A MAJOR BIGHORN SHEEP DIE-OFF FROM PNEUMONIA IN SOUTHERN ALBERTA

D. K. Onderka, Animal Health Division, Department of Agriculture, 6909-116 Street, Edmonton, Alberta T6H 4P2.

W. D. Wishart, Fish and Wildlife Division, Department of Energy and Natural Resources, 6909-116 Street, Edmonton, Alberta T6H 4P2.

ABSTRACT

The chronological course of a bighorn sheep (Ovis canadensis canadensis) die-off due to pneumonia is charted from southern British Columbia into southern Alberta and northern Montana. Initial losses were primarily breeding age adults followed by poor lamb production from the survivors. The southern Alberta herd was reduced by approximately 65% during the period September 1982 to November 1983. Clinical signs included decreased exercise tolerance, harsh coughing often under severe strain, and sometimes mucoid nasal discharge.

Postmortem examination of sheep found dead or collected revealed acute, subacute or chronic bronchopneumonia, sometimes with extensive pleuritis. A unique Pasteurella hemolytica biotype T was isolated from the lesions. Attempts to isolate other agents on bovine or bighorn cell cultures were unsuccessful. Pasteurella spp. were not found in an extensive culture survey of clinically normal bighorns. There was no correlation between the severity of lungworm infection and the occurrence of pneumonia. Preliminary results of a transmission study using the bighorn Pasteurella sp. isolate in domestic sheep are discussed.

INTRODUCTION

Periodic die-offs of Rocky Mountain bighorn sheep have occurred throughout their range during the past several decades (Buechner 1960, Stelfox 1971). Some of the more recent declines have been attributed to a complex of factors ultimately causing fatal pneumonia (Forrester 1971, Spraker 1979, Wishart et al. 1980). The present report describes an epizootic of bacterial induced bronchopneumonia in bighorns in southern Alberta.

HISTORY AND CLINICAL SIGNS

In December 1981, a die-off from pneumonia was reported in bighorn sheep after they had mixed with a domestic sheep herd on Maguire Creek in southeastern British Columbia (Davidson 1982). By the fall of 1982, the die-off had spread northward up the East Kootenay river to Premier Ridge and eastward across the continental divide into southern Alberta and into Waterton National Park. The northern extent of the die-off in Alberta was the Crowsnest Pass. By early winter 1983, dead and dying bighorns were reported in Glacier National Park, Montana.

In Alberta, the die-off appeared to diminish significantly by the fall of 1983 and the last sick bighorn observed was collected on October 27. Fortunately, a forest barrier north of the Crowsnest Pass may have prevented an interchange of sheep and the continuation of the disease northward.

Affected bighorns were unthrifty in behavior or appearance or both. Various stages of clinical signs observed ranged from animals with a few coughing bouts to animals with labored breathing to the extent that they had difficulty holding their heads up or even standing. Diseased animals were often detected by their inability to keep up with the herd. Generally, the body condition of the affected animals was good, but a few had a scruffy hair coat and some of these were emaciated. On occasion, yellow exudate or clear mucus nasal discharge could be observed in the coughing sheep.

The year following the outbreak (1983) was characterized by a poor and staggered lamb crop. Some unhealthy individuals were observed coughing during the summer and fall.

FIELD SAMPLING PROCEDURES

Following the first reports of a die-off, we initiated intensive ground and aerial surveys to locate diseased sheep and to determine the magnitude of the losses. Bighorns observed suffering from respiratory distress were collected whenever possible. Several dead sheep were located during the culling process. Whole carcasses of collected animals were delivered to the Alberta Veterinary Services Laboratories in Edmonton and Lethbridge and when this was not possible a composite sample of head, trachea, lung, heart, liver, kidney, femur and feces from each specimen was submitted. Also submitted were various combinations of the above body components salvaged from some of the animals that were found dead.

In addition to the culling program, apparently healthy bighorns from within and outside the die-off area were trapped or darted and immobilized to obtain nasal and pharyngeal swabs and blood sera. After sampling, the animals were tagged and released for further observation.

Specimens were obtained in the fall of 1983 when hunters brought in lungs and heads (non-trophy) of bighorn sheep that were shot north of the affected area, that is, from the Crowsnest Pass to the Bow River, a distance of approximately 150 kilometres. In addition to the Alberta samples, we also received a few specimens and samples from the die-off area in British Columbia and Glacier National Park, Montana.

In total, 19 animals were collected and 23 were found dead. Ten of the latter had suitable tissues for laboratory examinations. Nasal and pharyngeal cultures were collected from 22 live bighorns in the die-off area and 176 cultures were obtained north of the die-off area. Cultures were obtained from nasal passages and lungs from 37 animals that were turned in by hunters. Carcasses of two bighorn sheep and one mountain goat were received from Glacier National Park.

MATERIALS AND METHODS

Body condition was assessed by evaluating subcutaneous, mesenteric and perirenal body fat deposits as well as quantification of midshaft femoral bone marrow fat using ether extraction of a preweighed dried sample. The lungworm load was estimated by the size and number of grey nodules in the parenchyma and a rating of very light through moderate, to very heavy was assigned. First stage larvae were quantified in the dried fecal pellets after Samuel and Gray (1982). Kidney, blood and liver were retained frozen for future trace mineral evaluation; sera and fecal samples were collected for future cortisol determinations. Microscopic examination was done on routine formalin fixed tissues stained with hematoxylin / eosin and Masson's trichrome stain.

Swabs for microbiologic examination were obtained using cotton swabs wetted with Stuarts transport media[1]. Pharyngeal swabs were taken through 13 millimetre diameter, 23 centimetre long pre-sterilize plastic tubes inserted into the oral cavity. Culture isolates were exchanged with the Veterinary Laboratory at Abbotsford to compare the organisms isolated from bighorns from British Columbia. Routine cultures were prepared using blood agar and MacConkey plates followed by a series of sugar inoculations to determine biochemical characteristics. Virus isolations were attempted using bovine kidney cells and lung and kidney cell cultures prepared from a young bighorn lamb.

RESULTS

We estimated that approximately 65 percent of a herd of about 400 between Waterton Park and the Crowsnest Pass died during the first year of the outbreak (Table 1). Park wardens estimated a loss of about 150 sheep or approximately 50 percent of the Waterton Park herd. The most remarkable losses were in the older age classes, particularly rams (Table 1, Figure 1). Only four rams were harvested from the die-off area in the fall of 1983 compared to an average annual harvest of 16 trophies during the last decade. Lamb survival and production were low in 1982 and 1983 with less than 20 lambs/100 ewes observed in March, 1984 (Table 1).

Although none of the tagged sheep showed any signs of pneumonia, three tagged ewes lost their lambs over the summer of 1983 while another tagged ewe had a sick lamb with pneumonia which was collected in September (Festa-Bianchet 1983). Most of the animals collected and many of the dead sheep were in good body condition with bone marrow fat of 79 to 91 percent. Some ewes in moderate conditions ranged from 42 to 49 percent while the thin sheep had a fat content

1. Culturettes^{R} - Can Lab.

of 8.5 to 27 percent. Lungworm lesions were usually mild to moderate with few very heavy infections. There was a poor correlation between the estimated lungworm burden and fecal larval output.

Table 1. Numbers of bighorns observed from winter aerial surveys north of Waterton Park to Crowsnest pass (1975-1984).

Year	Total	Lambs	Ewes[1]	Rams <1/2 curl	Rams >1/2 curl
1975	353	81	187	50	35
1979	389	77	248	44	29
1983	123	20	86	4	5
1984	159	19	108	21	3

1. Includes yearling ewes.

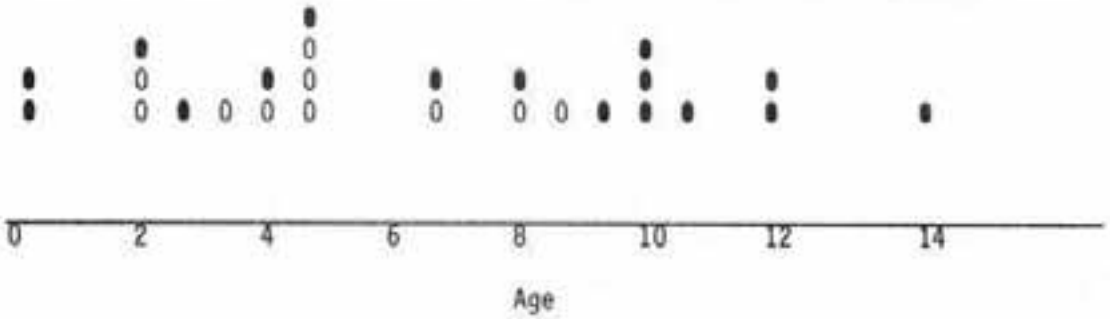

Figure 1. Age distribution of bighorn sheep with pneumonia (● = male, 0 = female).

The principal lesion was bronchopneumonia expressed as red consolidation of the anterior ventral lung lobes. The tissue was firm and the airway walls were thickened, often containing cream colored mucoid exudate. Pneumonic tissue was usually sharply demarcated from normal lung. There was no overlap with lungworm infected areas. Some animals had severe fibrinous pleuritis with adhesions to the chestwall. Subacute lesions had progressed to necrosis of the lung tissue and some had developed abscesses. In chronically affected sheep, fibrous tissue had formed fusing the anterior and middle lobes as well as causing firm adhesions to the thoracic pleura. These animals were usually in poor body condition.

Microsopic examination confirmed a range of acute to chronic active bronchopneumonias characterized in the subacute phase by proteinaceous alveolar exudate mixed with macrophages, pneumocytes and neutrophils. Bronchioles were often obliterated by a mainly neutrophilic exudate and mucosal infiltrations. Occasionally, a first stage lungworm larva was found trapped in the alveolar exudate. The lungworm nodules consisted of adults, eggs and L_1 larvae causing modest interstitial lymphocyte reaction and some fibromuscular hyperplasia of alveolar septae. Bronchial lymph nodes were enlarged with prominent germinal centers and much neutrophil influx into subcapsular and medullary sinusoids.

Pasteurella hemolytica biotype T of a non-hemolytic variety was isolated from the pneumonic lung tissue in 48 percent of the sheep with bronchopneumonia. In addition, Corynebacterium sp. was recovered from lungs that had progressed to form abscesses. Pasteurella sp. was also recovered from bronchial lymph nodes and nasal swabs in animals with bronchopneumonia.

Virus isolation attempts were consistently negative even from tissues of animals submitted live to the laboratory. Some tissue was suitable for Chlamydia and Mycoplasma cultures but no isolations were made.

Two hundred and forty-five nasal and pharyngeal swabs examined to evaluate the prevalence of Pasteurella spp. in the nasopharyngeal bacterial flora were negative except for one lamb closely associated with a group of sheep affected by pneumonia (Table 2).

Table 2. Apparently healthy bighorn sheep surveyed by culture for Pasteurella spp. in Alberta, 1982-84; includes nasal swabs, pharyngeal swabs and lungs (shot or accidental deaths).

Crowsnest	Sheep River	Banff Park	Ram Mtn.	Jasper Park	Lung Submissions	Total
1/22*	0/33	0/7	0/143	0/3	0/37	1/245

* one lamb (accidental death) positive for P. hemolytica biotype T, variant

DISCUSSION

The pneumonia outbreak spread rather rapidly from B. C. to Montana (1981 to 1983) and was first observed in each jurisdiction in the fall or early winter. The timing of the outbreaks corresponded to the breeding season of bighorns (November and December) when the animals are concentrated and the rams are moving long distances from herd to herd. Festa-Bianchet (1984) reported a linear spread of approximately 100 kilometres between radio-tagged rams from the same herd during the rut at Sheep River, north of the die-off area. The highly mobile component of rutting rams was probably responsible for spreading the disease over such a large area.

Pasteurella sp. has been previously reported in bighorn sheep with pneumonia (Post 1962, Forrester 1971, Spraker and Hibler 1977 and others). The isolates were identified as P. multocida, P. hemolytica or unidentified

species. The biotype isolated in this outbreak was consistently found in affected sheep throughout the die-off area. An interesting cultural feature was the complete absence of hemolysis which made this P. hemolytica type T a variant that was used to trace the spread of the pneumonia. This biotype variant is rarely found in livestock and has not yet been isolated from domestic sheep in Alberta. It must be cautioned not to place too much emphasis on the biochemical characteristics of the bacteria as they could change when the organisms infect a different host species.

Histopathology of the lungs was suggestive of a bacterial bronchopneumonia, although an early viral involvement cannot be excluded even in the face of negative virologic cultures. Some respiratory viruses are extremely labile and may have been destroyed by the host. Nevertheless, it is possible for Pasteurella hemolytica to cause a systemic or pneumonic infection in domestic sheep depending on the biotype (Ellis 1984). Less clear is the role of Pasteurella spp. in shipping fever pneumonia in feedlot cattle. There is little doubt that Pasteurella sp. induced the terminal lung lesions seen in the bighorns, but at the same time one has to keep in mind that Pasteurella spp. are opportunists and other factors may have played a role. The inability to recover Pasteurella sp. from the nasal passages and the pharynx of healthy bighorns may reflect presence of very low numbers or inaccessibility by the swab method if Pasteurella spp. reside in pharyngeal lymph nodes or tonsils.

There was no apparent association of the level of lungworm infection with the bronchopneumonia. It is possible however, that sheep with a high lung parasite load are more susceptible to colonization by opportunistic bacteria and thus may have formed part of the nidus from which the infection may have spread. To reflect a possible multi-etiologic cause, the disease outbreak would best be termed: 'respiratory disease complex of bighorn sheep'.

The question still remains, what was the triggering mechanism that touched off the die-off in the first herd? This problem was discussed by bighorn experts from western North America at a workshop on bighorn die-offs in Cranbrook in May 1983 (Schwantje 1983). The general concensus was that multiple stress factors appear to affect the herds susceptibility through the immunity or resistance level of individual sheep. A triggering factor that started the die-off could have been contact with domestic sheep as in Washington and California (Foreyt and Jessup 1982). However, the workshop participants agreed that there was no solid evidence that domestic sheep could be implicated as the cause of the die-off. Other factors such as inter- and intra-species competition for forage and space, harassment, or inclement weather may have initiated the outbreak. In addition, rutting activity severely drains body reserves of the older, larger rams (Geist 1971) and likely increases their susceptibility to predation, starvation or diseases.

In any event, bacteria would proliferate in the most susceptible sheep and those animals most compromised by stressors would probably die from acute pneumonia. More resistant animals might survive, develop subacute or chronic forms of pneumonia and could serve as transmitters. The possibility that some individuals survive the disease and are carriers seems to be borne out by the fact that poor lamb crops were observed following the die-offs in both British Columbia and Alberta. Festa-Bianchet (1983) noted several barren ewes and a staggered lamb crop during the summer of 1983. He was able to collect two of four coughing lambs in the fall, and both were affected with the Pasteurella

hemolytica type T variant. One of the pneumonic lambs was from a tagged ewe that showed no evidence of the disease throughout the die-off.

Poor lamb production and delayed parturition may have been due in part to a shortage of rams capable of breeding in 1982. Ewes with a subacute infection may not have come into estrus or had a delayed estrus. Subacute or chronic pneumonia may have caused some ewes to abort or to resorb fetuses. It remains to be seen if the disease continues to be manifested by poor productivity.

Experimental work using domestic sheep is underway to test two hypotheses: (a) did disease transmission occur from domestic livestock to a susceptible bighorn sheep population or (b) have opportunistic pathogens overwhelmed bighorns which were stressed by one or more factors to decrease their resistance?

So far, one trial using three sheep has failed to transmit the Pasteurella sp. isolate to domestic sheep using intranasal and intratracheal inoculations and dexamethazone as a stressor. The clinical reaction of these sheep did not go beyond a brief period of fever in response to the foreign bacterial protein. The Pasteurella sp. variant could not be reisolated. However, these sheep were previously found positive for Pasteurella multocida in their nasal flora and may have been refractory to experimental infection. Any antibody response remains to be evaluated. Before any conclusions can be drawn, further experiments will have to be carried out along with an attempt to serotype the bighorn sheep Pasteurella sp. isolate for comparison with isolates from domestic animals.

ACKNOWLEDGMENTS

The authors wish to thank Marco Festa-Bianchet, University of Calgary, Jon Jorgenson and Dale Eslinger, Alberta Fish and Wildlife and the Warden staff of Waterton National Park for their dedicated assistance in surveys and specimen collections. Our special thanks to Brenda Christian and Shirley Rawluk for their excellent microbiological culture work.

LITERATURE CITED

Buechner, H. 1960. The bighorn sheep in the United States, its past, present and future. Wildl. Monogr. No. 4. 174pp.

Davidson, P. W. 1982. East Kootenay bighorn sheep progress report. Typewritten report in files of B. C. Fish and Wildlife Branch. 56pp.

Ellis, J. A. 1984. Pasteurella hemolytica infections in sheep. The Compendium on Continuing Education for the Practicing Veterinarian. 6:360-366.

Festa-Bianchet, M. 1983. Bighorn sheep die-off in the Waterton-Crowsnest Pass area. Typewritten report in files of Alberta Fish and Wildife Division. 8pp.

Festa-Bianchet, M. 1984. Sheep River Progress Report. Typewritten report in files of Alberta Fish and Wildlife Division. 43pp.

Foreyt, W. J. and D. A. Jessup. Fatal pneumonia of bighorn sheep following association with domestic sheep. J. Wildl. Dis. 18:163-168.

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.

Geist, V. 1971. Mountain sheep: A study in behavior and evolution. Univ. Chicago Press, Chicago. 383pp.

Post, G. 1962. Pasteurellosis of Rocky Mountain bighorn sheep (Ovis canadensis canadensis). Wildl. Disease. 23:1-14.

Samuel, W. M. and J. B. Gray. 1982. Evaluation of the Baermann technic for recovery of lungworm (Nematoda, Protostrongylidae) larvae from wild ruminants. Bienn. Symp. North Wild Sheep and Goat Counc. 3:232-243.

Schwantje, H. M., ed. 1983. Bighorn sheep die-off workshop proceedings. Typewritten report in files of British Columbia Fish and Wildlife Branch. 41pp.

Spraker, T. R. and C. P. Hibler. 1977. Summer lamb mortality of Rocky Mountain bighorn sheep. Trans. Desert Bighorn Counc. pp.11-12.

Spraker, T. R. 1979. The pathogenesis of pulmonary protostrongylosis in bighorn lambs. Ph. D. Thesis. Colorado State Univ., Fort Collins. 233pp.

Stelfox, J. G. 1971. Bighorn sheep in the Canadian Rockies: A history 1800-1970. Can. Field-Naturalist. 85:101-122.

Wishart, W. D., J. Jorgenson and M. Hilton. 1980. A minor die-off of bighorns from pneumonia in southern Alberta (1978). Bienn. Symp. North. Wild Sheep and Goat Counc. 2:229-245.