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AN EPIZOOTIC OF *MYCOPLASMA OVIPNEUMONIAE* INFECTION IN CAPTIVE DALL'S SHEEP (*OVIS DALLI DALLI*)

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ABSTRACT: In late spring of 1986, 10 of 23 Dall's sheep (*Ovis dalli dalli*) at the Metropolitan Toronto Zoo were moved to a new exhibit, where all developed severe respiratory signs refractory to anthelmintic and antibiotic therapy. In July, two animals died with chronic active bronchopneumonia, and a third was euthanized because of pneumonia several months later. Bacteria were not isolated from the lungs of the first, streptococci and *Pasteurella hemolytica* were isolated from the other two, respectively; *Mycoplasma ovipneumoniae* was isolated from both. Pulmonary lesions in all three sheep were consistent with *Mycoplasma* sp. infection. Nasal swabs of the remaining animals yielded no consistent bacterial isolates; however, four of eight sheep were positive for *M. ovipneumoniae*. Viral cultures yielded an as yet unidentified herpesvirus. Sheep in the original and new herds had no serologic titers to parainfluenza-3, equine viral rhinopneumonitis, or infectious bovine rhinotracheitis, and had variable titers against bovine respiratory syncytial virus. No titers against *M. ovipneumoniae* were present in 13 sheep still in the original exhibit, but titers varied from 1:32 to 1:256 in eight pneumonic sheep. Sera taken from three sheep before or early in the outbreak were all negative for antibody to *M. ovipneumoniae*. Two of the affected Dall's sheep had been in contact with domestic sheep in the winter of 1985–1986, and *M. ovipneumoniae* was subsequently cultured from the domestic flock. Exposure to a new pathogen, and environmental and social stress in a new exhibit may have resulted in this severe disease in Dall's sheep.

Key words: Dall's sheep, *Ovis dalli dalli*, pneumonia, *Mycoplasma ovipneumoniae*, pathology, epizootiology, virology, serology.

INTRODUCTION

Dall's sheep, *Ovis dalli*, live at altitudes from 1,800 to 3,000 m in Alaska, the Yukon and northern British Columbia in North America. In general, the climate is cold and dry (Banfield, 1974). High mortality, particularly of lambs and old sheep, occurs during severe winters (Burles and Hoefs, 1984). Little information is available regarding infectious disease in this species with the exception of several serosurveys (Foreyt et al., 1983; Zarnke, 1983; Zarnke et al., 1983). A captive herd of *O. dalli dalli*, the northern subspecies, is held at the Metropolitan Toronto Zoo (Toronto, Ontario, Canada M1E 4R5). In this paper we report an outbreak of pneumonia, associated with *Mycoplasma ovipneumoniae*, in this herd.

HISTORY AND CLINICAL FINDINGS

Toronto, Ontario, Canada lies at a latitude of 43°N, and usually experiences a cold wet winter and hot, humid summer. The Dall's sheep at the zoo originated from four males and eleven females obtained from the Yukon Game Farm (P.O. Box 4100, Whitehorse, Yukon Territory, Canada) and Calgary Zoo in Alberta, in 1973. Since then, only three outside animals, all captive bred from the Yukon Game Farm, Calgary Zoo, and Los Angeles Zoo, have been added to the herd. The last acquisition was made in 1983.

The original exhibit in the Metropolitan Toronto Zoo is in a wooded river valley, with a low concrete hill and shade trees. The area is accessible to visitors only by monorail and to staff by truck; human con-

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tact is limited to daily visits by one or two keepers. There are no other species of sheep or goats in this area. In fall 1984, a few females were temporarily moved to the health unit building for an embryo transfer project. The next year (winter 1985–1986), two females involved in that study and designated here as number 4 and number 10, were housed there for several months for observation of parturition. Two domestic Dorset sheep were held in an adjacent pen in the health unit. The pens were separated by chain link fencing covered with 6-mm plywood, but were serviced by the same keeper. The two Dall's sheep developed an occasional cough, and clear nasal discharge in April 1986.

A new exhibit for Dall's sheep was built on the main zoo grounds in 1985. In early June 1986, the two females from the health unit (number 4 and number 10) as well as eight sheep from the valley exhibit (numbers 1 to 3, 5 to 9) were moved to the new exhibit, leaving 13 sheep (numbers 11 to 23) in the original exhibit. In the new holding area there was a large rock-work concrete mountain and relatively close contact with zoo visitors. The shade inside the mountain was not utilized and summer daytime temperatures were in excess of 30 C on the concrete.

All animals were clinically normal when they entered the new exhibit, but by 26 June, seven of these 10 sheep had occasional dry coughs and a mild nasal discharge. There was a mild increase in pulmonary sounds on auscultation, mainly during the expiratory phase.

Clinical examinations throughout the epizootic involved visual assessment of condition, nasal and ocular discharges, mucous membrane colour and capillary refill time. Auscultation was conducted. Body temperature was considered inapplicable as animals were excited and struggling.

Within 10 days, despite treatment with oxytetracycline (Agrioxytet 62®, Agri-Vet Pharm. Ltd., 410 Ormont Dr., Weston, Ontario, Canada M9L 1N9) in drinking water, and intramuscular long-acting penicillin (Penlong XL®, Rogar/STB Inc., 1 Wilton Grove Rd., London, Ontario, Canada N6A 4C6), all animals developed a mucopurulent nasal discharge and severity of the cough, now productive, increased. About 40% of the herd had severe respiratory distress, with rales audible over the cranial half of the thorax. Treatment was changed to intramuscular trimethoprim sulfa (Trivetrin®, Cooper Products Div. Burroughes Wellcome, 60 Riverview Ave., LaSalle, Quebec, Canada H8R 3S1) and all animals were given ivermectin (Ivomec®, Merck Frosst Canada Inc., 1745 Meyerside Dr., Mississauga, Ontario, Canada L4T 3Y6), but there was no reduction of clinical signs. After another 4 days, one half of the herd had very rapid respiratory rates, mouth breathing and very loud coarse rales over most of the thorax. Two older females developed emphysema; fine rales were audible at the nose, without auscultation. At this time, the most severe cases were treated with ampicillin (Penbritin®, Ayerst Laboratories, 1025 Laurentian Blvd., Montreal, P.Q. H4R 1J6) and flunixin meglumine (Banamine®, Schering Canada Inc., 3535 Trans-Canada Highway, Pointe Claire, Canada H9R 1B4) intravenously, the others with ampicillin intramuscularly. A moderate response was noted after 10 days of therapy.

Shortly after cessation of this antibiotic regime, clinical signs deteriorated. Again the signs were mucopurulent nasal discharge, productive hacking cough, rales audible over most of the thorax and mouth breathing. Sheep number 10, an old female, died. Treatment for 1 wk with a combination of penicillin G (Ethacillin®, Rogar/STB Inc., 1 Wilton Grove Rd., London, Ontario, Canada N6A 4C6) and gentamicin (Gentocin®, Schering Canada Inc., 3535 Trans-Canada Highway, Pointe Claire, Canada H9R 1B4) was successful in decreasing the severity of clinical signs in most animals, though sheep number 9, a yearling male, died.

This pattern continued through August

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and September with variable to poor responses seen to prolonged courses of treatment with spectinomycin (Spectam®, P.V.U. Inc., Rural Route 6, Highway 24, Guelph, Ontario, Canada N1H 6J3), levamisole phosphate (Ripercol®, Cyanamid Canada Inc., 2255 Sheppard Ave. E., Willowdale, Ontario, Canada N2J 4Y5), tylosin (Tylan®, Elanco Ltd., 535 Blvd., Sir Wilfred Laurier, Suite 303, Beloeil, P.Q. J3G 5E9), and oxytetracycline (Liquamycin LA®, Rogar/STB Inc., 1 Wilton Grove Rd., London, Ontario, Canada N6A 4C6). Several animals lost considerable body condition, and clinical signs never disappeared completely. A breeding male (number 13) was introduced from the unaffected herd in early December. Within 3 wk, he developed some nasal discharge and occasional coughing. As the disease progressed, he lost weight and his activity level decreased.

In January 1987, animal number 8, a 2-yr-old male, was euthanized because of chronic pneumonia and regurgitation. In July 1987, 1 yr after the onset of the epizootic, some animals still exhibited poor body condition, impaired exercise tolerance, mucopurulent nasal discharge and occasional coughing.

LABORATORY METHODS

Several methods were used in attempting to diagnose the cause of the outbreaks of pneumonia. The investigation was constrained by difficulty in handling the animals, and attempting to keep handling stress to a minimum.

Blood was collected by jugular venipuncture both before the pneumonia began, and at intervals during subsequent handling. Serum was frozen at −20 C.

Nasal swabs were taken using swabs with transport medium (S. P. Culturette, American Scientific Products, A Division of Travenol Laboratories Inc., 1430 Waukegan Rd., McGaw Park, Illinois 60085, USA), avoiding outer membranes and swabbing mucosa approximately 5 cm within the nasal cavity. Bacterial cultures were conducted on blood agar and MacConkey agar, and incubating at 37 C. The Enterotube® system (Hoffmann-La Roche Ltd., Suite 700, 401 The West Mall, Etobicoke, Ontario M9C 5J4), was used to identify gram-negative bacteria. Cultures for *M. ovipneumoniae* were carried out using Friis' broth with 0.1% glucose and solidified with 0.8% purified agar (Goltz et al., 1986). Specimens were cultured for other mycoplasmas and ureaplasmas as previously described (Goltz et al., 1986).

Nasal swabs for viral cultures were placed in 3 ml of Hanks' balanced salt solution with 0.5% lactalbumin hydrolysate (GIBCO Laboratories, Grand Island, New York 14072, USA). To this solution had been added by sterile means 20 μg/ml of streptomycin, 500 I.U./ml of Mycostatin, and 500 I.U./ml of penicillin (GIBCO Laboratories, Grand Island, New York 14072, USA). Swabs were frozen at −20 C until cultured in Madin Darby bovine kidney cells (American Type Culture Collection No. CCL 22). A herpesvirus isolated was tested by virus neutralization with hyperimmune rabbit serum prepared in this laboratory against equine herpesvirus type 1, bovine herpesvirus type 1, and a herpesvirus isolated from pronghorn antelope (Thorsen et al., 1977).

Serum samples included (1) a few obtained from sheep just prior to developing pneumonia or very early in the outbreak, (2) some from pneumonic sheep in September, during a recrudescence of clinical signs, and (3) sera obtained from the 13 clinically normal sheep from the original exhibit in October 1986.

Mycoplasma ovipneumoniae titers were determined by an indirect hemagglutination test (Cho et al., 1976), using strain B321B, isolated from a field case in an Ontario goat by S. Rosendal, as an antigen. This strain was identified as *M. ovipneumoniae* on the basis of colony morphology and growth inhibition by hyperimmune rabbit serum against *M. ovipneumoniae,* reference strain Y-98 (FAO-WHO International Reference Center for Animal Mycoplasmas, Aarhus, Denmark).

Serology was conducted to determine titers against infectious bovine rhinotracheitis (IBR) and parainfluenza-3 (PI_3) strains originally acquired from Dr. M. Savan (Ontario Veterinary College, Guelph, Ontario, Canada N1G 2W1), a Kentucky D strain of equine viral rhinopneumonitis (EVR) from J. Thorsen (OVC, Guelph) and a cell culture adapted bovine respiratory syncytial virus (BRSV) isolate, a gift from L. Babiuk (Western College of Veterinary Medicine, Saskatoon, Saskatchewan, Canada S7N 0W0).

Standard virus neutralization assays were performed in microtiter to determine serum antibody titers for IBR, EVR and BRSV. Two-fold serial dilutions of heat inactivated (56 C, 30 min) test serum were mixed with equal volumes of virus, containing 100 $CCID_{50}$. For IBR and EVR, the mixtures were incubated for 1 hr at 37 C, while BRSV mixtures were incubated at 4 C for

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1 hr. At the end of this incubation period, embryonic bovine spleen cells (courtesy of L. Babiuk) were added to the IBR and EVR tests. Georgia bovine kidney cells were used for the BRSV assay. Plates were incubated for 4 or 5 days at 37 C in a CO_2 humidifier incubator. Sera were tested in duplicate and known positive and negative control sera were included.

Antibodies to PI_3 were determined using a microtiter hemagglutination-inhibition assay. Two fold serial dilutions of heat inactivated test serum were mixed in V-bottomed microtiter plates with an equal volume of cell culture supernatant containing 4 hemagglutination units of PI_3 hemagglutinin. After incubation at room temperature for 30 min, a 0.3% bovine red blood cell suspension was added and the plates further incubated for 3 to 4 hr at room temperature. Known positive and negative control sera were included in the assay.

Necropsies were performed on the two Dall's sheep which died during July of 1986, and a third, that was euthanized in January 1987. Tissue samples were preserved in buffered 10% formalin, routinely processed and sections for examination were stained with hematoxylin and eosin. Congo Red and Gram (Brown + Brenn) stains (Luna, 1968) were applied to selected sections for identification of amyloid and bacteria, respectively.

Samples of tissue approximately 2 cm^3 were collected in sealed plastic bags and frozen at −20 C for bacterial, viral and mycoplasmal culture. Tissue was collected from two sites in the lung, anteroventral and dorsocaudal, as well as from the liver, spleen, and kidney. Samples for culture were taken from the area adjacent to where samples were collected for histology.

RESULTS

Pathology

Sheep number 10, an old female, died with uncomplicated chronic active fibrinopurulent bronchopneumonia. The trachea was congested and contained froth and purulent mucus distally. There was marked consolidation of anteroventral lung lobes, which were pale and exuded small amounts of purulent material from airways on cut section. There was fibrinous pleuritis, and there were numerous pleural adhesions. The remainder of the lung parenchyma was dark red, heavy and exuded blood on cut section. There were numerous 1-mm white foci, representing peribronchiolar lymphoid cuffs, on the cut surface of dorsal lobes. The liver was pale, as were adrenal cortices, which had a 1:1 ratio with the medulla. Bacteria were not isolated from lung, liver, spleen or kidney. Cultures for *Mycoplasma* sp. were not carried out.

Microscopic lesions were variable in case number 10; there was mild lymphoid cuffing of airways and many alveolar septa were thickened by congestion and fibrosis, or by infiltration with mixed inflammatory cells. Neutrophils, alveolar macrophages, free erythrocytes and proteinaceous edema fluid filled alveoli to varying degrees. Pleura and interlobular septa were thickened by fibrin, edema and inflammatory cells. Accumulations of amyloid were present in several tissues, including the adrenal gland, renal glomeruli, splenic follicles, liver and intestinal mucosa.

The second death, animal number 9, was due to subacute necrotizing bronchopneumonia, complicated by fibrinous pleuritis and peritonitis. Findings in the trachea and lungs were similar to the first case, although there was less consolidation, and more widespread and severe edema. Additionally, extensive sheets of fibrin up to 2 cm thick covered thoracic and abdominal organs. Large numbers of alpha streptococci were isolated from the lung and liver, and *M. ovipneumoniae* was isolated from the lung.

Microscopic lesions in the lung of animal number 9 varied from marked consolidation with neutrophils and edema filling alveoli, and areas of caseous necrosis containing gram-positive cocci, to less severe thickening of alveolar septa by mononuclear inflammatory cells, with scattered neutrophils and macrophages in alveoli. Cuffing of airways by mononuclear inflammatory cells was prominent. Pleura and interlobular septa were thickened as in the previous case, and sheets of fibrin covered the pleura.

At necropsy of case number 8 the trachea and bronchi were filled with heavy mucopurulent exudate. Both lungs were very firm and dark red anteroventrally, with prominent peribronchiolar cuffing on

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cut surface. Dorsocaudally the lungs were pink and slightly firmer than normal. Adrenal glands had a thick pale cortex, and the liver had a zonal red and yellow pattern. Low numbers of *Pasteurella hemolytica* and *M. ovipneumoniae* were cultured from the lung.

Microscopic pulmonary lesions in animal number 8 were very severe (Fig. 1). Consolidation of the parenchyma was widespread, airway epithelium was hypertrophic and lacked cilia in many areas, and the lumina were filled with neutrophils and cellular debris. Lymphocytic cuffs were very marked around airways. Some foci of parenchyma were scarred. Alveoli had thickened septa, containing edema, neutrophils and other inflammatory cells. Alveolar macrophages were prominent, and most alveoli were flooded by edema fluid. A few areas of parenchyma were emphysematous. Amyloidosis was similar to that observed in case number 10.

Microbiology and serology

Results of bacterial cultures of nasal swabs were variable. Swabs taken from three separate individuals before the onset of clinical signs yielded no growth in one case, and *Staphylococcus epidermidis* in the other two animals. After the onset of clinical signs, nasal swabs from seven sheep were cultured for bacteria. Several mixed cultures were obtained, with no consistent isolates (*Klebsiella* sp. in one of seven; *Pseudomonas* sp. in two of seven; *Streptococcus* in four of seven; *Yersinia enterocolitica* in one of seven; *Pasteurella hemolytica* in one of seven; *Bacillus* sp. in two of seven).

Mycoplasma cultures were completed on eight nasal swabs from pneumonic sheep in August, and *M. ovipneumoniae* was isolated from four of these. The breeding male (number 13) introduced in December had a positive culture for *M. ovipneumoniae* in January 1987. All 13 healthy sheep from the original exhibit (including sheep number 13) had negative *Mycoplasma* sp. cultures on nasal swabs taken in September 1986. *M. ovipneumoniae* was isolated from nasal swabs of two of six domestic sheep on the zoo site in October 1986. No other mycoplasmas or ureaplasmas were isolated from any of the animals.

Viral cultures of nasal swabs from affected Dall's sheep yielded an unidentified herpesvirus in four of eight samples. This virus is antigenically similar to equine herpesvirus 1 (EVR), and to another unidentified herpes virus previously isolated from pronghorn antelope in Alberta (Thorsen et al., 1977).

Serologic titers to *M. ovipneumoniae* are summarized in Table 1. All four affected animals for which paired samples were available seroconverted to *M. ovipneumoniae* during the course of the epizootic (Table 1). Titers in animals sampled once during the course of the outbreak were all elevated, while none of the animals held in the original exhibit had a titer to *M. ovipneumoniae*. Titers to BRSV in eight clinically affected animals were: <1:2 (1), 1:16 (1); 1:32 (1); 1:48 (2) and <1:256 (3). Titers in some affected animals were higher after the onset of the outbreak than those in the 13 animals from the original herd, which ranged from 1:2 to 1:96. Titers against EVR, IBR or PI_3 were not detected in any animal.

DISCUSSION

Direct and circumstantial evidence strongly implicates *M. ovipneumoniae* as the etiologic agent responsible for this outbreak. Environmental stressors may have been involved as well. The organism was isolated from the lungs of animals dying with pneumonia, and from nasal swabs of some affected sheep. The gross and microscopic lesions seen at necropsy, particularly lymphocytic cuffing of airways, are characteristic of *Mycoplasma* sp. pneumonia in sheep (Sullivan et al., 1973a, b; Foggie et al., 1976) and goats (Goltz et al., 1986).

Seroepidemiologic studies demonstrated that the Dall's sheep herd of origin had not been exposed to *M. ovipneumoniae*,

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FIGURE 1. Peribronchiolar lymphoid cuff, and marked exudation of proteinaceous edema fluid and inflammatory cells into alveoli in lung of Dall's sheep number 8.

but animals transferred to the new exhibit seroconverted to the organism during the course of the outbreak. The probable source of the present infection was asymptomatic domestic sheep, in some of which *M. ovipneumoniae* infection was demonstrated. The organism was likely introduced to the Dall's sheep herd by animals (numbers 4 and 10) housed adjacent to several of these domestic sheep during the course of embryo transfer studies.

No other pathogenic bacterial agents were consistently recovered, nor, with the exception of BRSV, was there serological evidence for widespread infection with potential pneumopathic viral agents. The herpesvirus isolated is considered incidental because of the lack of evidence of seroconversion to the antigenically related EVR virus. The significance of the BRSV as a contributing factor in this outbreak is unclear. A few affected animals did develop high titers, and although serological evidence for infection suggests that it is widespread in healthy and affected sheep, as it is in wild herds of bighorn sheep (Dunbar et al., 1985; Spraker et al., 1986), there is a possibility that it may have promoted pathogenicity of *Mycoplasma* sp. infection.

A similar outbreak of chronic pneumonia with high morbidity occurred in captive bighorn sheep (*Ovis canadensis*), in which gross and histological lesions resembled those described here (Woolf et al., 1970). *Mycoplasma* sp. were isolated consistently from the lungs of dead sheep and nasal cultures of affected live sheep. There is a second report of a *Mycoplasma* sp., isolated from the lungs of five pneumonic bighorn sheep, that was found to be the same strain as that cultured by Woolf (Al-Aubaidi et al., 1972). This organism was typed as *M. arginini* and its role in disease was not investigated further. It is possible that *M. arginini* causes lesions in bighorn sheep similar to those seen in the present outbreak.

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TABLE 1. Results of IHA titers to *Mycoplasma ovipneumoniae* in sera from clinically affected and healthy Dall's sheep.

Animal no.	Date of sample	Condition of animal	Titer
1	7 July 1986	Early mild pneumonia	<1:2
	20 Sept. 1986	Chronic pneumonia	1:128
2	7 July 1986	Early mild pneumonia	<1:2
	20 Sept. 1986	Chronic pneumonia	1:256
3	19 June 1986	Healthy	<1:2
	20 Sept. 1986	Chronic pneumonia	1:128
4	19 June 1986	Healthy	<1:2
	20 Sept. 1986	Chronic pneumonia, wasting	1:256
5	20 Sept. 1986	Chronic pneumonia, wasting	1:128
6	20 Sept. 1986	Chronic pneumonia, wasting	1:32
7	20 Sept. 1986	Chronic pneumonia, wasting	1:256
8	20 Sept. 1986	Chronic pneumonia, wasting	1:64
11 to 23	2 Oct. 1986	Healthy, original exhibit	<1:2
13	15 Jan. 1987	Mild pneumonia, weight loss	1:128

The mode of infection from domestic to Dall's sheep can be speculated to be through aerosol or fomites. Sullivan et al. (1973b) found that *M. ovipneumoniae* infection could be induced by exposing lambs to aerosolized bacteria, or to lambs which had been previously infected by aerosol. The clinical history of *M. ovipneumoniae* in the Dall's sheep herd, that of a chronic low grade recrudescent infection, resembles that described in domestic sheep (Sullivan et al., 1973a). Although *M. ovipneumoniae* is potentially pathogenic in domestic sheep, it is a common isolate from the nasal cavity and lungs of clinically normal sheep (Alley et al., 1975).

Mycoplasma ovipneumoniae appears to have high pathogenicity in Dall's sheep. On domestic lamb and goat tracheal explant cultures, the organism multiplies, is persistent and causes ciliostasis (Jones et al., 1985; Goltz et al., 1986). However, results from studies on live lambs are variable. It has been associated with mild cuffing pneumonia in lambs (Sullivan et al., 1973b; Foggie et al., 1976; Alley and Clark, 1979) and may potentiate pneumonia due to *Pasteurella hemolytica* (Jones et al., 1982); other studies have found that it causes little disease in lambs (Davies et al., 1981).

There are several conditions which may have predisposed these Dall's sheep to clinical disease, enhancing the virulence of *M. ovipneumoniae*. The presence of high BRSV titers in some pneumonic sheep suggests that infection may have been active, although normal and diseased sheep had titers to BRSV. This virus is associated with a mild, non-fatal pneumonia in domestic lambs (Cutlip and Lehmkuhl, 1979; Lehmkuhl and Cutlip, 1979). BRSV may promote the virulence of *M. ovipneumoniae* by causing mild pulmonary lesions.

There were numerous environmental and social stressors acting on the herd involved in the epizootic at the Metropolitan Toronto Zoo. A large herd was fragmented, and eight sheep from the valley were moved to a new exhibit and mixed with two others. The new exhibit had increased human contact; the animals tended not to seek the shade provided and hence were subject to high temperatures. Outbreaks of *Mycoplasma* sp. infections in several species are associated with stressors such as crowding, chilling, shipping and fatigue (Stalheim, 1983). Spraker et al. (1984) describe 75 to 80% mortality due to pneumonia within a wild bighorn sheep herd subjected to several environmental stressors.

Dall's sheep in this outbreak suffered from more severe disease than that asso-

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ciated with *M. ovipneumoniae* in domestic sheep. Some studies undertaken at the zoo indicate that Dall's sheep may have less ability to respond immunologically to potential pathogens than either mouflon (*Ovis musimon*) or domestic sheep. At 1 day and 1 mo of age, serum IgG and IgM levels are significantly lower in Dall's sheep than in the other two species, although immunoglobulin levels in adults are not significantly different (G. Crawshaw, K. Mehren, R. Maser and S. Carstairs-Grant, 1986, unpubl. data). The lack of splenic lymphoid tissue in the most severely affected sheep dying in the present study may indicate some level of immune suppression or exhaustion. In addition, virulence of *Mycoplasma* spp. infections may be enhanced by immunosuppression associated with *Mycoplasma* sp. cell membranes (Bergquist et al., 1974; Bennett and Jasper, 1977).

Many forms of pneumonia have been associated with clinical disease and mortality in wild sheep in North America. This outbreak is not of direct relevance to the most widespread problem, the bighorn sheep pneumonia complex (Spraker and Hibler, 1982), but it serves to illustrate the susceptibility of wild sheep to relatively innocuous pathogens of domestic stock. Many wild populations are immunologically naive to many of these common pathogens. Foreyt and Jessup (1982) describe two epizootics of fatal pneumonia in bighorn sheep shortly after exposure to clinically normal domestic sheep. This kind of incident can, and should, be avoided in captive wildlife. Similar outbreaks in wild situations may have a more severe impact. If isolation of wild hoofstock from domestic animals is not possible, populations must be maintained at numbers high enough to recover from epizootic disease, or managed so as to minimize transmission within or between herds.

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