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SPONTANEOUS PASTEURELLOSIS IN CAPTIVE ROCKY MOUNTAIN BIGHORN SHEEP (*OVIS CANADENSIS CANADENSIS*): CLINICAL, LABORATORY, AND EPIZOOTIOLOGICAL OBSERVATIONS

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ABSTRACT: We observed clinical signs, compared adrenal responses, and performed diagnostic tests on 12 captive Rocky Mountain bighorn sheep (*Ovis canadensis canadensis*) during a spontaneous outbreak of pasteurellosis. Cortisol in urine and feces was measured for bighorns sampled three times between 20 October and 1 November 1986. By 6 November, four of these had developed pneumonia, four showed only mild rhinitis, and four remained clinically normal. Bighorns that ultimately developed pneumonia showed elevated mean urinary ($P = 0.003$) and fecal ($P = 0.046$) cortisol excretion over the 12-day sampling period. Twenty-four hour mean urine cortisol:creatinine ratios ranged from 10 to 57 ng/mg dry matter for affected and 5 to 22 ng/mg for healthy individuals; 24 hr mean fecal cortisol concentrations ranged from 7.2 to 20 ng/g dry matter for affected and 3.6 to 9.1 ng/g dry matter for healthy individuals. Elevated cortisol excretion preceded clinical pneumonia in affected bighorns by ≤ 16 days. Beta-hemolytic *Pasteurella haemolytica* biotype T, serotype 3 or 4, was isolated from nasal and pharyngeal swabs from all eight bighorns with pneumonia or mild rhinitis. We detected no evidence of parainfluenza 3, bovine respiratory syncytial virus, or *Chlamydia psittaci* using fluorescent antibody and/or serologic tests. Although elevated cortisol excretion was associated with pneumonia, we also believe age, reproductive physiology, and/or prior recovery from clinical pasteurellosis may have influenced individual susceptibility to pneumonia during this epizootic.

Key words: Rocky Mountain bighorn sheep, *Ovis canadensis canadensis*, pneumonia, cortisol, stress, epizootiology, pasteurellosis, *Pasteurella haemolytica*.

INTRODUCTION

Chronic stress is one factor believed to predispose bighorn sheep (*Ovis canadensis*) to pneumonia (Hudson, 1972, 1973; Feuerstein et al., 1980; Spraker and Hibler, 1982; Onderka and Wishart, 1984; Spraker et al., 1984). Anecdotal accounts of pneumonia outbreaks in bighorn populations suggest that environmental disturbances frequently precede disease outbreaks (Feuerstein et al., 1980; Spraker et al., 1984; Bailey, 1986; Festa-Bianchet, 1988). In theory, adrenal responses stimulated by these environmental stressors cause corticoid-mediated immunosuppression, increase susceptibility of bighorns to infectious diseases, and thereby increase mortality rates.

Although circumstantial evidence from field observations suggests that environmental stress may be associated with pneumonia in bighorn sheep, experimental data supporting this hypothesis have not emerged. To the contrary, experimentally-imposed stress resulting in measurable adrenal responses in captive bighorns failed to induce pneumonia (Hobbs et al., 1985; Harlow et al., 1987; Miller, 1988; Miller et al., 1991). Here, we describe adrenal responses of affected and unaffected captive bighorns during a pneumonia epizootic. We also report epizootiological and clinical observations, as well as diagnostic findings, made during this outbreak.

HISTORY

The Colorado Division of Wildlife (CDOW) Wildlife Research Center (317 West Prospect Road, Fort Collins, Colorado 80526, USA) has maintained a herd of captive Rocky Mountain bighorn sheep (*O. canadensis canadensis*) for research purposes since 1977. Most of these sheep

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have been hand-raised, and all appear well-adapted to captivity. Pasteurellosis first spread through our captive herd during October to December 1984. The index case for this epizootic was a dam-raised lamb that died suddenly in mid-October. Gross and histologic examinations revealed lesions of acute fibrinous bronchopneumonia, and *Pasteurella* sp. was isolated from lung tissue. In all, seven of 19 bighorns developed clinical pneumonia by December, and three of those (two lambs and a 6-yr-old ewe) died. Isolation procedures and prolonged oral and injectable long-acting oxytetracycline (Liquamycin® LA200®, Pfizer Agricultural Division, New York, New York 10017, USA) therapy were credited with halting this outbreak and saving four affected ewes (A82, C83, G78, T82) (Table 1). It is possible that *Pasteurella* sp. was introduced by one of eight bighorns that had been added to our herd within the previous year, but no samples were collected to screen new animals prior to their introduction.

Aside from occasional nasal discharge in two ewes (C83, T82) and one ram, none of the adult bighorns showed respiratory signs after the outbreak subsided in December 1984. However, two of three dam-raised lambs born in May 1985 to previously affected ewes (C83, T82) died by late July. Gross lesions were consistent with acute bronchopneumonia, and *Pasteurella* sp. was isolated from lungs of both lambs. The third lamb (A85) developed pneumonia in September, after bighorns from the Research Center were moved to CDOW's Foothills Wildlife Research Facility (Fort Collins, Colorado 80521, USA; 40°35′N, 105°10′W). That lamb recovered after a 3-wk course of injectable long-acting oxytetracycline therapy. Over the next 12 months, no other bighorns were affected (Table 1).

In October 1986, we initiated a study at our foothills facility to examine utility of cortisol concentrations in bighorn urine and feces for detecting adrenal responses elicited by chronic adrenocorticotropic hormone (ACTH) administration (Miller et al., 1991). Before we could begin imposing experimental treatments, pneumonia developed in four of 12 captive bighorns selected for that study. This development allowed an unusual opportunity to examine relationships between adrenal responses and respiratory disease in bighorn sheep.

TABLE 1. Abbreviated health histories emphasizing prior pneumonia experience for 12 captive bighorns sampled during the 1986 pasteurellosis outbreak. See text for a more complete historical account.

Ear tag number[b]	Sex	Clinical condition—September to October[a]		
		1984	1985	1986
B82	CM[c]	N (A)	N	P/D[d,e]
G77	CM	N (A)	N	P/R[f]
M86	M	—	—	P/R[f]
Q86	F	—	—	P/R[e]
A85	F	—	P/R	RH[e]
C83	F	P/R	RH	RH[g]
D83	M	N	N	RH[f]
T82	F	P/R	RH	RH[f]
A82	F	P/R	N	N
E83	F	N	N	N
G78	F	P/R	N	N
H83	M	N	N	N

[a] N, normal; N (A), normal, held at alternate facility; RH, rhinitis; P/R, pneumonia, recovered; P/D, pneumonia, died.
[b] Ear tag numbers indicate name/lineage (A-T) and year of birth (1977 to 1986); i.e., A82 and A85 are related, born in 1982 and 1985, respectively.
[c] CM, castrated male.
[d] Beta-hemolytic *Pasteurella haemolytica* biotype T, serotype 3 isolated from pharyngeal swabs.
[e] Beta-hemolytic *P. haemolytica* biotype T, serotype 4 isolated from nasal swabs.
[f] Beta-hemolytic *P. haemolytica* biotype T, serotype 3 isolated from nasal swabs.
[g] Unserotyped beta-hemolytic *P. haemolytica* biotype T isolated from nasal swabs.

MATERIALS AND METHODS

Animals and housing

Twelve hand-raised bighorns (Table 1) had been selected for use in the aforementioned experiment. On 13 October 1986, all 12 sheep entered individual isolation pens (about 50 m²), where they were housed before and between excreta collections (described below). During excreta collections, each bighorn was confined in an individual metabolic cage (about 5 m²). Pens and cages were arranged so that confined bighorns could see and contact each other, and

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all sheep were previously trained for and appeared acclimated to this type of confinement. We provided alfalfa hay, pelleted feed (Baker and Hobbs, 1985), mineralized salt, and water *ad libitum*.

Clinical observations

Because we detected upper respiratory signs (coughing, nasal discharge) in one bighorn just prior to beginning excreta collections, we began monitoring all penned sheep daily for clinical signs and/or progression of respiratory disease on 20 October. In particular, we noted presence and severity of coughing, as well as changes in nasal secretions or behavior suggestive of respiratory disease. We classified nasal discharge with or without occasional coughing in the absence of depression and anorexia as rhinitis. Clinical pneumonia was distinguished from rhinitis by a combination of signs including depression, anorexia, coughing, and mucopurulent nasal discharge. Bighorns that developed pneumonia were treated using about 22 mg/kg of long-acting oxytetracycline (Liquamycin® LA200®, Pfizer Agricultural Division, New York, New York 10017, USA) injected subcutaneously every other day.

Urine and fecal cortisol measurements

We made three 48 hr excreta collections from bighorns on 21 to 22 October, 26 to 27 October, and 31 October to 1 November. Bighorns were confined in individual metabolic cages throughout each collection period. These cages were constructed of galvanized steel with mesh flooring. A galvanized steel pan that narrowed into a funnel was overlayed with screening and placed beneath each cage floor to collect excreta. Feces were deposited on the screening, while urine passed through to the pan and drained into a 1-L plastic bottle. This layering effectively separated feces from urine and minimized cross-contamination of excreta samples. We collected urine and feces twice daily, and made two 24 hr composites for each bighorn per sampling period. Urine samples were filtered immediately after collection. All samples were stored at −20 C until assayed.

Cortisol was measured by an extracted double-antibody radioimmunoassay validated for use with bighorn urine and feces (Miller, 1988; Miller et al., 1991). Our methods for measuring urine and fecal cortisol and urine creatinine in bighorn samples followed those described by Miller et al. (1991). Cortisol assays were performed by the Endocrinology Laboratory, Department of Physiology, Colorado State University (Fort Collins, Colorado 80523, USA). Creatinine assays were performed by the Clinical Pathology Laboratory, Department of Clinical Sciences, Colorado State University (Fort Collins, Colorado 80523, USA). We used urine cortisol:creatinine ratios (ng/mg) and fecal cortisol concentrations (ng/g dry matter feces) to compare cortisol levels in 24 hr composite samples.

Diagnostic samples

We collected nasal and pharyngeal swabs from B82 on 31 October, and nasal swabs from the other 11 bighorns on 6 November. Swabs were transported in modified Amies medium with charcoal (BBL Microbiology Systems, Becton Dickinson and Co., Cockeysville, Maryland 21030, USA) to the Colorado Veterinary Diagnostic Laboratory (CVDL) (Colorado State University, Fort Collins, Colorado 80523, USA) for bacterial isolation using standard techniques (Carter, 1984); we serotyped *P. haemolytica* isolates using the rapid plate agglutination methods of Frank and Wessman (1978). We bled all 12 bighorns on 12 November, collected serum, and submitted those samples to CVDL for serology. Titers to parainfluenza 3 (PI3) were measured using hemagglutination inhibition (Rosen, 1969), and titers to bovine respiratory syncytial virus (BRSV) were measured by serum neutralization (Carbrey, 1971).

One bighorn that died was necropsied, and gross and histologic lesions were described. Lung and suprapharyngeal lymph node were submitted to the Wyoming State Veterinary Laboratory (University of Wyoming, 1174 Snowy Range Road, Laramie, Wyoming 82070, USA) for bacteriology, and lung was examined by fluorescent antibody tests (Jones et al., 1978) for BRSV, PI3, and *Chlamydia psittaci*.

Data analysis

Differences in urine and fecal cortisol excretion related *post hoc* to clinical condition were examined by one-way analysis of variance for a completely random design with a repeated measures structure using the SAS System for General Linear Models (Freund et al., 1986). Individual animals were replicates in the analysis, and we repeated over collection periods. We assumed all effects were fixed.

RESULTS

Clinical course

Four of 12 bighorns (B82, G77, M86, Q86) developed pneumonia between 20 October and 7 November (Table 1). In those animals, upper respiratory signs preceded clinical pneumonia by 8 to 16 days. We first observed mild coughing and serous nasal discharge in B82 on 20 October,

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1 day before excreta sampling began. Within 2 days, two more animals (G77, Q86) were coughing occasionally, but no nasal discharge was evident. By 29 October, B82 and G77 had developed pneumonia, and M86 and Q86 showed frequent coughing and serous nasal discharge. Within 9 days, M86 and Q86 also developed pneumonia. We began antibiotic therapy for B82 on 31 October, and for G77, Q86, and M86 on 8 November. One of these four animals (B82) died 15 days after antibiotic treatment began, but the other three recovered after 3 to 4 weeks of therapy. Of the eight bighorns remaining, four developed mild rhinitis characterized by serous nasal discharge, and four showed no signs of respiratory disease (Table 1).

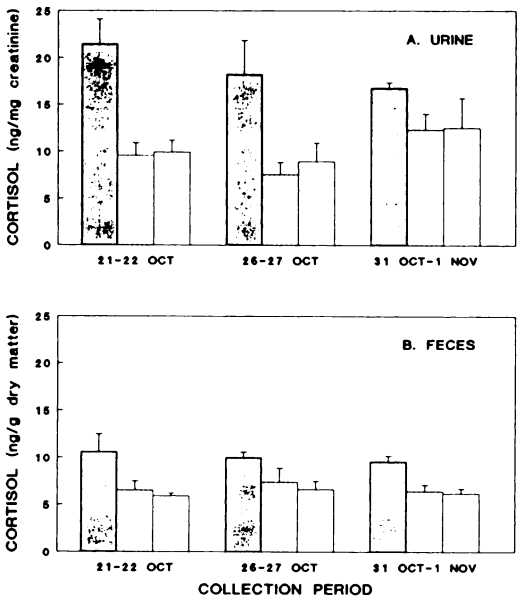


FIGURE 1. Three bighorns that developed clinical pneumonia (heavily shaded bars) showed elevated mean (A) urinary ($P = 0.003$) and (B) fecal ($P = 0.046$) cortisol excretion over the 12-day sampling period. Mean cortisol excretion in mildly affected (lightly shaded bars) and healthy (open bars) bighorns appeared indistinguishable. Bars represent means of 48 hr collections for each clinical grouping; vertical lines are +1 standard error.

Diagnostic findings

Beta-hemolytic *P. haemolytica* biotype T was isolated from nasal and pharyngeal swabs of all eight bighorns with pneumonia or mild rhinitis (Table 1); these isolates included both serotypes 3 and 4. We observed no apparent relationship between serotype and clinical condition (Table 1). Swabs from the other four bighorns yielded no significant bacterial growth. All serologic tests for PI3 and BRSV were negative.

Based on gross and histologic lesions, we diagnosed suppurative bronchopneumonia accompanied by lymphoid depletion, mild hepatic lipidosis, and mild chronic interstitial nephritis in B82. *Escherichia coli* and *Bacillus* sp. were cultured from a tonsillar swab, lung, and suprapharyngeal lymph node. In addition, *Actinomyces pyogenes* and an untyped *P. haemolytica* were recovered from suprapharyngeal lymph node. Fluorescent antibody tests for PI3, BRSV, and *C. psittaci* were negative.

Urine and fecal cortisol excretion

Only three of four affected bighorns (G77, Q86, M86) were sampled in all three collection periods, and their data were used in subsequent analyses. Bighorns that ultimately developed pneumonia showed elevated mean urinary ($P = 0.003$) and fecal ($P = 0.046$) cortisol excretion over the 12-day sampling period (Fig. 1). Differences among animals grouped by clinical condition were not influenced by collection period ($P \geq 0.28$) or interactions of clinical condition and collection period ($P \geq 0.49$). Mean cortisol levels in excreta from mildly affected and healthy bighorns appeared indistinguishable (Fig. 1). Twenty-four hour mean urine cortisol : creatinine ratios ranged from 10 to 57 ng/mg for affected and 5 to 22 ng/mg for healthy individuals; 24-hr mean fecal cortisol concentrations ranged from 7.2 to 20 ng/g dry matter for affected and 3.6 to 9.1 ng/g dry matter for healthy individuals. Elevated cortisol excretion was detected up to 16 days before clinical pneumonia developed.

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DISCUSSION

The pasteurellosis outbreak described here shares features of pneumonia epizootics in free-ranging bighorns, and may provide insights into epizootiology of the bighorn pneumonia complex. Our data reveal that elevated cortisol excretion can precede pneumonia in captive bighorns. Cortisol concentrations in urine and feces from the four sheep that developed pneumonia were elevated when sampling began, demonstrating that a rise in cortisol excretion may be detected in some bighorns at least 16 days before pneumonia develops. Such as association could prove useful to managers in warning of impending disease outbreaks in captive or wild bighorns, and warrants further evaluation.

Despite this association, our findings do not necessarily demonstrate a causal relationship between cortisol elevation and pneumonia in bighorns. Data collected during another of our experiments seem to contradict traditional views of that relationship. Treating bighorns with ACTH elevated urine cortisol excretion to levels 4 to 5 times those of controls (and more than twice levels of bighorns that developed pneumonia here) (Miller et al., 1991). Corticotropin-treated sheep failed to develop pneumonia, even though some were shedding *Pasteurella* spp. in nasal secretions (Miller, 1988). Reduced lymphocyte blastogenesis responses in ACTH-treated bighorns indicated immunosuppression may have occurred, but was apparently insufficient to predispose those animals to pneumonia (Miller, 1988). However, bighorns that developed pneumonia here had lymphocyte responses indistinguishable from those of unaffected animals (M. W. Miller, unpubl. data), suggesting in vitro blastogenesis responses may be inappropriate indices of in vivo susceptibility to disease in bighorns. In light of these conflicting observations, we can conclude only that cortisol excretion may be elevated before pneumonia develops in bighorns. Whether this represents predisposing stress or simply physiological responses during subclinical incubation remains undetermined.

Examined alone, cortisol data implicate stress as a dominant predisposing factor in the epizootic described here. However, such an interpretation seems equivocal when other epizootiological observations are considered. Age and/or reproductive physiology may have influenced relative susceptibility of lambs and castrated rams to pneumonia during this outbreak. Bighorn lambs appear particularly vulnerable to pasteurellosis: all lambs born into our research herd between 1984 and 1986 developed pneumonia before six months of age. Similar patterns have been described in wild bighorn populations (Demarchi, 1972; Spraker and Hibler, 1982; Bailey, 1986). In addition, two *P. haemolytica* serotypes (3 and 4) were recovered from nasal swabs of affected individuals. Although each strain's relative significance in causing pneumonia could not be discerned here, presence and/or transmission of one or both may have contributed to the outbreak.

Preexisting immunity to *P. haemolytica* may also have influenced the pattern of this epizootic. The four bighorns that developed pneumonia were among seven previously unaffected by pasteurellosis; all five sheep that had recovered from pneumonia in 1984 or 1985 remained healthy or were only mildly affected during this outbreak (Table 1). Protective immunity against *P. haemolytica* challenge can develop after pneumonic pasteurellosis in domestic sheep (Donachie et al., 1986) and cattle (Confer et al., 1984; Cho and Jericho, 1986; Confer, 1988). Similar responses have not been described in bighorns, but our observations suggest earlier infections may have conferred protection to some individuals that resisted pneumonia during this outbreak.

Differential susceptibility to pasteurellosis probably exists among and within wild bighorn populations. We suggest two general processes operate to create conditions favoring pneumonia outbreaks in bighorn

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populations; we view both as ultimately affecting overall susceptibility of a bighorn population to a specific pathogen. In the first process, differential susceptibility among bighorn populations may contribute to epizootics. Novel *Pasteurella* spp. strains may be introduced into a bighorn herd by an immigrating carrier. This may partly explain how an epizootic spread among southwest Canada's bighorn populations during 1981 to 1983 (Onderka and Wishart, 1984, 1988). Introduced strains of *Pasteurella* spp. have also been incriminated in some pneumonia outbreaks in bighorns following their association with domestic sheep (Goodson, 1982; Foreyt and Jessup, 1982; Coggins, 1988; Onderka and Wishart, 1988; Foreyt, 1989). We infer from experimental data (Onderka and Wishart, 1988; Onderka et al., 1988; Foreyt, 1989) that bighorns are extremely susceptible to some strains of *Pasteurella* spp. carried by domestic sheep. It follows that epizootics caused by novel *Pasteurella* spp. strains can initially operate in a random, density-independent manner because a large proportion of a bighorn population may be susceptible at any time.

In the second process, the proportion of susceptible animals within a population may increase through recruitment of immunologically naive individuals (via birth or immigration) and/or through individuals losing existing immunity (via stress-mediated immunosuppression, temporal waning, or some other process). Density-dependent variability in social, nutritional, reproductive, and/or immunological status could affect overall susceptibility (Anderson, 1979; May and Anderson 1979). Several pneumonia epizootics have followed rapid increases in bighorn numbers that could have contributed to the subsequent decline (Feuerstein et al., 1980; Onderka and Wishart, 1984; Bailey, 1986; Coggins, 1988; Festa-Bianchet, 1988; CDOW, unpubl. data). These increases occurred largely through improved lamb recruitment that produced young and presumably naive populations in a relative short time. We believe that pneumonia outbreaks occurring under these conditions can arise from *Pasteurella* spp. infections carried by some individuals in affected herds, and that in these cases pasteurellosis operates in a cyclic and perhaps density-dependent fashion. We regard our 1986 epizootic as an outbreak of preexisting pasteurellosis.

We believe that periodic nature of pneumonia epizootics in some bighorn herds may reflect gradual accumulation of susceptible sheep in these populations, rather than episodic bouts of widespread stress responses. It seems likely that some critical proportion of susceptible individuals and *Pasteurella* spp. carriers may be required to propagate pneumonia epizootics in those bighorn herds. Relative pathogenicity of *Pasteurella* spp. strains may affect that proportionality. Demographic and/or environmental stochasticity may influence the periodicity of outbreaks (Anderson, 1982; May, 1986). We do not discount stress completely in defining circumstances favoring an outbreak: at higher ecological densities, more individuals may suffer effects of cumulative social and environmental stressors, and subsequent stress responses could lead to pathogen shedding and/or to increased susceptibility. However, we view environmental stress as one of several possible, and perhaps simultaneous, mechanisms operating on bighorn populations to increase overall susceptibility to pasteurellosis and other diseases.

Developing reliable criteria for assessing population susceptibility to pasteurellosis, including serologic tests and refined epizootiological techniques for identifying strains of *Pasteurella* spp., could enhance management efforts to predict and prevent pneumonia outbreaks in bighorns. Measures of stress responses could be included in these criteria, but monitoring only "stress levels" may fail to predict pneumonia epizootics in bighorns. Similarly, management strategies targeting only "stress reduction" may be insufficient to

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prevent these outbreaks. When viewed in terms of their effects on population susceptibility, some management practices (including baiting associated with anthelmintic treatment and relocation efforts) may actually promote pneumonia outbreaks by concentrating sheep or by introducing carriers into susceptible populations (Sandoval et al., 1987). Until tools allowing biologists to assess population susceptibility to pasteurellosis become available, both management experiences (Wishart, 1970; Thorne et al., 1979; Jorgenson and Wishart, 1986; Festa-Bianchet, 1988) and simulation analyses (Hudson and Stelfox, 1976; Hobbs et al., 1990) suggest a comprehensive combination of habitat management and population control that maintains herds at low ecological densities may prove most effective in minimizing herd susceptibility and preventing some pneumonia epizootics in bighorns.

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