

PNEUMONIA IN BIGHORN SHEEP: EFFECTS ON POPULATIONS

David A. Jessup, DVM
Wildlife Investigations
Laboratory
Sacramento, CA

ABSTRACT.

Pneumonia has frequently been identified as an important mortality factor in bighorn sheep (*Ovis canadensis*) populations in the western United States. Resulting poor lamb survival and low recruitment rates as well as fatalities in adults may strongly influence population trends. Bacteria, in particular *Pasteurella* species, are the most frequently isolated pathogens. Predisposing factors allowing bacterial penetration of the mucous membranes of the respiratory tract may be necessary for initial infection and are discussed. The outcome is almost invariably fatal for the individual and serial passage may result in enhanced virulence and devastating epizootics. Previously healthy populations of bighorn in four locations were devastated or destroyed by bacterial pneumonias in 1980.

INTRODUCTION

The bighorn sheep of North America numbered between 1,500,000 to 2,000,000 at the beginning of the 19th century (Buechner, 1960). Today, less than 40,000 survive in all of North America (Trefethen, 1975). Although market hunting at the end of the 19th century, degradation of and competition for habitat by domestic species, and human intrusion into the rugged terrain favored by bighorn have been cited as detrimental factors, diseases, in particular, respiratory diseases, have been perhaps the single most important factor the last few decades (Buechner, 1960; Hibler, et al., 1972; McQuivey, 1978; Spraker, 1977; and Spraker and Hibler, 1977). Since it is difficult to reproduce purely bacterial pneumonias in experimental animals, much effort has been spent toward identifying initiating or "stressing" factors which allow bacteria to penetrate the mucous membranes of the upper respiratory tract breaching the first line of defense. Although many predisposing factors have been identified, none other than "stress" as a general phenomenon have proven consistent common denominators. Bacterial bronchopneumonias and resulting bacteremias, however, are usually the ultimate cause of death whether lungworms, viral pneumonia, nutritional or cold stress, chemical or physical irritation or immune suppression are the initiating factors (Marsh, 1938; Mills, 1937 and Potts, 1937).

Numerous species of bacteria have been cultured from pneumonias in bighorn, but *Pasteurella* sp. stand out as the most common and most pathogenic (Howe, et al., 1966; Potts, 1938; Rush, 1927; Spraker, 1977; Spraker and Hibler, 1977). Paradoxically, *Pasteurella* sp. have frequently been isolated from the upper respiratory tract of apparently normal bighorn sheep and domestic animals (Spraker, 1977). This organism is known to be capable of changing morphology, toxicity and invasiveness rapidly (Titche, 1979). Once initiated in bighorn, bacterial pneumonias seem to increase virulence and infectivity (Spraker, 1977). Whether bighorn sheep are particularly susceptible to bacterial pneumonias, whether domestic sheep can harbor more pathogenic strains of bacteria, particularly *Pasteurella* sp., or whether domestic sheep harbor other predisposing viral, mycoplasmal or bacterial agents, is unknown (Jang, et al., 1978; Parks, et al., 1972; Pearson and England, 1979).

What is known is that precipitous bighorn population declines in the form of pneumonia die-offs affecting all age classes of bighorn have been documented subsequent to exposure of apparently healthy bighorn sheep to apparently healthy domestic sheep. In three of the four outbreaks cited fibrinopurulent, bacterial bronchopneumonia and pleuritis killed all or many of the exposed bighorn, with no apparent effect on the exposed domestic sheep.

Previous reports on bacterial pneumonia in bighorn have attributed increased susceptibility to bacterial overgrowth of the respiratory tract to adrenal hyperplasia, increased steroid output, and immune suppression as a result of various "stress" factors (Spraker, 1977). Unusual stressing factors and adrenal changes were not identified in these cases (Figure 1).

In each of the four outbreaks, however, history or evidence of exposure to respiratory tract irritants was identified.

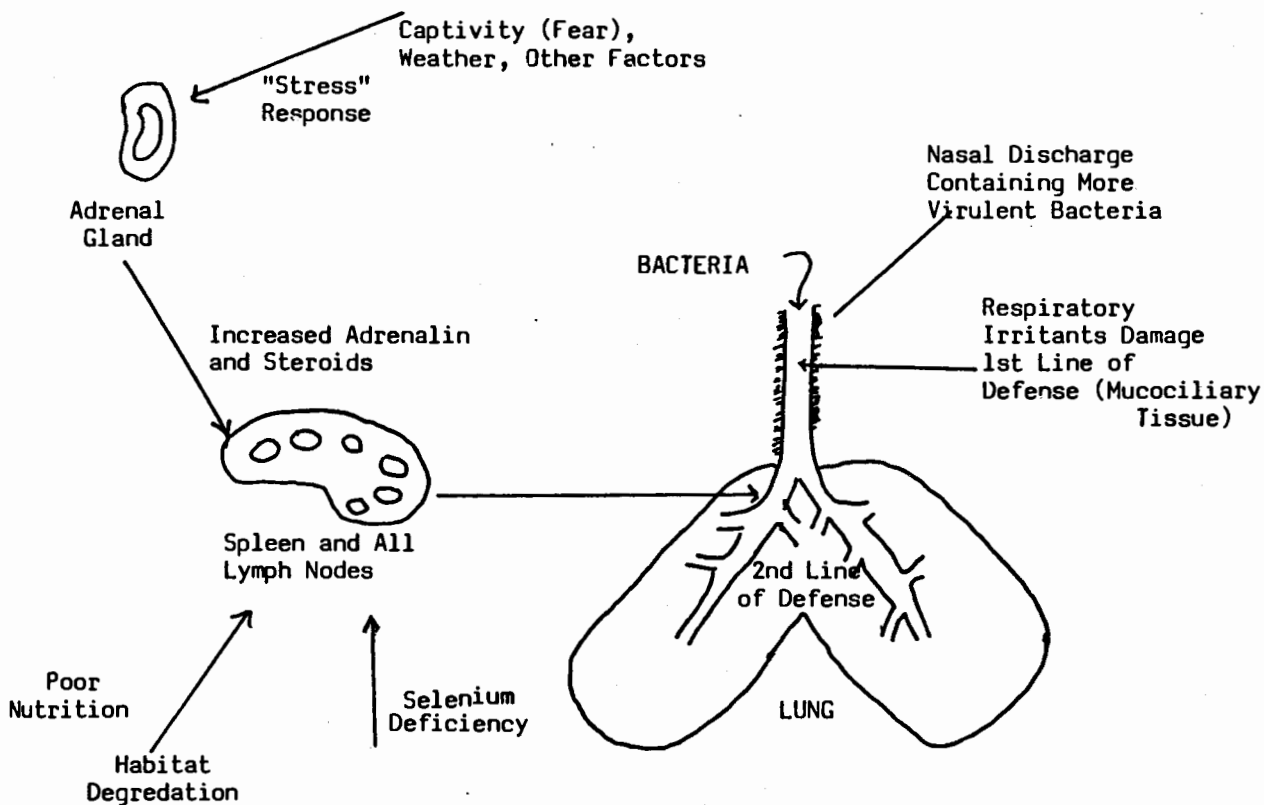


Figure 1. Factors affecting resistance to bacteria.

CASE REPORTS

In 1980 four outbreaks of pneumonia occurred in the western United States resulting in major losses of bighorn sheep (Table 1).

The subspecies involved in case 1 were Rocky Mountain bighorns (*Ovis canadensis canadensis*) and were housed in captivity at a game range outside Winthrop, Washington. These animals were captured in Montana and had been on a diet of native grasses and alfalfa, mineral supplement and grain supplement for eleven months. An experimental anthelmintic, Albendazole, was administered to half the bighorn to eliminate *Protostrongylus* sp. lungworms. With the research completed, apparently healthy domestic sheep were introduced to the game farm for hybridization experiments in mid-November 1979. By mid-December, 8 bighorn were dead of bronchopneumonia, by early January all bighorn but 1 were dead. Domestic sheep remained clinically normal. Lesions were those of fibrinopurulent pleuritis and bronchopneumonia, but postmortem degeneration obviated meaningful bacteriology. All bighorn appeared to be in excellent body condition.

California bighorn sheep (*Ovis canadensis californiana*), obtained from British Columbia in 1971, contained within an 1,100 acre enclosure at the Lava Beds National Monument, Siskiyou County, California constitute case 2. Diet was unsupplemented grazing of native and introduced grasses and forbs; water was limited to three artificial sources. Since introduction,

Table 1. Bighorn sheep losses due to pneumonia.

Location	Date	# Bighorn Mortalities	Bacteria Isolated	Stressing Factors	Domestic Sheep
Game Farm Twisp, WA	12/79- 1/81	11/12	Post-Mortem Degeneration Too advanced	1. Captivity 2. Lungworm (low no. in six individuals)	Yes
Lava Beds National Monument Tule Lake, CA	7/80- 8/80	31/31	<u>Pasteurella</u> sp. <u>P. multocida</u> <u>C. pyogenes</u>	1. Selenium deficiency 2. Irritant smoke 3. Predisposing virus	Yes
Mormon Mts. Carp, NV	9/80- 10/80	31/450 (initially)	<u>P. multocida</u> <u>C. pyogenes</u>	1. Silicosis	Yes
Waterton Canyon, CO	9/80- 11/80	80/88	<u>C. pyogenes</u> <u>Nisseria</u> sp.	1. Human encroachment 2. Silicosis	No

the herd had grown slowly and steadily except for the death of approximately five adults from what appeared to be bluetongue in 1974 and the loss of approximately six lambs in 1975 from what appeared to be soremouth (contagious ecthyma). Neither of these situations is well documented, but both led to discussions concerning intermittent use of adjacent National Forest Service land by domestic sheep. Fecal sampling in 1978 and 1979 revealed no lungworm ova. In February 1980, with the population at 43, an attempt was made to capture bighorn for transplant. Six animals died at that time from capture myopathy. Except for cardiac and skeletal muscle lesions, all individuals were in excellent condition. A paucity of intestinal nematodes and no lungworms were noted. At that time, glutathione peroxidase tests indicated 1 animal was selenium deficient when compared to domestic sheep normal valves and 2 were marginal of 5 tested. On 4/1/80 a pesticide and chemical fire in Tule Lake, 10 miles north, resulted in a cloud of irritating smoke in the area. In mid-May a ewe died from an abscessed puncture wound and cellulitis of the hind leg, from which Corynebacterium pyogenes was cultured. In mid-June heavy domestic sheep use of the north and west boundaries of the pen and close contacts with bighorn were noted. Thirty-one apparently healthy bighorn were counted on June 28 by air and ground crews.

The first mortality was noted July 7, but not reported. In retrospect, approximately 8 animals died during the first 10 days of July prior to extensive on-site investigations. Three dead bighorn found between 7/7/80 and 7/12/80 were shipped to the Wildlife Investigations Lab (WIL), Sacramento; all other animals were field necropsied. In all cases lesions were those of fibrinopurulent pleuritis and bronchopneumonia. Bacteriologic cultures of lung, pleural fluid, bronchial lymph node, and serum grew Corynebacterium pyogenes, Pasteurella multocida, or Pasteurella sp. (Buchanan and Gibbons, 1975; Jang et al., 1978). Serologic testing revealed no evidence of previous viral diseases. A feral domestic sheep from the adjacent pen and 2 deer from within the pen were collected for analysis. The domestic sheep spleen yielded a Type 11 bluetongue virus. Both deer had serologic titers to parainfluenza Type 3 (PI-3) virus which has been experimentally proven to predispose sheep to severe bacterial pneumonias.

Free-ranging desert bighorn sheep (Ovis canadensis nelsoni) of the Mormon Mountains of east Central Nevada were subjects of case 3. In early September 1980, hunters reported 8 dead bighorn rams in very close proximity to a water source. Others in the area, mostly ewes, were noted to be coughing. Although domestic sheep are normally grazed only at the base of the Mormon Mountains, 2 domestic sheep were observed running with the band of bighorn (McQuivey, pers. com.). Lungs from 4 individuals, 3 hunter-kills and a fresh dead bighorn were collected (R. Taylor, pers. com.). Lesions were those of bronchopneumonia, microscopically the disease process appeared to be less acute in nature. Amorphous mineral debris within macrophages in bronchiolar lumens was noted. At least 1 individual appeared to be emaciated. Bacteriologic cultures grew Corynebacterium pyogenes, Pasteurella multocida, Actinobacillus sp. and a Bacillus sp. (R. Taylor, pers. com.).

The fourth case of bronchopneumonia occurring in 1980 involved a Rocky Mt. bighorn at Waterton, Colorado. The herd had been rather stable at a population of about 100 for several years (E. Williams, pers. com.). Construction of a dam at the head of the drainage of Waterton Canyon and a service road to the dam site in the spring of 1980 disturbed normal bighorn foraging and watering habits. Bighorn had to cross the rather busy dirt road to reach water and large amounts of dust were noted on foliage as high as one-half mile above the road (E. Williams, pers. com.). In October the first deaths were noted. During November individuals obviously ill were collected (E. Thorne, pers. com.). By December only 8 of the 88 bighorn counted in September remained alive (E. Thorne, pers. com.). Lesions were similar to those in the three previous cases, bacterial bronchopneumonia and pleuritis. Microscopic lesions were most similar to case three, the presence of amorphous mineral debris in macrophages being notable (E. Williams, pers. com.). Pasteurella sp. were strongly suspected as being the primary bacterial pathogen, but only Corynebacterium pyogenes, a Nisseria sp. were cultured.

DISCUSSION

The clinical, pathologic and bacteriologic similarities between these 4 cases are striking, particularly considering the differences in location, weather, temperature, terrain, husbandry practices and race or subspecies. Adults from prime age classes as well as young were affected and the outcome was invariably fatal. Bacterial bronchopneumonia and pleuritis in otherwise healthy individuals was consistent if not a specific lesion. Pasteurella sp. and Corynebacterium pyogenes were isolated from multiple individuals in two cases and suspected in case one, but obviated by post-mortem changes. C. pyogenes is a very common opportunistic bacteria of livestock. Although common inhabitants of the upper respiratory tract of bighorn sheep and many other mammals (Spraker, 1977), Pasteurellas can vary tremendously in pathogenicity and serial passage of this bacteria can greatly enhance virulence (Titche, 1979). A symbiotic relationship between C. pyogenes and other fastidious gram negative bacteria has been noted, but the exact relationship between P. multocida and C. pyogenes in these cases is unclear. Other species of bacteria isolated are either not known to be primary pathogens or thought to be contaminants.

As previously noted, bacterial pneumonias are often thought to be predisposed by various stressing factors. The bighorn in case 1 were maintained in captivity, and as some authors have noted, captivity and fear tend to increase levels of adrenal hormones and steroids. These in turn suppress the immune response and lead to increased susceptibility to disease. This phenomenon usually occurs within 60 days of captivity, since adaptation to captivity leads to decreased fear, decreased adrenal stimulation and recovery of the immune response. It should be noted that the animals in case 1 had been in captivity for over 11 months and were apparently adapted to captivity. One-half the bighorn had low levels of Protostrongylus sp. lungworm having served as the negative control group in anthelmintic experiments. The pneumonia did not distinguish between lungworm positive or negative individuals. Onset of disease was between 21 and 30 days following introduction of apparently healthy domestic sheep into an adjacent paddock. Nutritional deficiencies were not apparent at necropsy. Weather was not excessively cold during the winter of 1979-80, milder than the bighorns original home in Montana.

In case 2, several possible predisposing factors were examined. Moderate selenium deficiency had been previously identified in these bighorn. Adequate levels of selenium are

necessary for proper functioning of the immune system. A respiratory irritant was known to have transited the area 90 days prior to the first pneumonia fatalities. However, no microscopic evidence of chronic respiratory irritation was noted in the bighorn examined or 2 collected deer known to have been exposed. No evidence of toxicants was found in vegetation or trapped rodents. Nutritional and weather stresses appear to have not been factors. Although these bighorn were technically in captivity, their pen was quite large and they were well-adapted to it. A physically stressful attempt at capturing these bighorn had occurred 180 days prior to the onset of disease. Although this stress may have resulted in initially compromised immune responses, recovery of normal immune responses should have been complete within about 60 days as previously noted. Adrenal lesions and/or lymphoid depression suggestive of stress were not noted in tissues examined microscopically. Lungworms were not present and husbandry practices had not changed significantly in 9 years. Although domestic sheep had been present in the area periodically for 9 years, the usual practice was to maintain some separation from the bighorn pen. Approximately 30 days prior to the onset of disease, a flock of over 100 domestic sheep were noted grazing along the northern and western boundaries of the bighorn enclosure. It should be noted that a Type 11 bluetongue virus was isolated from the spleen of the feral domestic sheep collected adjacent to the bighorn pen. Both deer collected from the bighorn pen had low titers to parainfluenza III virus. The most severe and fatal bacterial pneumonias of domestic sheep often involve this virus. Domestic sheep carcasses were noted near the pens. Although the three water sources were treated with tetracycline (150 mg/gal) for three weeks, no effect was apparent.

Case 3 involved free-ranging bighorn in an area where lungworm had previously been identified (R. McQuivey, pers. com.). Lungworms were not present in lungs of affected individuals. Pasteurella pneumonia in conjunction with lungworm infection have previously been reported as a population regulating disease in desert bighorn in Nevada, with lambs most commonly affected (McQuivey, 1978).

Although one hunter-killed bighorn with pneumonia was emaciated, other affected animals were not. Further observation of the range in the fall did not reveal evidence of excessive grazing pressure. Although initial counts revealed only modest population reductions, surveys in the spring of 1981 revealed only approximately one-half the expected numbers of bighorn. The presence of mineral debris in macrophages within bronchial lumens is an interesting finding. The exact nature of this debris is unknown but it appears to be fine silicaceous material. Sand and blowing dust are not unusual in desert bighorn habitat, but this material may have damaged or overwhelmed the mucociliary lining of the respiratory tract predisposing the lungs to bacterial infection. The domestic sheep observed among the bighorn were not collected. How long they had been feral is unknown, but their presence with this bighorn herd was not previously reported. Their role in the bighorn pneumonia is unknown, but they were not clinically affected.

In case 4 factors predisposing bighorn to pneumonia include the stress of human encroachment, isolation of water sources and environmental degradation. The presence of silicaceous material in bronchi was similar to that seen in case 3. Domestic sheep were not present in the Waterton Canyon area. Attempts to treat the bighorn with antibiotic treated bait was apparently unsuccessful.

The morbidity and mortality figures in the 4 cases cited speak for themselves. Bacterial pneumonias of bighorn sheep are not only a population limiting factor, they are a population eliminating factor. Traditional wildlife management stresses the importance of habitat in the maintenance of healthy wild ruminant populations. Too often habitat is considered to be primarily or solely food, water and cover. Obviously there is much more to the concept of habitat, especially if the species under consideration is bighorn sheep.

Bighorn sheep appear to be a classic example of a climax species and a native species exquisitely sensitive to habitat degradation and diseases of domestic livestock. Beside bacterial pneumonias, bighorn are susceptible to bluetongue, contagious ecthyma, chronic frontal sinusitis, and scabies, all of which are fairly common livestock diseases that have been identified as mortality factors in bighorn in the last decade. The analogy of native Americans and native bighorn sheep being similarly sensitive to the diseases of their European counterparts is almost irresistible.

It is quite possible that domestic sheep are more resistant to colonization of the respiratory tract by Pasteurellas, and thus, they may harbor more pathogenic strains without manifesting disease. This would explain why in 3 cases cited, domestics among or adjacent to bighorn, dying of pneumonia, were apparently unaffected.

Alternatively, domestic sheep may harbor other bacteria, mycoplasma or viruses such as the bluetongue virus isolated in case 2, that predispose to pneumonic bacterial overgrowth. The source of the PI-3 virus exposure seen in deer, a resistant species, is unknown. Domestic stock is a possible source again, however, the difficulty of proving this in retrospect using tissues from bacteremic bighorn should be obvious.

The purpose of this report is not to condemn the use of public lands for grazing domestic sheep. Indeed, we were not able to prove transmission of disease from domestics to bighorn; although it should be noted that little or no attempt could be, or was made to do so. However, it seems more than a coincidental observation in 3 of the cases cited. The purpose of this report is not to condemn all forms of human encroachment into bighorn sheep habitat. However, some forms of human activity may well have adverse impact on bighorn sheep populations and this certainly should be considered when Environmental Impact Reports are written.

The purpose of this report is to point out a consistent association of habitat degradation and/or domestic sheep with bighorn sheep dying of fibropurulent bacterial pneumonias in captive, semi-captive and free-ranging situations. This association resulted in significant to total death loss of bighorn with no apparent affect on domestics. These observations are consistent with observations spanning 15 decades, that association of domestic sheep/habitat degradation with bighorn, results in the loss of the latter (Spraker, 1977). Whether the cause is primarily or secondarily bacterial pneumonia, viral, chemical or stress-related, the observation remains consistent. Land management agencies and wildlife professionals have the responsibility of administering public lands in a manner consistent with preservation of native species, particularly rare or sensitive species such as the various races of bighorn. At the management level, decisions must frequently be made concerning conflicting uses of public resources. These decisions should reflect a knowledge of consistent observations and of biological and medical knowledge critical to the preservation of native species.

ACKNOWLEDGEMENTS

Dr. Bill Foreyt and Dr. Robert Taylor contributed data and assisted in analyzing cases 1 and 3 respectively. Dr. Terry Spraker and Dr. Beth Williams did the same on case 4. Mr. Dick Weaver provided professional wildlife expertise and Ms. Karen Jones assisted with bacteriology and laboratory support. This paper was supported by Pittman-Robertson Project W52-R21, through the California Department of Fish and Game wildlife investigations laboratory at Sacramento, California.

LITERATURE CITED

- Buchanan, R. and N. Gibbons. 1975. Bergey's manual of determination bacteriology. Williams and Wilkins, Baltimore, MD.
- Buechner, H. 1960. The bighorn sheep in the United States, its past, present and future. Wildl. Monogr. No. 4, 174 p.
- Hibler, C.P., R. Lange, and C. Metzger. 1972. Transplacental transmission of Protostrongylus sp. in bighorn sheep. J. Wildl. Dis. 9:384.
- Howe, D., G. Woods, and G. Marquis. 1966. Infection of bighorn sheep (Ovis canadensis) with Parinfluenza-3 myxovirus and other respiratory viruses: results of serologic tests and culture of nasal swabs and lung tissue. Bull. Wildl. Dis. Assoc. 2(2):34-47.
- Jang, S., E. Biberstein, and D. Hirsh. 1978. A diagnostic manual of veterinary clinical bacteriology and mycology.

- McQuivey, R. 1978. The desert bighorn sheep of Nevada. Nev. Dept. Wildl., Reno, NV Biol. Bull. No. 6. 81 p.
- Marsh, H. 1938. Pneumonia in Rocky Mountain bighorn sheep. J. Mammal. 19(1):214-219.
- Mills, H. 1937. A preliminary study of the bighorn of Yellowstone. J. Mammal. 18(2):205-212.
- Parks, J., G. Post, T. Thorne, and P. Nash. 1972. Parainfluenza-3 virus infection in Rocky Mountain bighorn sheep. J. Amer. Vet. Med. Assoc. 161(6):669-672.
- Pearson, N.J., J.J. England. 1979. Isolation of chlamydial agent from Rocky Mountain bighorn sheep. J. of Wildl. Dis. 15(4):499-503.
- Potts, M. 1937. Hemorrhagic septicemia in the bighorn of the Rocky Mountain National Park. Trans. N. Am. Wildl. Conf. 3:893-897.
- _____. 1938. Observations on diseases of bighorn in the Rocky Mountain National Park. Trans. N. Am. Wildl. Conf. 3:893-897.
- Rush, W. 1927. Notes on diseases in wild game. J. Mammal. 8:163-164.
- Spraker, T.R. 1977. Fibrinous pneumonia of bighorn sheep. Desert Bighorn Council, Trans. 21:17-18.
- _____, and C.P. Hibler. 1977. Summer lamb mortality of Rocky Mountain bighorn sheep. Desert Bighorn Council Trans. 21:11-12.
- Taylor, R.E.L. 1973. Disease losses in Nevada bighorn. Desert Bighorn Council Trans. 17:47-51.
- Titche, A. 1979. Avian cholera in California, California Resources Agency pub.
- Trefethen, J.B. 1975. The wild sheep in modern North America. The Winchester Press, New York. 302 p.