

## **FATAL PNEUMONIA OF BIGHORN SHEEP FOLLOWING ASSOCIATION WITH DOMESTIC SHEEP**

Authors: Foreyt, William J., and JESSUP, D.A.

Source: Journal of Wildlife Diseases, 18(2) : 163-168

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-18.2.163>

---

The BioOne Digital Library (<https://bioone.org/>) provides worldwide distribution for more than 580 journals and eBooks from BioOne's community of over 150 nonprofit societies, research institutions, and university presses in the biological, ecological, and environmental sciences. The BioOne Digital Library encompasses the flagship aggregation BioOne Complete (<https://bioone.org/subscribe>), the BioOne Complete Archive (<https://bioone.org/archive>), and the BioOne eBooks program offerings ESA eBook Collection (<https://bioone.org/esa-ebooks>) and CSIRO Publishing BioSelect Collection (<https://bioone.org/csiro-ebooks>).

Your use of this PDF, the BioOne Digital Library, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at [www.bioone.org/terms-of-use](http://www.bioone.org/terms-of-use).

Usage of BioOne Digital Library content is strictly limited to personal, educational, and non-commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

---

BioOne is an innovative nonprofit that sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

## FATAL PNEUMONIA OF BIGHORN SHEEP FOLLOWING ASSOCIATION WITH DOMESTIC SHEEP

WILLIAM J. FOREYT, Department of Veterinary Microbiology and Pathology, Washington State University, Pullman, Washington 99164, USA.

D.A. JESSUP, California Department of Fish and Game, Wildlife Investigation Laboratory, 1701 Nimbus Road, Rancho Cordova, California 95819, USA.

**Abstract:** During 1979-1980 acute fibrinopurulent bronchopneumonia resulted in high mortality or total loss of herds of bighorn sheep (*Ovis canadensis*) in California and Washington. Contact with domestic sheep occurred shortly before the onset of disease in each case. Circumstantial evidence indicated that the apparently healthy domestic sheep transmitted pathogenic bacteria to the bighorns, resulting in mortality. *Pasteurella multocida* and *Corynebacterium pyogenes* were isolated from pulmonary tissue of dead bighorns. The presence of domestic sheep may have been an important stress which initiated or compounded the disease.

### INTRODUCTION

North American bighorn sheep numbered between 1.5 and 2.0 million at the beginning of the 19th century (Buechner, 1960). Today, their number is estimated at less than 40,000. The decline has been attributed to several factors: market hunting at the end of the 19th century, loss of suitable habitat through human development and competition with domestic livestock, and diseases (Marsh, 1938; Cowan, 1940; Buechner, 1960; Forrester, 1971; Hibler et al., 1972; Spraker and Hibler, 1977; Lange et al., 1980). Disease-related mortality is perhaps the most important factor producing dramatic population declines during the last few decades. Major diseases included the lungworm-pneumonia complex (Forrester, 1971; Hibler et al., 1972), psoroptic mange (Lange et al., 1980), and hemorrhagic septicemia (pasteurellosis) (Potts, 1937; Spraker, 1977).

Bacterial pneumonia may follow predisposing factors such as lungworms, viruses, nutritional, cold, or behavioral stress, or immune suppression (Rush, 1927; Mills, 1937; Potts, 1937; Marsh, 1938; Howe et al., 1966; Rosner, 1971; Hibler et al., 1972; Parks et al., 1972;

Taylor, 1973; Spraker, 1977; Spraker and Hibler, 1977; Pearson and England, 1979). Several genera of bacteria have been isolated from penumonic bighorn sheep, but *Pasteurella* spp. are the most common and pathogenic (Hibler et al., 1972; Taylor, 1973; Spraker and Hibler, 1977). *Pasteurella* spp. have been isolated from clinically normal bighorn sheep and domestic animals (Rosner, 1971; Spraker and Hibler, 1977).

The present report documents two separate outbreaks of fatal pneumonia in bighorn sheep which resulted shortly after their exposure to domestic sheep. Domestic sheep were unaffected.

### CASE HISTORIES

#### Washington

In January, 1979, eight wild Rocky Mountain bighorn sheep (*Ovis canadensis canadensis*) (two yearling rams and six adult ewes) were transported from Wildhorse Island in Flathead Lake, Montana, to the Methow Game Range, Okanogan County, in central Washington. Sheep were confined in a 2.5 ha enclosure in a remote area. A natural stream traversed the pen and native trees, shrubs, and grasses were

present for feed and shelter. In winter, feed was supplemented with alfalfa hay and a pelleted alfalfa and grain mixture. Mineralized salt was available at all times. In March, 1979, the six ewes each had a single lamb. Excellent growth was noted in the lambs and adult sheep throughout the summer.

On 2 November 1979, 10 months after the bighorns were placed in the enclosure, 11 adult crossbred domestic ewes, 3-4 years of age, were introduced into the pen with the bighorns. The domestic sheep had been examined by veterinarians and determined to be healthy. All animals were fed at a common source, but the bighorns and domestic sheep were not seen to associate with each other. No behavioral problems were observed.

Twenty-six days later (28 November) seven bighorns were found dead. These included one ram, three ewes, and three lambs. The carcasses were in excellent nutritional condition. Death had occurred within 5 days of the last visit, but extensive autolysis was consistent with death several days previously. Due to the deteriorated condition of the carcasses, only histologic examination of selected organs was accomplished.

The domestic sheep were removed from the pen on 8 December 1979, and at that time, the seven bighorns appeared to be healthy. Six of the remaining seven bighorns were found dead on 29 January 1980. They also had been dead for several days, and postmortem examination was similar to that conducted on the first group. In both groups of dead bighorns, animals were found together in one part of the pen in their beds, and no external lesions were observed. Based on direct observation, each die-off had occurred within a few days before 28 November and 29 January.

#### California

California bighorn sheep (*Ovis canadensis californiana*), acquired from

British Columbia in 1971, were confined to a 445 ha enclosure at the Lava Beds National Monument, Siskiyou County, California. Since introduction, the herd had grown slowly and steadily except for the deaths of approximately five adults in 1973 from presumptive bluetongue, and the loss of approximately six lambs in 1976 from other causes. Domestic sheep intermittently grazed adjacent National Forest Service land.

In February, 1980, an attempt was made to capture the 43 bighorns in the population for transplant. Of these, 16 were captured and six died at that time from capture myopathy. Few gastrointestinal nematodes and no lungworms were grossly detected at necropsy. No lungworm larvae were detected in feces (Baermann technique). Glutathione peroxidase tests indicated a low level (<15 ppm) of selenium in one of five animals, when compared to established values for domestic sheep. The remaining 37 individuals appeared to be in excellent physical condition and were left in the enclosure. In mid-June, domestic sheep were intensively grazed along the north and west boundaries of the enclosure, and nose-to-nose fenceline contact with bighorn sheep was noted. Changes in weather, range condition, movement of people etc. were not observed. Approximately eight bighorns died during the first 10 days of July. A presumptive diagnosis of pasteurellosis was made on 12 July, based on gross lesions, preliminary histopathologic interpretations and bacteriologic results.

On 12 July 1980, the water sources were emptied, cleaned and flushed. Oxytetracycline at 0.13 gm/L water was added to water on 16 July at which time five additional bighorn carcasses were found. Water sources were flushed and medicated every other day for 3 weeks, but water consumption was not monitored. All 43 bighorn sheep were presumed dead by August 15, 1980, since no live animals could be located.

## MATERIALS AND METHODS

Gross and histologic examination of 10 of the 13 dead bighorns from Washington was done at the Washington Animal Disease Diagnostic Laboratory, Pullman, Washington. Bacterial and viral cultures were not done because tissues were autolyzed. Fecal samples were examined for lungworm larvae with a Baermann apparatus.

In California, three dead bighorns found between 7 and 12 July 1980, were shipped to the Wildlife Investigations Lab (WIL), Sacramento, for necropsy and five other animals were examined in the field. Sections of hilar lymph node, liver, lung, kidney, spleen and trachea were collected in 10% buffered formalin, sectioned at 5  $\mu$ m and stained with hematoxylin and eosin (H&E). Fresh tissues and blood were kept on ice for as long as 24 h. Bacteriologic examinations were performed at the WIL or School of Veterinary Medicine, Davis, California. Pulmonary tissue from three bighorns, pleural fluid from one and bronchial lymph node from one were processed and inoculated onto blood agar and observed daily for bacterial colonies. Bacteria recovered were identified (Buchanan and Gibbons, 1975; Jang et al., 1978). Gross and histologic examinations for parasites were done, and feces were examined for lungworm larvae (Baermann technique).

Serum was collected from two of the fresh bighorn carcasses on 10 and 17 July 1980, for serologic evaluation. Serology was performed by the California Department of Agriculture Laboratory.

## RESULTS

### Washington

Weights of the five dead adult bighorns from the first group ranged from 41 to 80 kg (mean of 53). Lambs and bighorns from the second episode were not weighed. Parasites were not detected grossly. Body fat was considered ade-

quate in all animals, and each was characterized by advanced autolytic changes which limited histopathologic interpretation. Multiple sections of lung parenchyma were examined from different areas of the lung from each of ten bighorns. Acute pneumonia was apparent in all animals from both episodes. Pleural margins were thickened by accumulations of fibrin, macrophages and neutrophils. There was generalized pulmonary congestion with marked alveolar accumulations of fibrin and edema. Focal areas of necrosis were present throughout the pulmonary parenchyma. Sections of spleen, liver, kidney and trachea were congested and autolyzed. Lungs from three of ten animals contained sections of adult nematodes. *Protostrongylus* sp. larvae were present in feces in low numbers (<25 larvae/gram of feces) from four of ten bighorns and not detected in the other six.

One bighorn ewe survived the pneumonia episodes, and produced a lamb in 1980. All domestic sheep survived for four months after removal from the enclosure; no signs of disease were apparent.

### California

All bighorns were in good to excellent physical condition with ample reserves of body fat. Lesions found in the eight bighorns examined were similar. Lungs were bilaterally reddened, wet and consolidated in an anteriorventral distribution. Fibrinous pleural adhesions and pulmonary abscesses with cavitations were noted. Purulent exudate could be expressed from sectioned bronchioles. Six sheep had extensive amounts of yellow friable material (fibrin) covering the pleura and had fibrinous adhesions between individual lobes of the lung and to the costal pleura. Bloody intratracheal froth was a consistent finding. No lungworms or nodules were observed grossly. A small amount of hemorrhage was noted in the left adrenal gland of one

animal. No other gross abnormalities were recorded.

Microscopic examination of five lungs revealed fairly consistent lesions despite varying degrees of post-mortem autolysis. Sections from dorsal areas of lung contained proteinaceous fluid and occasional mononuclear and polymorphonuclear (PMN) cells filling bronchiolar lumina. Sections of ventral lung contained scattered islands of bronchial necrosis and hemorrhage. An intense, predominately mononuclear inflammatory response surrounded these islands. Bronchi contained large amounts of cellular debris, fibrin, and PMNs. Bacteria were most evident in areas adjacent to the necrotic foci.

Bronchial and abdominal lymph nodes and spleen had active germinal centers. Subcapsular inflammatory cells and proteinaceous fluid were noted in bronchial lymph node sections. Adrenals were not examined microscopically. All other major organs examined were considered histologically normal.

*Pasteurella multocida* was isolated from lung, bronchial lymph node and serum from one bighorn. *Pasteurella* sp. isolated from lung and pleural fluid in a second bighorn could not be speciated due to its repeated negative indole reaction. *Corynebacterium pyogenes* was also isolated from lung of the three bighorns sampled. Serum antibodies were not detected against *Brucella*, *Anaplasma*, infectious bovine rhinotracheitis virus, parainfluenza-3 virus, contagious ecthyma virus, or bluetongue virus. One bighorn had a titer of 1:32 by indirect complement fixation against *Leptospira hardjo*.

## DISCUSSION

Bacterial bronchopneumonia and pleuritis in otherwise healthy individuals were the consistent findings in these two outbreaks. Adults from prime age classes as well as young were affected and the outcome generally was

fatal. *Pasteurella* sp. and *Corynebacterium pyogenes* were isolated from several individuals in one outbreak and suspected in the second one. Possibly a bacterial pathogen, was introduced into the bighorns by domestic sheep, and was responsible for the pneumonia which resulted in mortalities in the bighorns. The presence of domestic sheep no doubt also was a stress factor.

We base those conclusions on several factors. The bighorns in California and Washington were in good physical condition and had been in the enclosures for 10 months or more before any deaths occurred, suggesting that acclimatization to the new habitat was not a major problem. However, initial mortality in the bighorns in Washington occurred within 26 days after introduction of the domestic sheep, suggesting possible transmission of a pathogen from the domestics. The domestic sheep were determined to be healthy before introduction into the enclosure, and none became sick or died during or after their presence in the enclosure.

In both outbreaks, bighorns were well nourished when found dead, suggesting an acute disease process. Histopathologic examinations confirmed acute fibrinopurulent bronchopneumonia characteristic of pasteurellosis, and *Pasteurella* was isolated in the California episode. *Pasteurella* spp. have been isolated commonly from the upper respiratory tract of bighorn sheep, other mammals and birds. These bacteria can vary in pathogenicity, and serial passage can enhance virulence (Spraker, 1977). Stress however is a major predisposing factor to clinical pasteurellosis (Gillespie and Timoney, 1981). It is possible that a nonpathogenic strain of *Pasteurella* in domestic sheep was increased in pathogenicity when transmitted to the bighorns which may have been under additional stress due to the presence of the domestic sheep. Domestic sheep may also be more resis-

tant to colonization of the respiratory tract by *Pasteurella*, and thus, may harbor more pathogenic strains without being affected.

It is also possible that domestic sheep may harbor other bacteria, mycoplasma or viruses that predispose to pneumonic bacterial overgrowth. However, the exact relationship between *P. multocida* and *C. pyogenes* in these cases is unclear.

*Pasteurella* pneumonia in conjunction with lungworm infection has been reported previously as a population regulating disease most commonly affecting bighorn lambs (Forrester, 1971; Hibler et al., 1972; Spraker and Hibler, 1977). That relationship is apparently not the same situation described in this paper since morbidity and mortality occurred in all cohorts, and lungworms were detected only in a few individuals in Washington, and none were detected in California.

Severe fibrinous pneumonias affecting captive bighorn sheep of all ages have been associated with adrenal cortical hyperplasia and stress of captivity (Spraker, 1977). The outbreaks in bighorns from California and Washington occurred during captive and semi-captive conditions, however, animals appeared to be well adapted to captivity and isolated from noise and contacts with humans and most other animals. No extensive or consistent gross adrenal lesions were noted at necropsy in these bighorns.

In the California outbreak, husbandry practices had not changed significantly in nine years, but several possible predisposing stresses may have contributed to initial infection. Selenium deficiency, based on known values for domestic sheep, had been previously identified in one animal and a respiratory irritant in the form of a cloud from a chemical fire, was known to have transited the area 90 days prior to the first pneumonia-related fatalities. No microscopic evidence of chronic respiratory irritation or lungworms was noted in these bighorns.

In these two geographically separated outbreaks, inclement weather, temperature, terrain, husbandry practices, physical exertion, nutritional disorders, excessive lungworm infections or other major stress factors were not identified and probably were not present. However, the presence of domestic sheep in close proximity to these bighorns may have been an important stress which initiated or compounded the outbreaks. In both outbreaks there was an association of domestic sheep with captive, or semi-captive bighorn sheep dying of fibrinopurulent (bacterial) pneumonias. This association was related to a significant death loss of bighorns with no apparent effect on domestic sheep. We conclude that bighorn sheep and domestic sheep should not occupy the same ranges or be managed in close proximity to each other, because of the potential adverse effect on the bighorn sheep.

#### Acknowledgements

The authors wish to thank Rolf Johnson, Washington Department of Game, Karen Jones, California Department of Fish and Game, and the Montana Department of Game for assistance in the work. This project was supported in part by Pittman — Robertson Project W52-R21, Wildlife Investigations Laboratory.

#### LITERATURE CITED

- BUCHANAN, R. and N. GIBBONS. 1975. *Bergey's Manual of Determinative Bacteriology*. Williams and Wilkins, Baltimore, Maryland, pp. 370-372.

- BUECHNER, H. 1960. The bighorn sheep in the United States, it's past, present and future. Wildl. Monogr. No. 4. 174 pp.
- COWAN, I. McT. 1940. Distribution and variation in the native sheep of North America. Am. Midl. Nat. 24: 505-580.
- FORRESTER, D.J. 1971. Bighorn sheep lungworm-pneumonia complex. In: *Parasitic Diseases of Wild Mammals*. J.W. Davis and R.C. Anderson, eds. Iowa State Press, Ames, Iowa, pp. 158-173.
- GILLESPIE, J. H. and J.F. TIMONEY. 1981. *Hagen and Bruner's Infectious Diseases of Domestic Animals*. Cornell Univ. Press, Ithaca, New York, p. 107.
- HIBLER, C.P., R. LANGE and C. METZGER. 1972. Transplacental transmission of *Protostrongylus* spp. in bighorn sheep. J. Wildl. Dis. 9: 384.
- HOWE, D., G. WOODS and G. MARQUIS. 1966. Infection of bighorn sheep (*Ovis canadensis*) with *Parainfluenza-3 myxovirus* and other respiratory viruses: Results of serologic tests and culture of nasal swabs and lung tissue. Bull. Wildl. Dis. Assoc. 2: 34-37.
- JANG, S., E. BIBERSTEIN and D. HIRSCH. 1978. *A Diagnostic Manual of Veterinary Clinical Bacteriology and Mycology*. Univ. Calif., Davis, California. 171 pp.
- LANGE, R.E., A.V. SANDOVAL and W.P. MELENEY. 1980. Psoroptic scabies in bighorn sheep (*Ovis canadensis mexicana*) in New Mexico. J. Wildl. Dis. 16: 77-82.
- MARSH, H. 1938. Pneumonia in Rocky Mountain bighorn sheep. J. Mammal. 19: 214-219.
- MILLS, H. 1937. A preliminary study of the bighorn of Yellowstone. J. Mammal. 18: 205-212.
- PARKS, J.G. POST, T. THORNE and P. NASH. 1972. Parainfluenza-3 virus infection in Rocky Mountain bighorn sheep. J. Am. Vet. Med. Assoc. 161: 669-672.
- PEARSON, N.J. and J.J. ENGLAND. 1979. Isolation of a chlamydial agent from Rocky Mountain bighorn sheep. J. Wildl. Dis. 15: 499-503.
- POTTS, M. 1937. Hemorrhagic septicemia in the bighorn of the Rocky Mountain National Park. J. Mammal. 18: 105-106.
- . 1938. Observations on diseases of bighorn in the Rocky Mountain National Park. Trans. N. Am. Wildl. Conf. 3: 893-897.
- ROSNER, S.F. 1971. Bovine parainfluenza Type 3 virus infection and pasteurellosis. J. Am. Vet. Med. Assoc. 159: 1375-1382.
- RUSH, W. 1927. Notes on diseases in wild game. J. Mammal. 8: 163-164.
- SPRAKER, T.R. 1977. Fibrinous pneumonia of bighorn sheep. Transactions of the Desert Bighorn Council, pp. 17-18.
- and C.P. HIBLER. 1977. Summer lamb mortality of Rocky Mountain bighorn sheep. Trans. Desert Bighorn Council, pp. 11-12.
- TAYLOR, R.E.L. 1973. Disease losses in Nevada bighorn. Trans. Desert Bighorn Council, pp. 47-51.

Received for publication 1 March 1981