risk to bighorn sheep. Alone, PI3 may not be important but in combination**

with other pathogens and/or stressors infection may be fatal.

Respiratory syncytial virus:

Respiratory syncytial virus (RSV) is a common organism in domestic cattle populations and is responsible for lung infections and mortality, especially in naïve animals (Lehmkuhl and Cutlip 1979). It is also recognized in domestic sheep, and RSV was isolated from a domestic sheep with rhinitis (Evermann et al. 1985). When RSV virus was re-inoculated into naïve lambs alone or with *Pasteurella haemolytica* bacterial isolates, lambs developed mild conjunctivitis and mild histological inflammatory changes in the lung. The virus has also been identified as a potential pathogen in free-ranging wildlife. Johnson et al. (1986) tested blood samples from hunter-harvested mule deer (*O. hemionus*) and white-tailed deer in Nebraska for antibodies against RSV. Twenty-nine percent of mule deer samples showed exposure, whereas 37 % of white-tailed deer samples had antibodies against RSV. Seroprevalence for RSV antibodies in these deer mimicked those of cattle in Nebraska. Dunbar et al. (1985) identified antibodies against RSV in 187 of 447 (42 %) bighorn sheep sera from 9 western states from 1977 through 1985. Bighorn sheep from several states sampled had severe pneumonia infections and some individuals died from pneumonia. An RSV isolate was cultured from a clinically ill bighorn lamb in Colorado as well (Spraker and Collins 1986). This virus was also isolated from several mortalities during pneumonia epizootics in bighorn herds in British Columbia. All of these isolates were made from lungs affected by multiple pathogens (H. Schwantje, unpublished data). Foreyt and Evermann (1988) inoculated 5 bighorn sheep lambs (3 vaccinated against RSV and 2 unvaccinated) with an RSV isolate from a domestic lamb with rhinitis. Clinical signs of pneumonia were not observed in either vaccinated or unvaccinated lambs, but antibody titers against RSV were identified from all animals. It seems that RSV alone is not an obligate pathogen in bighorn but further research is needed. **STATUS – Widespread and believed to pose a low risk to bighorn sheep, but information is lacking. Alone, RSV may not be important but in combination with other pathogens and stressors may be fatal.**

Infectious bovine rhinotracheitis:

Infectious bovine rhinotracheitis virus (IBR) belongs to the herpesvirus group and causes respiratory disease in cattle (Richards 1981). The virus is ubiquitous in cattle and vaccines have been developed to combat clinical illness. Virus is found in secretions from the respiratory, ocular, and reproductive tracts, but experimentally infected deer showed limited ability to shed virus. Infected deer show depression, anorexia, excessive salivation, and increased respiration. Ingebrigtsen et al. (1986) investigated IBR exposure of white-tailed deer in Minnesota. They tested 504 sera from 1976-1980 and 15 % had antibodies against IBR, with exposure being statewide. Few studies have investigated IBR in bighorn sheep, but serologic evaluation of 20 desert bighorn sheep in Arizona showed no evidence of exposure to IBR (T. McKinney, unpublished data). Hampy et al. (1979) tested 6 Barbary sheep (*Ammotragus lervia*) for antibodies against IBR and 1 had a titer of 1:4, 1 had a titer of 1:8, and another had a titer of 1:16. Similar titers have been documented in bighorn sheep herds in British Columbia as well (H. Schwantje, unpublished data). Titers

lower than 1:16 are often considered negative. Therefore, these levels are of doubtful significance. IBR has not been implicated in bighorn sheep epizootics in the literature, and likely is not a significant cause of mortality. **STATUS – Widespread and appears to pose little health risk to bighorn sheep.**

Bovine viral diarrhea:

Bovine viral diarrhea (BVD) is caused by a *Pestivirus* and was first described in cattle in 1946 (Richards 1981). The virus is quite resistant to sunlight, freezing, and desiccation, and is spread several ways: 1) through food and water contaminated with feces, urine, or nasal discharge from infected animals, 2) through inhalation of aerosols containing virus, 3) from pregnant animal to fetus. Clinical signs in cattle include fever, depression, alimentary tract erosions, dehydration, diarrhea, weak neonates, and abortion. BVD virus is immunosuppressive and can predispose herds to epizootics of concurrent infections. An epizootic in mule deer and white-tailed deer in North Dakota in 1955 was associated with infected cattle (Richards 1981). Dead and clinically ill deer were located within a 0.3-km radius of clinically ill cattle. Symptoms in deer include weakness, lack of fear of humans, dehydration, diarrhea, impaired vision and hearing, and convulsions, but animals appeared to recover as the epizootic progressed. Serologic surveys in New York showed that approximately 3-6 % of the deer tested had antibodies against BVD, but mule deer herds in New Mexico and Colorado had higher exposure rates, 34 % and 85 % respectively (Richards 1981). McKinney (Arizona Game and Fish Department, unpublished data) determined viral exposure via antibody levels in desert bighorn sheep in Arizona. A total of 20 animals were tested during 5 captures and none had antibodies against BVD. Elliott et al. (1994) measured antibody levels against BVD in 998 serum samples from bighorn sheep captured in California from 1978 to 1990. The highest seroprevalence for BVD was 18 %, and the lowest was 4.9 %. In Texas, Hampy et al. (1979) tested 6 Barbary sheep for antibodies against BVD, and none showed evidence of exposure. To our knowledge, BVD has not been implicated in disease epizootics in bighorn sheep, and the significance of antibody evidence of exposure to bighorn sheep health is unknown. However, since exposure is widespread, serologic evidence should not impede translocation of bighorn sheep. **STATUS – Widespread exposure. Uncertain significance and requires more research.**

Scabies:

Scabies is a parasitic mite infection of the skin and is commonly seen in certain populations of desert and Rocky Mountain bighorn sheep (*O. c. canadensis*), elk (*Cervus elaphus*), and white-tailed deer in the western United States (Thorne et al. 1982). Several mite species of the genus *Psoroptes* cause clinical disease in free-ranging wildlife. Clinical signs of disease are caused by mechanical insult from mouthparts of mites. The mites feed on serum that oozes from abrasions on the skin, and excrement and other proteins emitted from the mites cause an immune response by the host. As inflammation progresses, the host sloughs portions of the epidermis and secondary bacterial infections often occur at the site of sloughing. Ear and body scabs are seen on bighorn sheep infected with *Psoroptes* mites and large plaques of loosely attached scabs are easily lifted off the body in extreme infestations. Welsh and Bunch (1982) investigated the causes of decline

in bighorn sheep from Arizona and identified psoroptic scabies as a potential contributor to decreased population levels. Increased prevalence of scabies occurred concurrent with decreased body condition of animals in the herd. deVos et al. (1980) also identified scabies infection from bighorn sheep in Arizona. Ear lesions were seen in 2 rams and 1 ewe and serologic evidence was detected in another 5 animals in the herd. Foreyt et al. (1985) identified scabies lesions from animals transplanted to Oregon from Idaho. Transplanted animals were treated with 0.2 mg/kg body weight ivermectin to eliminate mites prior to release. Kinzer et al. (1983) used 0.5 to 1.0 mg/kg ivermectin to treat scabies in desert bighorn sheep in New Mexico. Sandoval (1980) discussed another epizootic of scabies in New Mexico, and all 5 bighorn rams harvested from San Andreas National Wildlife Refuge in 1978 had clinical symptoms of scabies infection. The population had declined significantly prior to the hunt, and only 70 of 200 animals remained in 1979. The remaining animals were captured and given emergency medical treatment including dipping in toxaphene solution. Several bighorn sheep had scabies lesions over their entire body, suggesting that scabies contributed to the population decline in New Mexico. Scabies infections are not known to occur in wild sheep in Canada and sampling of bighorn sheep translocated to the United States has confirmed these findings (H. Schwantje, unpublished data). Naïve Canadian bighorn sheep have become severely infected with scabies once translocated into infected populations. Given the severity of scabies infection and the ease of diagnosis in most cases, all translocated bighorn sheep should be examined for scabies lesions and treated with an effective medication prior to release into a new area. Animals from populations without evidence of the mite should not be relocated to endemic areas. **STATUS – Localized with potential for substantial morbidity and mortality, especially in naïve animals.**

Anaplasmosis:

Anaplasmosis is a vector-borne rickettsial infection of cattle and free-ranging ruminants (Thorne et al. 1982). The causative agent in cattle is *Anaplasma marginale*, but *A. ovis* infects domestic sheep and goats, and wildlife species. Anaplasmosis is transmitted by a number of tick species and biting flies and is most prevalent in the Southeast, intermountain West, and California in the United States. Infected animals develop anemia when rickettsia destroy red blood cells, but animals usually recover and remain carriers of the parasite for several months or years. *Anaplama ovis* may be more pathogenic than *A. marginale*, particularly during periods of stress. Clinical signs of infection are usually mild in wildlife, but lack of appetite and weakness are identified as signs in black-tailed deer (*O. hemionus columbianus*). Wild ruminants can act as reservoirs for domestic livestock. *Anaplasma marginale* was inoculated into 2 bighorn sheep and red blood cells in 1 animal became infected with the organism, but clinical disease was not seen in either animal (Goff et al. 1993). Tibbitts et al. (1992) inoculated 2 bighorn sheep with an *Anaplasma ovis* isolate from clinically ill domestic sheep. Both inoculated animals developed severe anemia and became lethargic. Given that the animals were given a very high dose of infected cells (2×10^9), that the isolate may have been relatively virulent, and that the bighorn sheep were stressed due to confinement and frequent handling, clinical disease may have been

accentuated. Goff et al. (1993) isolated *A. ovis* from bighorn sheep in California and then inoculated infected blood into 1 splenectomized domestic sheep, 1 splenectomized calf, and 1 intact bighorn sheep. The bighorn sheep and domestic sheep developed anemia and were treated with antibiotics. The calf showed no evidence of infection. It is likely that *Dermacentor* spp. ticks transmit *Anaplasma* spp. to bighorn sheep in California. Jessup et al. (1993) investigated the presence of antibodies against *Anaplasma* spp. in bighorn sheep herds in California. All 20 Rocky Mountain bighorn sheep tested had antibodies against *Anaplasma* spp., 11 of 18 peninsular bighorn sheep (*O. c. cremnobates*) had antibodies, and 0 of 20 California bighorn sheep (*O. c. californiana*) had antibodies. *Anaplama ovis* was thought to be responsible for antibody responses in these bighorn sheep, and differences in vector and host abundances were likely responsible for differing prevalence rates with geographic region and bighorn sheep subspecies. Jessup et al. (1993) believed that naïve bighorn sheep may become infected with anaplasmosis from carrier animals after a translocation event, if vector populations exist in the translocation area. Anaplasmosis is considered to be a foreign animal disease in Canada, and there have been no isolations of either *Anaplasma* sp. in wild ruminants, including bighorn sheep (H. Schwantje, unpublished data). Although bighorn sheep have been experimentally infected with *Anaplasma* sp., it is unlikely that they are important carriers of disease (Thorne et al. 1982), and Kuttler (1981) stated "the greatest importance of wild animals with regard to anaplasmosis is their potential as secondary or reservoir hosts." Given that, evidence of exposure to *A. ovis* or *A. marginale* should not influence bighorn sheep transplants. **STATUS – Widespread but appears to pose little direct health risk for bighorn sheep.**

Johne's Disease or Paratuberculosis:

Paratuberculosis is a bacterial infection caused by *Mycobacterium avium* subsp. *paratuberculosis* and causes chronic enteritis in cattle, sheep, goats, llamas, camels and some free-ranging ruminants (Timoney et al. 1988, Williams 2001). The primary lesions are observed in the digestive tract and infected individuals show deterioration of body condition and diarrhea (Williams 2001). Bacteria are shed in feces and naïve animals are exposed by ingesting contaminated feed or water. Individual carriers can shed the bacterium in feces for years after infection. Paratuberculosis has been documented in farmed deer and in free-ranging Tule elk in California, but free-ranging wildlife populations are rarely impacted by the disease (Williams 2001). Williams et al. (1979) documented 6 cases of paratuberculosis in bighorn sheep in Colorado. Affected individuals were emaciated, had rough hair coats, and had dried feces from the perineum to the lower rear legs. Five of the 6 cases were clinical, but 1 case was subclinical suggesting that carriers could expose herdmates to infection in free-ranging wildlife. These bighorn sheep were thought to acquire infection naturally, perhaps from infected domestic livestock in the area. Williams et al. (1983) orally inoculated Rocky Mountain elk, mule deer, white-tailed deer, bighorn sheep X mouflon (*O. musimon*), and domestic lambs with a *M. avium paratuberculosis* isolate from the bighorn cases documented in 1979. All animals exposed became infected but clinical disease with diarrhea occurred only in mule deer. It was

hypothesized that some free-ranging species could become infected with paratuberculosis by sharing ranges with infected domestic livestock or wild ruminants. In addition, bighorn sheep were thought to maintain the disease in the population without a re-introduction of the disease into that population. **STATUS – Causes isolated problems in bighorn sheep. Managers and veterinarians need to monitor animals for clinical signs of paratuberculosis if the disease has been documented in that herd in the past and not use these herds for translocations.**

Leptospirosis:

Leptospirosis is a contagious bacterial disease of animals including humans, and is due to infections of members of the genus *Leptospira* (Thorne et al. 1982). Several serovars, or serologic strains, can cause clinical disease. The severity of disease ranges from asymptomatic to fatal, depending upon the host and serovar involved. Clinical signs of disease may include fever, jaundice, loss of appetite, abnormally colored urine, and abortion. Bacteria are primarily transmitted from animal to animal in water contaminated with infected urine, but bacteria also invade broken skin and mucous membranes including those of the eyes, intestinal tract, genital tract, and nose. Animals usually recover from disease but can carry and shed bacteria after clinical signs cease. Leptospires are found worldwide in numerous domestic and wild species. Serologic surveys are commonly used to determine the presence of *Leptospira* spp. in free-ranging animals (Thorne et al. 1982). Fournier et al. (1986) measured antibody levels in 258 sera from white-tailed deer in Ohio. Eighteen animals (7 %) had antibody titers against at least 1 of 5 serovars identified. Given that white-tailed deer shed bacteria for approximately 30 days post-experimental infection, a much shorter interval than carnivores, deer are less likely to transmit disease to other wildlife. New et al. (1993) evaluated 590 blood samples from white-tailed deer in Tennessee for antibodies against *Leptospira* spp., and 21 % had antibody reactions to at least 1 serovar. They concluded that most infections are probably clinically mild and unlikely to influence populations in Tennessee. Hampy et al. (1979) investigated the presence of antibodies against *Leptospira* in 12 Barbary sheep and 11 mule deer and no antibodies against leptospirosis were detected. Chilelli et al. (1982) measured antibody titers against *Leptospira* spp. from 77 bighorn sheep in Arizona and only 1 animal had a titer higher than 1:64. deVos (1989) compiled serologic data for desert bighorn sheep captured from Arizona in 1985 and 1986. Three herds were tested for antibodies against *Leptospira* spp. in 1985, and antibodies were present in 1 herd (23 % of samples). In 1986, 2 herds were evaluated and antibodies against at least 1 serovar of leptospirosis were detected in animals from both herds, but clinical illness was not detected. Evidence of exposure to leptospirosis is present in several free-ranging ungulate species, but clinical illness appears to be rare. **STATUS – Widespread in many wildlife species, uncertain from bighorn sheep, but seems to pose minor health risk.**

Brucellosis:

Brucella spp. bacteria are the causative agents of brucellosis in free-ranging wildlife and domestic livestock. At least six species and more than 19 biovars of *Brucella* affect animals: 1) *B. abortus* is found primarily in cattle, elk, and bison, 2)

B. melitensis is found in domestic sheep and goats, 3) *B. suis* is found in swine, caribou (*Rangifer tarandus*), and moose (*Alces alces*), 4) *B. neotomae* is found in woodrats (*Neotoma lepida*), 5) *B. ovis* is found in domestic and wild sheep, and 6) *B. canis* is found in dogs (Thorne 2001). *Brucella* spp. are maintained in primary hosts through horizontal or vertical transmission, but accidental transmission can occur into secondary hosts through ingestion or contact with contaminated materials. These diseases are of economic importance worldwide due to their effect on the livestock industry and their zoonotic potential. Infection is most often linked to reproductive problems, particularly abortion or birth of nonviable offspring, but infertility can also result from brucellosis infections. *Brucella abortus* is the primary species involved in free-ranging wildlife in the Greater Yellowstone Area, but *B. suis* biovar 4 has been isolated from clinically ill caribou and reindeer and occasionally from moose (Thorne 2001). Zarnke and Yuill (1981) used the rapid slide agglutination and the complement fixation techniques to test 9 bighorn sheep sera for antibodies against *B. abortus*, and none were detected. Davis (1990) reported that 9 bighorn sheep from Canada and 43 bighorns from Arizona were negative for antibodies against *Brucella* spp. Foreyt et al. (1983) tested 73 Dall's sheep (*O. dalli*) for antibodies against *Brucella* sp. using the plate agglutination test. Three animals had antibodies but the authors did not discuss potential exposure routes. Seropositive tests for *Brucella ovis* have resulted from bighorn sheep captured in Idaho and California (M. Drew, Idaho Department of Fish and Game, unpublished data), and at this time, it is unclear what positive results mean. Serological tests used for bighorn sheep were developed for livestock species and have never been validated for wild sheep, making results difficult to interpret. Brucellosis caused by *B. abortus* is a reportable disease in Canada. It was eradicated in Canadian livestock in 1985, however is present in wood bison in and around Wood Buffalo National Park. *B. suis* biovar 4 and *B. ovis* are not reportable. *B. suis* biovar 4 is restricted to certain caribou and reindeer herds and occasional secondary hosts. There appears to be no risk of transmission to livestock (S. Tessaro, Canadian Food Inspection Agency, personal communication). *B. ovis* is rare in domestic sheep with no isolations in western Canada in the past decade. Since 1990, a large number of bighorn sheep from British Columbia and Alberta have been examined serologically for *B. abortus, B. suis* and *B. ovis* by a range of serological tests performed by accredited laboratories in the United States and Canada (H. Schwantje, unpublished data). The vast majority of these tests have been negative for any *Brucella* exposure, however, some results have been considered to be "incomplete", false positive or equivocal and have resulted in live animal shipments being held for extended periods of time or the removal of animals from shipments. All sera, when retested with more specific testing methodology have been confirmed as negative. Unfortunately, all serological tests used to test for *Brucella* in bighorn sheep were developed for livestock species and have never been validated for wild sheep. Brucellosis has never been reported in wild sheep in Canada and none of the bighorn populations are in contact with species known to be infected with any *Brucella* species. Despite a small number of reactions on serologic tests in certain individuals, brucellosis has never been reported in wild sheep in Canada (H. Schwantje, unpublished data). **STATUS –**

Uncertain for bighorn sheep. Additional research needs to be conducted with bighorn sheep that are sympatric with infected elk and bison populations in endemic areas. Testing bighorn sheep from endemic areas should be considered.

Pasteurellosis:

Pasteurella spp. (*Mannheimia* spp.) are reported to be normal bacterial flora of the nasal mucosa and tonsillar crypts of both domestic and bighorn sheep (Ward et al. 1990) and are equally common in domestic cattle (Friend et al. 1977, Yates 1982, Cutlip and Lehmkuhl 1983). Some species and biotypes can cause serious pneumonia or septicemic disease outbreaks in livestock, often following environmental stress or concurrent infections (e.g., PI3). A similar pneumonic outbreak syndrome is well documented in bighorn sheep and is responsible for many population declines, and is therefore one of the most important diseases of wild sheep in general. Queen et al. (1994) examined nasal and tonsillar samples from apparently healthy bighorn sheep and domestic sheep and successfully isolated *P. haemolytica* from 5 of 5 domestic and 7 of 8 bighorn sheep tonsil samples. Although some biotypes of *Pasteurella* are considered to be normal flora, others are frequently reported in pneumonia die-offs of bighorn sheep (Foreyt and Jessup 1982, and Foreyt 1992). Cassirer et al. (1998) chronicled an epizootic that was attributed to *Pasteurella* associated pneumonia. In this epizootic, 4 of 10 herds associated with Hells Canyon in Washington and Oregon were adversely affected and approximately 325 bighorn sheep died. Prior to onset of the die-off, bighorn sheep were reported to be in excellent physical condition and some environmental stressors such as poor range conditions, adverse winter conditions, and high population levels were absent. Hibler et al. (1980) suggested that under most situations, bacteria cannot cause disease because of the lack of damaged or compromised tissues. One factor that may predispose bighorn sheep to bacterial pneumonia are heavy loads of an endemic wild sheep lungworm (*Protostrongylus stilesi*), which causes damage to lung tissue, and allows bacteria such as *Pasteurella* to invade the lower respiratory tract and cause clinical disease (Hibler et al. 1980, Spraker et al. 1984). Concurrent infections with upper respiratory viruses such as RSV and PI3 have also been implicated as predisposing factors for *Pasteurella* spp. infection (Miller 2001). A common factor seen in many bighorn sheep pasteurellosis outbreaks is close contact with domestic sheep or goats (Callan et al. 1991, Ward et al. 1997, Cassirer et al. 1998). Different biotypes of *P. haemolytica* are more pathogenic, especially to bighorn sheep. Foreyt (1989) found that *P. haemolytica* biotype T was more pathogenic for desert bighorn, and suggested that it was transferred from domestic sheep and caused clinical disease. Cassirer et al. (1998) identified a genetic similarity between isolates from at least 4 bighorn sheep and 3 feral domestic goats in the Hells Canyon epizootic. There is much to learn about pasteurellosis in bighorn sheep, yet it is clear that this disease is a major mortality factor. Bighorn sheep managers across jurisdictions consider the prevention of pasteurellosis in bighorn sheep to be a management priority and believe that the primary way to accomplish this is to ensure the separation of wild and domestic sheep. **STATUS - Many *Pasteurella* spp. and biotypes are widespread and present in most bighorn sheep and domestic livestock herds. Many**

***Pasteurella* spp. of domestic sheep origin are considered to be fatal to bighorn sheep. Those of bighorn sheep origin may present a health risk to naïve animals, but are difficult to predictably identify. The capacity to predict the effects of *Pasteurella* on either the source or recipient bighorn sheep populations is not yet available. Therefore, pre-movement culturing of bighorns in the source and recipient herds can be considered, however consideration of the disease history of the herds is more important. Of paramount importance is the prevention of contact between all domestic and wild sheep.**

CONCLUSIONS AND RECOMMENDATIONS

In general, many diseases are considered to be widespread in livestock and certain species of wild ruminants in the western United States and Canada. Others are restricted to specific geographic areas or their effects are most significant in specific species. Translocation of animals with similar health profiles between areas with similar disease risk appears to present the lowest risk to translocated animals, or to livestock and wild animals in recipient areas. We believe that movement of bighorn sheep across most jurisdictional lines poses minimal risk to wildlife and livestock in the receiving area. However, managers must be aware of potential risks that recipient area herds may pose to relocated individuals (e.g., if scabies is endemic in the recipient areas). Prior knowledge of the health status of bighorn sheep populations, consultation between wildlife and livestock management agencies, and proactive management, such as vaccination (if available, practical, and effective) or reassessment of the suitability of recipient sites are necessary to prevent certain disease epizootics. Many epizootics are much more likely to occur in bighorn sheep than in other wild or domestic species.

Although many diseases reviewed are generally of low pathogenicity to bighorn sheep, there is no way to predict when other factors can combine to predispose apparently healthy animals to disease, especially when multiple pathogens and adverse or unpredicted environmental conditions are involved. Therefore, we have compiled recommendations to minimize the possibility of disease transmission during translocation efforts. The success of any translocation depends on releasing healthy animals into areas with conditions that will promote continued health. General veterinary management protocols often recommend animal isolation or quarantine to ensure health of animals prior to or following movements. However, this technique is impossible, impractical, or dangerous with most free-ranging species. This is particularly true with bighorn sheep, because confinement increases stress, increasing the likelihood of development of pneumonia (Spraker and Hibler 1977; Spraker et al. 1984).

Specifically, we recommend:

- Due to the time required to obtain test results for many diseases, instead of relying on testing of translocated animals alone, we recommend background testing of source herds in order to increase data sets and to obtain general health profiles of the populations. Data obtained should be shared with agencies involved and a general or detailed risk assessment produced and evaluated prior to the translocation event.
- Biologists should choose standard test protocols and procedures most

appropriate for the pathogen and animal species to be tested. If necessary, encourage research for test validation in bighorn sheep.

- Wherever possible, all translocated and resident animals should have serum archived for disease profiles and retrospective analyses. This could be particularly useful in the event of post-translocation disease outbreaks.
- All captured animals should be examined by a veterinarian experienced with that species at capture locations and only healthy animals should be shipped.
- Specific examinations should be conducted for signs associated with infestation of *Psoroptes* mites.
- Due to livestock risk, difficulty of diagnosis, and lack of knowledge of the disease in bighorn sheep, bighorn sheep taken from an area where brucellosis exists in other wildlife species should be tested for *Brucella* spp.

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