EVALUATION OF HEALTH STATUS OF ROCKY MOUNTAIN SHEEP

(OVIS CANADENSIS CANADENSIS)

IN SOUTHEASTERN BRITISH COLUMBIA

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by

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selenium levels and pathological findings. Recommendations were
made for future monitoring and management of hsemoparasite larval
levels. The Columbia Lake herd was treated with trace minerals and an
anthelmintic after the 1973 collection. Longhorn larvae output
was less in four sheep examined in the following year, but no
change was seen in trace element levels.
ABSTRACT

Three herds of Rocky Mountain bighorn sheep (Ovis canadensis canadensis Shaw) in southeastern British Columbia were evaluated for health status during 1983 and 1984. Each herd was of similar size but varied in disease occurrence and herd dynamics, both historically and over the period of study. Samples taken from six sheep in each herd were examined for nutritional condition, microbiological, virological and serological status, general and pulmonary parasite loads, blood chemistry and trace mineral levels, as well as gross and histological lesions. The Columbia Lake herd at high animal density wintering on a poor quality, low elevation range was demonstrated to have high levels of lungworm infection, low total serum protein, fecal nitrogen and liver selenium levels. Higher total serum protein, fecal nitrogen, liver selenium levels and lower lungworm levels were present in bighorns from the lower density ER herd wintering at high elevation. Adrenal glands were larger, lungworm levels were lowest, and chronic clinical and subclinical systemic and respiratory diseases were common in adults and lambs from a second herd (Wigwam) wintering at low elevation, 2 years after an all-age dieoff. Study herd status was best determined by comparison of fecal lungworm larval output, fecal nitrogen, liver selenium levels and pathological findings. Recommendations were made for future monitoring and management of bighorn herds. The Columbia Lake herd was treated with trace minerals and an anthelmintic after the 1983 collection. Lungworm larvae output was less in four sheep examined in the following year, but no change was seen in trace mineral levels.
ACKNOWLEDGEMENTS

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1.0 INTRODUCTION

The Rocky Mountain bighorn sheep (*Ovis canadensis canadensis* Shaw) populations of North America have markedly decreased in number and distribution since the human settlement of their native habitat (Buechner 1960). The factors responsible for the decline are suggested to include heavy hunting pressures, the introduction of domestic animal diseases and, especially in this century, the widespread reduction of available and suitable ranges (Buechner 1960, Stelfox 1971).

More recently, bighorn sheep have suffered epizootic dieoffs in captive and wild situations in Canada and the U.S.A. The sheep have nearly always succumbed to pneumonia caused by opportunistic bacteria with varying degrees of lungworm involvement (Forrester 1971). Reports of sheep dieoffs have outlined a large number of environmental and animal-related factors which have been present before dieoffs and were believed to predispose sheep to disease. For example, Spraker et al. (1984) described a series of conditions preceding a dieoff at Waterton Canyon in Colorado. These included increased levels of vehicular traffic, noise, human harassment, high animal density and a reduction in forage quality and quantity. Reports of other dieoffs have included factors such as low trace mineral levels in tissue, inclement weather, habitat deterioration and loss, the presence of domestic sheep, concurrent infectious diseases and high lungworm or other parasite levels (Buechner 1960, Lange et al. 1980, Foreyt and Jessup 1982, Thorne et al. 1982, Schwantje 1983, Onderka and Wishart 1984).
Such factors are believed to function through direct and indirect mechanisms, their presence and effect varying with each situation. Chronic exposure to some of these conditions may act cumulatively to impair protective immune functions. The collective influence is believed to create a state of chronic stress (Spraker et al. 1984). The physiological responses of individual animals to chronic stress include adrenal gland enlargement and increased levels of adrenal corticosteroids (Feldman 1983). Long term maintenance of high levels of corticosteroids in other species is associated with an inhibition of immune responses and increased susceptibility to bacterial infections, particularly of the respiratory tract (Hunningshake and Fauci 1977, Kelley 1980). Studies in man and domestic animals have shown that concurrent viral respiratory infections (Yates 1982), malnutrition (Scrimshaw et al. 1968) and low tissue levels of selenium and copper (Chandra 1983) are associated with, and can directly cause depressed immune responses.

The proper function of immune systems is closely related to the overall nutrition of an animal (Chandra and Newberne 1977). Many of the factors discussed as influencing bighorn sheep herds play important roles in their nutritional condition. Forage quality and quantity have obvious direct influences. Other factors, such as intra and interspecific competition, harassment and high gastrointestinal parasite burdens may indirectly reduce the amount and type of feed consumed and utilized. Indices of nutritional status have been examined in controlled (Kirkpatrick et al. 1975, Seal et al. 1978, Bahnak et al. 1979, Warren et al.
1982) and studies of free-ranging white-tailed deer (*Odocoileus virginianus*), pronghorn antelope (*Antilocapra americana*) and wild sheep (Franzmann 1972, White and Cook 1974, Seal and Hoskinson 1978, Hebert et al. 1984, Spraker et al. 1984). The levels of total serum protein (TP), blood urea nitrogen (BUN), fecal nitrogen (FN) and body condition scores varied with season, habitat and physical condition in white-tailed deer and bighorns (Franzmann 1972, Seal et al. 1978, Hebert et al. 1984). These parameters were the most frequently used in these studies and were easily available from live animals.

Depressed TP has long been accepted as occurring with protein-energy malnutrition in man (Sauberlich 1983), and studies have demonstrated reductions in TP with starvation in wildlife species (Hebert 1973, Bahnak et al. 1979, Warren et al. 1982). Blood urea nitrogen levels were proportional to protein consumption in cattle (Biddle and Evans 1973), domestic sheep (Preston et al. 1965), white-tailed deer (Seal et al. 1972, Kirkpatrick et al. 1975) and pronghorn antelope (Seal and Hoskinson 1978). Franzmann (1972) stated that the nutritional status of bighorn sheep was best estimated by BUN and suggested that values below 15 mmol/L indicated low protein intakes.

Comparisons of fecal nitrogen content with feed nitrogen content have been made to assess the protein status of wildlife populations. Species examined include free-ranging elk (*Cervus elaphus*) (Gates and Hudson 1979), East African ungulates (Arman et al. 1975) and wild and captive bighorn sheep (Hebert 1973, Hebert et al. 1984). In most cases, studies have followed
seasonal trends in FN and related these to environmental changes (Seip and Bunnell 1985), feed quality and animal weight (Mould and Robbins 1981). Hebert et al. (1984) showed changes in animal condition between seasons and years in captive, supplementary fed, free-ranging and non-migratory free-ranging California bighorn sheep (Ovis c. californiana). He advised the use of FN determination over time to assess the effect of changes in population density and grazing on sheep ranges and herds. Bahnak et al. (1979) suggested that periodic monitoring of all nutritional indices was more valuable than conclusions drawn from samples taken at a single point in time.

The definition of trace mineral deficiencies requires the association of clinical signs of deficiency with low mineral concentrations in animal tissues, diet and soil. Deficiency syndromes are often subclinical and may be difficult to identify due to interactions and imbalances between individual minerals and the protein-energy content of the diet. Studies related to two of the more commonly recognized deficiencies, selenium and copper, are limited in free-ranging wild species; however, both minerals have been extensively reviewed for domestic cattle and sheep (Hidiroglou 1979, Underwood 1977, Van Vleet 1980).

Subtle or marked syndromes of muscle degeneration, ill thrift, neonatal weakness and infertility are responsive to the addition of selenium and vitamin E (Se/E) to the diets of ruminants (MacDonald et al. 1976, Maas 1983). In addition, suppressed cell mediated immune functions are restored (Sheffy and Schultz 1979). An acute stress-induced muscle degeneration
(capture myopathy) has been well recognized in wild ungulates and is associated with Se/E deficiency in some instances (Hebert and Cowan 1971). The other livestock syndromes are not well defined in wild species but are suspected to occur.

Copper deficiency in domestic sheep is also often subclinical but can reduce growth rate, reproductive performance and haircoat pigmentation (Ward 1978, Hidiroglou 1979). Studies in Alaskan moose (Alces alces) and Idaho mule deer (Odocoileus hemionus) suggested that these were reductions in fertility and abnormal hoof keratinization (Flynn et al. 1977, Dunbar and Foreyt 1985). Low copper levels are also associated with decreased immune responses and an increased susceptibility to parasitism (Chandra 1983). A possibility of interaction between copper and selenium has been proposed because of a lack of response to supplementation until both elements were provided to domestic livestock (Blood et al. 1983).

In this thesis I review the history and causes of disease and mortality in the wild sheep of North America. The principle of synergism of infectious agents and environmental factors in domestic animal pneumonia is outlined and associated with a similar pathogenesis of the respiratory disease complex of Rocky Mountain bighorn sheep. The project which surveys and compares sheep from three bighorn herds is described and the results discussed in terms of the assessment of bighorn sheep herd health status and possible remedial management actions.
2.0 LITERATURE REVIEW

2.1 BIGHORN SHEEP DIEOFFS

2.1.1 North America

The most widely distributed wild sheep species in North America is the Rocky Mountain bighorn sheep. Estimates of bighorn distribution and numbers prior to the settlement of western North America are few, but sheep were reported to be present over wide areas of what is now western Canada and the United States. Counts of up to 2 million bighorn sheep for the U.S.A., Canada and Mexico prior to 1800 have been suggested (Buechner 1960). This absolute figure is probably of little value due to inaccurate census methods, but does serve to contrast dramatically with counts of less than 20,000 bighorns in the U.S.A. in 1960 (Buechner 1960).

Bighorn population declines occurred as human-related activity was introduced into accessible bighorn habitat. Buechner (1960) attributes the majority of sheep losses in the last half of the nineteenth century to several factors acting singly or in combination. These included excessive hunting with firearms, disease epizootics, competition from livestock and restriction of winter range.

Legislation to preserve sheep habitat, reduce native predators and to prevent hunting was passed by wildlife agencies in the late 1800s and early 1900s. These actions, as well as reintroductions of sheep into empty U.S. ranges in the 1950s and
attempts at habitat restoration, were successful in slowing declines and increasing numbers in some areas. Many populations, however, continued to decline and remained as small remnant herds or vanished completely. Although specific causes were never determined, the disappearance of some herds coincided with the intensification of use of bighorn habitat for livestock grazing, recreational, urban and industrial development. It is believed that another major factor in the loss of sheep habitat was the effective prevention and control of fires (Demarchi 1972).

Grassland ranges previously maintained by periodic natural fires underwent successional growth by conifer trees or were allowed to overgrow with nonforage plant species. These processes combined to greatly reduce available range.

Certain diseases were noted in the sheep population as a whole and were believed to be responsible for at least some of the initial losses of bighorn sheep. Reports of bighorn mortality occurring in large scale epizootics were referred to as dieoffs. The earliest dieoffs were attributed to scabies mange caused by *Psoroptes ovis*. This external parasite was considered to have invaded bighorn ranges with domestic sheep herds (Buechner 1960). Later dieoffs of bighorns in the U.S.A. were reported to have a common disease pattern, almost exclusively of fatal acute and chronic pneumonia, caused by opportunistic bacteria such as *Pasteurella* spp. A lungworm, *Protostrongylus* spp., unique to mountain sheep, was involved in most cases. This bacterial-lungworm syndrome became known as the lungworm-pneumonia complex of bighorn sheep (Forrester 1971), but
is perhaps more accurately referred to as the respiratory disease complex of bighorn sheep (Onderka and Wishart 1984).

The serious influences of disease dieoffs on bighorn herd production have stimulated much interest in the species. Research, particularly in the past 10-20 years, has associated a wide range of animal and environmental conditions with outbreaks of pneumonia. The combination and overall importance of these factors has varied with each herd situation.

General types of dieoffs have been described. In addition to the classic "all-age" dieoffs with mortality initially seen in juvenile and adult sheep in good body condition, there are lamb dieoffs classified as summer lamb pneumonia and stress-induced lamb pneumonia (Spraker 1979). Other, primarily adult dieoffs, have been described on the basis of the prominence of specific factors such as severe lungworm infections (Spraker 1977), varying degrees of malnutrition, human harassment (Spraker et al 1984), interspecific competition and adverse weather conditions. These dieoff classifications will be discussed in detail.

2.1.2 Canada/East Kootenays

Canadian populations of Rocky Mountain bighorn sheep are concentrated in the Rocky Mountain ranges on both sides of the British Columbia-Alberta border. The development of this area for human use has, to a large extent, been restricted in comparison to the mountainous areas of the U.S.A. In fact, much of the area occupied by bighorns is now protected under National and Provincial park status. Bighorns within park boundaries are
not hunted and their ranges are protected from most human-related encroachment, as well as from natural environmental changes such as fires. Sheep living outside of the park systems for most of the time are currently managed in accordance with provincial big game hunting regulations. Provincial management practices have also focussed on range protection and improvement, mostly by manipulation of critical fall and winter ranges.

The human-related pressures responsible for declines in bighorn numbers in the U.S.A. have also caused reductions in overall numbers of Canadian bighorns. The population declined from 10,000 on the eastern slope of the Rockies in 1850, to 2600 in 1915 (Bandy 1968, Stelfox 1971). Stelfox (1971) reviewed the dynamics of Rocky Mountain bighorns in this area and demonstrated a markedly fluctuating pattern. During the period 1915-1936, the depleted population increased to 8500. The area had been completely closed to hunting and this was strongly enforced. Livestock grazing was eliminated or reduced from mountain grasslands. Other game, such as bison (Bison bison) and elk were not particularly abundant and it is thought that extensive fires in the late 1800s and early 1900s expanded grasslands on bighorn ranges.

All major park bighorn populations increased but these increases were followed by rapid declines. In some instances 75% or more of the population died, with many of the declines shown to be due to lungworm-related pneumonia. By 1950, the total population of bighorn sheep had again reached 2500 (Stelfox 1971). Although no sequential pattern was noted, Stelfox (1971)
outlined similar high densities, heavy interspecific forage competition resulting in range deterioration, severe winter weather and heavy lungworm levels prior to dieoffs in affected herds. With mild weather conditions and the reduction of grazing pressure on ranges after the dieoffs, sheep numbers increased once again to 10,000 by 1970 (Stelfox 1971).

The East Kootenay (E.K.) region of British Columbia contains 85% of the Rocky Mountain bighorn sheep in the province, and consists of from 1500 to 2000 animals. The population can be subdivided on the basis of winter range location into at least 20 herds. Twelve of these herds winter on low elevation, southeastern facing slopes on or near the eastern side of the Rocky Mountain Trench. An additional eight or more herds winter at high elevations surrounding the upper Elk River valley (Fig. 1).

The sheep in the E.K. have experienced large scale all-age dieoffs in the 1920s, 1940s, 1960s and 1980s. Although detailed records are not available for each dieoff, local interest preserved some facts. Certain common circumstances have been associated with the mortality.

The herds reported to have undergone dieoffs were those that were relatively accessible due to their habitual use of low elevation winter ranges in or adjacent to the Rocky Mountain Trench. Little information has been available on the status and mortality patterns of the herds wintering at high elevation. The inaccessibility of many of the ranges has prevented detailed observations; however, the area has been utilized frequently for
hunting and has been extensively explored by mining interests. None of these sources have reported sick or dead bighorn sheep in large numbers. More intensive aerial surveys and biological studies in the area since 1971 have shown no radical changes in the high elevation herds (Warkentin pers. commun., Schuerholz 1984).

The affected herds usually had population growth patterns which reached a population density considered to be high by biologists working in the area just prior to disease outbreaks. There was an interval of 16 to 25 years between dieoffs. In almost all cases the animals that were found dead or seen ill were of mixed age and sex. Those seen ill had obvious clinical signs of respiratory disease. The initial deaths in each outbreak were peracute and usually occurred during the fall or winter, although animals with chronic pneumonia were found in later stages of the dieoffs. Postmortem examinations performed on some of these sheep (Bandy 1966, Davidson 1982) confirmed diagnoses of bacterial pneumonias complicated by mild to severe lungworm infections. Gastrointestinal parasites and malnutrition were also present in various degrees. In addition, the dieoffs followed contact of bighorn sheep with domestic sheep in 1942, 1965 and 1981. No further investigations were attempted to investigate the presence of respiratory diseases in the domestic sheep herds. Heavy snowfalls and wet, early summers, which provided excellent conditions for snail intermediate hosts of Protostrongylus spp., preceded dieoffs in 1964 and 1981.

Finally, the dieoffs in the E.K. were rarely confined to one herd
but tended to occur sequentially through many of the herds. With better census techniques and observations during the 1960’s and 1980’s, the multiple herd nature of dieoffs in the E.K. became more obvious.

The first bighorn dieoff in the E.K. to be investigated intensively began in December 1964 at the Bull River winter range in the Rocky Mountain Trench. Strong winds, a heavy snowfall with a thick crust and poor winter range as a result of heavy big game and livestock grazing pressure limited feed intake and quality. The sheep were allowed free access to alfalfa hay on a neighbouring farm. Rumen impaction with the unnatural feed resulted. During the winter over 95% of the herd died of acute or chronic bacterial pneumonia complicated by heavy lungworm infections (Bandy 1966, Demarchi 1972). Similar respiratory disease reduced the Wigwam herd by 38-50%, Premier Ridge herd by 72%, Columbia Lake herd by 73% and Stoddart Creek herd by 76% over the next three years (Bandy 1968). Other adjacent herds were also affected. The total number of bighorns in the E.K. dropped to 720 from a high of at least 1650 in the early 1960s (Demarchi 1972).

Recovery efforts over the next decade and a half were concentrated on aggressive habitat management, acquisition and protection programs, as well as the reduction of elk numbers in strategic areas such as the Wigwam winter range. Sheep numbers responded, climbing to pre-1964 numbers in most areas. Previous sheep and other big game density and disease patterns suggested the development of a dieoff in the Wigwam...
range in the 1980s. The situation was expected to be critical by 1985, twenty years after the last dieoff. However, the bighorn sheep in this herd increased more rapidly than expected, reaching approximately 424 (Davidson pers. commun.) by the spring of 1981. Maximum counts of only 174 (Bandy 1968) proceeded the 1960s dieoff. During the late fall of 1980, lambs on the Wigwam range were observed with oral lesions of contagious ecthyma. Two-thirds of the lambs subsequently disappeared over the winter months. In the second week of December 1980, animals in good condition began dying acutely on the adjacent Maguire Creek range. The Maguire Creek bighorn sheep had shared this good quality range with domestic sheep during the fall of 1980. All of the carcasses examined had lesions of bacterial pneumonia with prominent lungworm infections.

Sixteen sheep from the Wigwam range were captured live, given prophylactic antibiotics and a vitamin complex and released at Bull River in March 1982. Eight of these sheep died within 6 weeks with respiratory disease similar to subsequent deaths on the Wigwam range. None of the resident Bull River herd became ill in spite of thoroughly mixing with the transplants.

In the following two months, at least 46% of the Wigwam herd died with acute to chronic pneumonia, many having light lungworm infections (Davidson 1982). Serum and liver selenium and copper levels were interpreted to indicate deficiencies of these elements in some sheep. Over 80% of 85 carcasses that were aged and sexed were between 4 and 7 years of age. A large proportion of the losses in this herd then consisted of the prime
reproductive age class. Chronically affected sheep were occasionally present on the range in the following years. Lamb production in the surviving sheep has been extremely poor with small size and high mortality observed in lambs less than 6 months of age.

Over the next 2 years there were reports of thin and/or coughing sheep at Premier Ridge, Wildhorse and Lussier ranges. In the opinion of the local biologists, the number of bighorn sheep declined in these and small associated herds. Several carcasses examined had "classic" lesions of the bighorn respiratory disease complex (R. Lewis pers. commun.).

The occurrence of disease in bighorn sheep in the E.K. has twice shown a progressive northward movement in the Rocky Mountain Trench wintering herds. In 1982–83 similar mortality was also seen in southwestern Alberta herds (Onderka and Wishart 1984). It can be suggested that contact between individuals of different herds can occur on common summer ranges or when wandering ram bands mix prior to or during the rut. Festa-Bianchet (1986) demonstrated that different ewe groups can share the same ranges at different times of year. This may result in another source of increased contact between groups of bighorn sheep. Mortality seen in adult sheep in the early fall may support the concept of transmission of types or numbers of pathogens between herds in summer months. It seems most likely that the pattern seen in the E.K. has a direct relationship to the large number of contiguous herds and the transmission of large numbers of pathogenic organisms between herds of differing susceptibility.
2.1.3 Dieoff Types

As previously discussed, the mortality of free-ranging and captive bighorn sheep has demonstrated a remarkable epizootic pattern. Dieoffs have been associated with Psoroptes mange infections (Buechner 1960, Lange et al. 1980) and pneumonia (Marsh 1938, Spraker 1977, Spraker et al. 1984, Onderka and Wishart 1984).

Spraker and Hibler (1982) discussed the respiratory diseases of bighorn sheep and classified pneumonia mortality into three clinical types. The types are subdivided primarily on the basis of age with secondary criteria of seasonal occurrence, pathological characteristics and predisposing conditions.

The first type of pneumonia dieoff has been recognized since the 1800s and is commonly known as the "all-age dieoff". In this form sheep of any age may be affected. The outbreak may occur in any season, but usually begins in fall on winter range and is associated with multiple environmental and density linked stress factors, including various degrees of lungworm infection (Spraker et al. 1984). Many animals die acutely in good physical condition with fibrinous bronchopneumonia, but emaciated sheep with chronic lesions may die subsequently after protracted illness. All-age dieoffs in captive bighorn sheep usually follow a similar pattern.

The organisms recovered from pneumonic lesions in this type of dieoff are generally considered to be opportunistic invaders. It is felt that a series of environmental and physiological events, which may vary among dieoffs, cause a state of chronic
stress in the herd. This type of chronic stress causes adrenal cortical hypertrophy and hypersecretion of adrenal corticosteroids (Feldman 1983). Immunological responses are reduced, resulting in an increased susceptibility to pathogenic organisms (Harlow 1983). Factors such as increased human activity, vehicular traffic, noise, dust, reduced forage quality and quantity, inclement weather, high inter- and intraspecific competition and high levels of parasitism have been suggested as contributing to all-age dieoffs (Buechner 1960, Bandy 1966, Schwantje 1983, Onderka and Wishart 1984, Spraker et al. 1984).

Other types of dieoff have been recognized as improved census and monitoring techniques, and have allowed the evaluation of the production of bighorn lambs and their survival to yearling status in some herds. Woodard et al. (1974) initiated a study in 1969 to investigate the declining Sangre de Cristo herd in Colorado. Over the two years of study they concentrated their efforts on the ewe-lamb segment of the herd and found that although ewe-lamb ratios were high in early July (72-83%), they were reduced to 17-22% by mid September. No adult mortality was seen. The authors observed progressive symptoms of respiratory disease in lambs throughout the late summer in both years. The lambs were lethargic, weak, and had paroxysmal coughing and dyspnea. Necropsies performed on two lambs with these clinical signs showed large numbers of first stage Protostrongylus spp. larvae and secondary bronchopneumonia. Bacteria of several species, including Pasteurella spp. were isolated. The mortality was also associated with periods of cold, wet weather (Woodard et
Descriptions of sick lambs during the summer have also been reported by Marsh (1938), Honess (1942) and Buechner (1960) in numerous locations. Spraker (1977, 1979) separated the syndrome into two types on the basis of the presence of heavy lungworm infections.

The first of these has been termed "summer lamb pneumonia" and appears to occur following transplacental transmission of large numbers of larvae of *P. stilesi* from the ewe to the lamb in utero (Hibler et al. 1974, Spraker 1979). The parasite matures in the lungs and produces larvae by the time the lambs are 1 month old. Lung tissues damaged by the first stage larvae are secondarily invaded by an assortment of opportunistic pathogens. Acute pneumonia is believed to be precipitated by additional stressors, such as inclement weather, but chronic pneumonia is common. Hence the affected lambs may survive for variable lengths of time.

The second form of pneumonia seen in lambs is characterized by little to no lungworm involvement and has been classified as "stress-induced lamb pneumonia" (Spraker 1979). All of the herds involved suffered all-age dieoffs prior to the dieoff of lambs. Affected lambs born after the dieoffs had lesions of bacterial bronchopneumonia and the thymus was markedly reduced in size. Chronic stress is believed to be responsible for reducing the immune system functions of these lambs at an early age. Spraker et al. (1984) suggested that the stresses which precipitated the all-age dieoff in the adult sheep may affect the pregnant ewe and inhibit the development of the fetal thymus. In addition, the
production of colostral antibodies may be diminished. Lambs would then be poorly prepared to encounter even normal microflora. Ewes suffering from chronic pneumonia may shed large numbers of bacteria which could overwhelm the immature defenses of lambs born with abnormal or normal thymic function.

2.2 BIGHORN MORTALITY - CAUSES

As previously discussed, the clinical disease and mortality of bighorn sheep has been most frequently caused by respiratory diseases (Buechner 1960, Forrester 1971, Spraker 1979). Fatal pneumonias usually include gross and microbiological evidence of bacterial and lungworm etiologies, but viruses and other organisms, such as mycoplasma and chlamydia, have also been recovered in some disease outbreaks (Pearson and England 1979, Woolf et al. 1970). Because bighorn sheep are susceptible to many diseases of domestic livestock, domestic animal pathogens are among those agents implicated in the decline of bighorn sheep populations (Foreyt and Jessup 1982, Goodson 1982). Dieoffs have frequently been associated with contact between these species and there apparently have been transfers of infection following interspecific association.

2.2.1 Viral

In recent years, increased awareness of synergism between infectious organisms in diseases of domestic species has given impetus to the search for multiple agents that might predispose bighorns to respiratory disease complex outbreaks. Serological surveys have indicated the exposure of many North American
populations of wild sheep to various respiratory and systemic viral diseases of wildlife and domestic animals.

Antibody titres to the respiratory viruses, parainfluenza virus type 3 (PI3), infectious bovine rhinotracheitis (IBR), bovine respiratory syncytial virus (RSV), as well as to bovine parvovirus 1 and adenoviruses have been present in all North American wild sheep species examined. Titres to PI3 were considered by the authors to be significant in Wyoming and Montana Rocky Mountain bighorns (Howe et al. 1966). Antibodies to PI3, bovine parvovirus 1 and adenovirus were present in a large proportion of bighorn samples from Colorado and Wyoming (Parks and England 1974). Desert sheep (*Ovis c. nelsoni*) herds in California frequently had high numbers of animals seropositive for PI3 (Clark et al. 1985), while only a few positive animals have been found among Dall sheep (*Ovis dalli dalli*) (Zarnke 1983).

Parainfluenza 3 virus has been isolated from both healthy and clinically ill free-ranging and captive sheep herds. Parks et al. (1972) reported an outbreak with mortality involving secondary bacterial pneumonia in Rocky Mountain bighorn sheep recently placed in captivity. The pattern of disease in these sheep suggested that previously exposed animals kept in close confinement infected others which lacked previous experience with the virus. Parainfluenza 3 virus was also isolated from free-ranging desert bighorn lambs with and without pneumonia (Jessup 1985). This virus is believed to be a contributing factor in the mortality of desert bighorn lambs in California (De Forge et al. 1982).
Parainfluenza 3 virus alone can cause acute pulmonary infections, but the uncomplicated infection in cattle and sheep rarely causes mortality (Dungworth 1985). Experimental studies have shown that the degree of lung damage is proportional to the virulence and dose of the virus and the resistance of the animal (Yates et al. 1983a). It appears that the most important role of the virus is to reduce pulmonary defenses, allowing the invasion of bacteria and the development of bronchopneumonia.

Little definitive information is available on bovine parvoviruses except that they are ubiquitous in cattle and appear to be most common in multifactorial diarrhea outbreaks in young calves (Barker and Van Dreumel 1985). Adenoviral infections are primarily associated with ocular or respiratory diseases and enteritis. Exposure to the virus is widespread in cattle populations but clinical disease is sporadic except in immunocompromised individuals. The significance of titres to either virus in bighorn sheep is unknown.

Clark et al. (1985) found widespread evidence of exposure of bighorn sheep to RSV throughout American sheep ranges. This virus was isolated concurrently with Pasteurella haemolytica type T from a single lamb with pneumonia in Colorado (Spraker et al. 1986). The significance of this disease in bighorns is unknown at this time but, with the identification of this case, it seems very likely that it may play a role similar to that in domestic cattle and sheep. There is evidence of its involvement in the bovine shipping fever complex, but it is also suspected to cause outbreaks and mortality as a single infection, perhaps more often
than is the case with pure PI3 infections (Trigo et al. 1984, Lehmkuhl and Cutlip 1979).

Other ruminant viruses can also cause systemic disease with symptoms of respiratory infection. Surveys for antibodies against some of these, such as IBR, bovine virus diarrhea (BVD), bluetongue (BT), contagious ecthyma (CE) and epizootic hemorrhagic disease (EHD) have suggested occasional exposure of bighorn sheep (Dunbar et al. 1985).

Bluetongue and EHD are arthropod-borne viral diseases of warm temperate areas which affect domestic and wild ruminants. Bluetongue has a wide range of native wild hosts. Clinical disease is most severe in sheep and wild species. Inapparent infections do occur and are particularly frequent in cattle. Epizootic hemorrhagic disease has been seen most commonly in white-tailed deer (Kistner et al. 1975).

Bluetongue virus infection was demonstrated in a young wild desert ram with clinical respiratory symptoms (Robinson et al. 1967). Kistner et al. (1975) reported that the virus was isolated from an epizootic in penned California bighorn sheep and suggested that the virus originated from a nearby herd of domestic sheep. Desert bighorn sheep in Texas were greatly reduced in number during a period when a serious epizootic of BT occurred in domestic sheep and a causal relationship was suspected (Robinson et al. 1967, Hailey et al. 1972). Captive and wild desert bighorn lambs in California with pneumatic disease consistently yielded several types of BT virus. In one case EHD virus was also isolated. Insect vectors for both BT and
EHD are particularly common during lambing season (DeForge et al. 1986) in the specific area of California where the mortality occurred.

Contagious echyma is a viral disease of domestic sheep and goats and has been reported to occur as natural and experimental disease in various wild species, including wild sheep (Blood 1971, Samuel et al. 1975, Lance et al. 1981, Lance et al. 1983). It is characterized by proliferative lesions of mucous membranes and skin, in particular in and surrounding the oral cavity. The disease is usually more severe in young animals, primarily affecting them by interfering with suckling and prehension. Older animals lacking previous exposure to the disease, or under stressful conditions, have also been severely affected (Dieterich 1981).

Outbreaks of CE have been seen in captive (Dieterich 1981) and free-ranging wild sheep (Blood 1971, Samuel et al. 1975), and have occurred in sequential years in the same herd. Researchers have suggested that this pattern was due to "latency" properties of the virus (Hebert et al. 1977, Lance et al. 1981, Zarnke and Dieterich 1985). Exogenous dexamethasone administration has resulted in reoccurrence of lesions in domestic sheep (Lance 1980), although the virus was not recovered from the resulting lesions. A similar attempt to reactivate the virus in two Dall ewes was unsuccessful (Zarnke and Dieterich 1985). Generally it is believed that cell-mediated immunity is involved in resistance to this disease (Zarnke 1984), and that compromise of this system may allow reactivation or more fulminating forms of the disease.
Serological evidence of exposure to CE was present in 9 of 18 herds of desert bighorn sheep (Clark et al. 1985) and 23-37% of Dall sheep (Foreyt et al. 1983, Zarnke et al. 1983). The disease has been reported repeatedly in Canadian Rocky Mountain bighorn sheep in both park and nonpark populations (Connell 1954, Blood 1971, Samuel et al. 1975, Davidson 1982). Persistent herd infections were associated with prolonged access to salt sources (Blood 1971, Samuel et al. 1975). High intraspecific competition, close contact of animals and the presence of an abrading substrate, such as salt blocks are believed to facilitate viral transmission (Lance et al. 1981).

2.2.2 Bacterial

The bacteria recovered from bighorn sheep include species causing specific diseases in both domestic and wild animals. Some of these bacteria are responsible for extensive bighorn mortality and have been associated with livestock contact (Goodson 1982). The majority of bacteria, however, cause only sporadic, usually localized infections. Although these may or may not be life-threatening, they are usually restricted to individuals rather than to groups of animals and are therefore of less importance to the management of wild sheep and not detailed here.

Pneumonia is the single most important disease condition of bighorn sheep. Bacterial agents isolated from pneumonic lungs of bighorn sheep include *Pasteurella haemolytica*, *P. multocida* (Foreyt and Jessup 1982, Marsh 1938, Spraker and Hibler 1977), *Bordetella* sp. (Thorne et al. 1982), *Neisseria* sp., *Hemophilus*
ovis, Actinomyces pyogenes (Buechner 1960, Marsh 1938, Spraker 1979), Streptococcus sp., Staphylococcus sp. and numerous others (Spraker and Hibler 1982).

Of these, the Pasteurella spp. and A. pyogenes have been cultured most often, both from outbreaks that occurred early in the century, as well as from more recent pneumonia outbreaks (Buechner 1960, Spraker et al. 1984). The remaining species mentioned have been isolated with less frequency in various locales without any significant patterns being evident. Some species (Streptococcus, Staphylococcus, Bordetella) are considered to be of most significance as secondary invaders (Woolf and Kradel 1973, Thorne et al. 1982), but have also been isolated as sole agents in pneumonic lesions (Spraker 1979).

Most of these bacteria have been cultured from the upper respiratory tracts of healthy ruminants and are considered to be part of the normal microflora. They are of low virulence alone and require either predisposing damage to upper and/or lower respiratory tracts, or a reduction in normal immunity, to allow their multiplication and invasion of pulmonary tissues.

Pasteurella haemolytica, in particular, has been recognized to play a major role in the mortality of bighorn sheep, at least since Potts (1937) diagnosed "haemorrhagic septicemia" among bighorn sheep in Colorado. Marsh (1938) surveyed pneumonia dieoffs of Montana bighorn sheep from 1927-1935 and reported them to be due to verminous pneumonia. He also cultured both Pasteurella spp. and A. pyogenes from these lungs. Pneumonia outbreaks among captive and wild bighorn sheep occurring since
this time in both the U.S.A. and Canada have consistently demonstrated lesions associated with the same bacteria, or yielded them on culture (Bandy 1968, Forrester 1971, Foreyt and Jessup 1982, Spraker et al. 1984). In fact, the reduction of wild bighorn numbers this century must be attributed in part to the apparently extreme sensitivity of this animal to Pasteurella.

Pasteurella spp. bacteria are comprised of several species, each with strains which are differentiated on the basis of biochemical properties (Biberstein et al. 1960). Strains may vary in their prevalence and effects in different animal species. For example, Pasteurella haemolytica biotype A is the type that is usually isolated during bovine pneumonia, domestic sheep pneumonia and preweanling lamb septicemia; whereas biotype T causes septicemia in postweanling lambs (Dungworth 1985).

Two overall syndromes in domestic animals are caused by Pasteurella infections, haemorrhagic septicemia and pneumonia. Haemorrhagic septicemia is caused by Pasteurella multocida and has been reported in deer, cattle, swine, horses, bison and camels (Camelus sp.) (Dungworth 1985). The condition in domestic sheep is more often caused by Pasteurella haemolytica and is characterized by sudden death with vague clinical signs (Blood et al. 1983).

The pneumonic form is common in cattle. Although it may be seen with various management conditions, it is often associated with transport stress, hence the disease is often referred to as shipping fever (Blood et al. 1983). Pasteurella haemolytica is the bacterium isolated most frequently from pneumonic
pasteurellosis of both cattle and domestic sheep; however, *P. multocida* may be present alone or in combination with *P. haemolytica*. Acute and chronic environmental stresses and viral respiratory infections have been described and experimentally induced to initiate the development of acute pneumonic pasteurellosis in these species (Biberstein et al. 1971, Cutlip and Lehmkuhl 1983, Friend et al. 1977, Yates 1982).

Both *Pasteurella* spp. can be recovered from the nasopharynx and tonsils of healthy bighorn sheep (Thorne et al. 1979, Marshall et al. 1983, Onderka and Wishart 1984). An atypical *P. haemolytica* biotype T has been especially common in pneumonic lungs from dieoffs in southeastern British Columbia and southwestern Alberta. The same biotype was also found in normal bighorn tonsils (Onderka and Wishart 1984, Spraker et al. 1986), and has been recently cultured from pneumonia cases in Colorado (Spraker et al. 1986). Isolates of biotype T from sick bighorn sheep and healthy domestic sheep tonsils produced fibrinous bronchopneumonia in captive bighorn sheep when inoculated intra-tracheally (Onderka 1986). Onderka (1986) also found that commercial intradermal biotype A bacterin caused septicemia and pneumonia in captive bighorn sheep within 3 days. It is not possible to compare the biotypes of recent and previous dieoffs retrospectively, since typing of *P. haemolytica* isolates was not done in the past. Both biotypes, however, appear to be capable of reproducing pasteurellosis in bighorn sheep under experimental conditions in the absence of other agents (Onderka 1986).
The development of pneumonic pasteurellosis in the bighorn sheep appears to follow the same general pattern as it occurs in domestic ruminants. Bacteria, normally resident within the pharyngeal region, multiply and subsequently invade the nasopharyngeal mucosa and lower respiratory tract. This change in the role of Pasteurella from commensal organism to pathogen is believed to be initiated by modifications in respiratory tract immune mechanisms (Harbourne 1979, Friend et al. 1982, Yates 1982). This may result from direct damage to cellular elements or interference in the normal functions of lung defenses by other agents such as respiratory viruses, and/or indirectly through physiological depression of immunity. Numerous environmental and animal-related factors such as concurrent diseases and stress from severe weather, overcrowding, harassment, heavy intestinal or lungworm infestations and poor nutritional condition appear to have significant effects on the incidence of pasteurellosis in both domestic ruminants and bighorn sheep (Thomson et al. 1975, Blood et al. 1983, Spraker et al. 1984).

Once the bacteria can multiply in an individual, transmission of large numbers of organisms to other individuals occurs by oral or nasal transfer of saliva or nasal secretions. Conditions allowing close contact within the herd increases the possibility of transmission (Yates 1982, Dungworth 1985).

Systemic disease, or what is referred to as haemorrhagic septicemia (Potts 1937, Thorne et al. 1982), may also occur in bighorn sheep. Although this condition and its clinical signs are well recognized in domestic animals, documentation of its
occurrence in free-ranging wildlife is poor, probably because of its rapid clinical progression and mortality. Pneumonic pasteurellosis in bighorn sheep can have a similar peracute clinical course, even in well observed captive animals. In most instances, affected wild sheep are found dead, preventing the observation of the progression of clinical signs.

*Pasteurella haemolytica* pneumonia does produce recognizable pathologic lesions in most animal species. These are characterized by acute fibrinonecrotizing to subacute fibrinopurulent pneumonia and fibrinous pleuritis. Chronic lesions may progress to abscessation and the formation of fibrous pleural adhesions. A variety of bacteria with or without *P. haemolytica* may be cultured from pneumonic lungs at this stage.

*Actinomyces pyogenes* is the agent recovered next most commonly from pneumonia in bighorn sheep. *Pasteurella* spp. and *A. pyogenes* are frequently found in combination in subacute and chronic bronchopneumonias of domestic sheep and cattle (Dungworth 1985). *Actinomyces pyogenes* causes sporadic infections in most animal species. It is considered to be a normal superficial inhabitant which can cause suppurative lesions of the skin and lungs as well as other tissues if permitted entry. Infections are often localized by the formation of abscesses. Mortality associated with the organism is usually due to chronic debilitating infections. The classic pulmonary lesions associated with the organism are marked suppurative inflammation of bronchioles or the formation of pulmonary abscesses.
Paratuberculosis or Johne's disease is a chronic, progressive condition caused by *Mycobacterium paratuberculosis*. The organism is ingested from a contaminated environment and multiplies within the intestinal mucosa and associated lymphoid tissues, causing focal to diffuse granulomatous inflammation. The infection may resolve or develop over months or years into an invariably fatal clinical course once intestinal absorption is severely affected. Animals become markedly emaciated and often have watery diarrhea before death.

Domestic and native wild ruminants are susceptible to the disease. It is considered to be widespread in some populations of cattle, sheep and goats; however, the inaccuracy of antemortem diagnostic tests has prevented gathering accurate prevalence data (Blood et al. 1983). Spontaneous cases of Johne's disease are reported in mountain goat (*Oreamnos americanus*), llama (*Lama glama*), moose, white-tailed deer and elk (Williams et al. 1983). It has never been diagnosed in Canadian bighorn herds but has been found repeatedly in two free-ranging bighorn herds in Colorado (Williams et al. 1979).

Williams et al. (1983) successfully transmitted a bighorn isolate of *M. paratuberculosis* to bighorn-mouflon hybrids, mule deer and elk. The latter species, as well as those previously mentioned, can potentially share ranges or contact bighorn sheep at low or high altitudes. Severe lesions considered capable of shedding large numbers of organisms were present in a mountain goat from a herd in contact with the infected bighorn herds (Williams et al. 1979), and the possibility of cross transmission...
was raised. It is therefore likely that close contact between infected and susceptible wildlife or domestic species could allow transmission of this disease and its maintenance in herds of bighorn sheep.

Glaze et al. (1982) reported a high prevalence of dental abnormalities and osteomyelitis of the mandibles of Dall sheep. Similar lesions were reported to be present in Rocky Mountain bighorn populations of Alberta (Green 1949) and Stone sheep (Ovis dalli stonei) in northern British Columbia (Howe 1981). The bony enlargements were referred to as lumpy jaw, a term usually reserved for the lesions caused by Actinomyces bovis in cattle. Actinomyces bovis, however, has never been identified in mandibular lesions in wild sheep. Actinomyces pyogenes, Fusobacterium necrophorum and miscellaneous bacteria have been cultured from lesions in sheep in Alaska (Neiland 1972) and the Yukon (Glaze et al. 1982). The pathogenesis of these lesions may be presumed to be similar to that of actinomycosis in cattle. It is postulated that conditions such as accelerated tooth wear by glacial silt contamination of forage, coarse vegetation and calcium imbalances in the diets of Dall sheep may predispose to mandibular infections (Glaze et al. 1982). Transmission of these bacteria between bighorn sheep was thought to occur at mineral licks and waterholes in Banff National Park (Green 1949).

Lesions of the oral cavity may allow the entry of other bacteria such as Fusibacterium necrophorum. This organism is a common secondary invader causing a condition recognized as necrotizing stomatitis or necrobacillosis. Necrotizing
stomatitis may remain superficial, extend into deeper tissues and bone or reach the lower gastrointestinal tract (Barker and Van Dreumel 1985). The condition is recognized widely in cattle, domestic sheep, deer, reindeer (Rangifer tarandis) and elk, and has been seen sporadically in other species (Wobeser et al., 1975, Rosen 1981).

Necrosis and acute inflammation of the lower limb and hoof are also associated with F. necrophorum as one form of foot rot in domestic animals. It appears to be the major cause of foot rot in wild ruminants. Outbreaks of necrobacillosis have been reported when winter conditions force dense feeding situations, particularly in areas where previous domestic animal or wildlife cases were present. Footrot epizootics have occurred when animals were concentrated in contaminated moist areas after periods of hot weather. Caking of limbs with mud was believed to allow entry of organisms (Rosen 1981). Neither foot rot nor necrotizing stomatitis are reported to be major disease problems in wild sheep species, except for the previous references to occasional diagnoses. It is, however, conceivable that given similar predisposing factors, the same potential exists for the development of these changes on a wider scale.

Other agents capable of initiating respiratory disease syndromes in domestic sheep, as well as other species, include Chlamydia psittaci and Mycoplasma spp. (Page and Erickson 1969, Blood et al. 1983). Chlamydia psittaci has been isolated from the nasal cavity of normal wild sheep (Pearson and England 1979). The organism is known to be involved in pneumonia in domestic
sheep and is considered to be ubiquitous with low host specificity. Subclinical chlamydial infections occur in many animal species and the organism may be harboured in the intestinal tract of some ruminant species.  

*Mycoplasma arginini* was consistently cultured during an acute outbreak of respiratory disease in captive Rocky Mountain bighorn sheep (Woolf et al. 1970, Woolf and Kradel 1973) in addition to assorted species of opportunistic bacteria. Although mycoplasma are prominently involved in respiratory diseases of other species (Blood et al. 1983), they are also isolated from the upper respiratory tracts of clinically normal animals (Springer et al. 1982, Yates et al. 1983b) without association with pathologic lesions. In the bighorn outbreak it was not possible to determine the specific significance of bacterial and mycoplasma agents; however, it seems clear that mycoplasma must be included as an agent potentially involved in bighorn disease outbreaks.  

2.2.3 Parasitic  

Bighorn sheep are hosts to a variety of types of parasites including arthropods, protozoa, cestodes and nematodes (Cowan 1951, Buechner 1960, Becklund and Senger 1967, Uhazy et al. 1971, Uhazy et al. 1973, Kistner et al. 1977). Parasitism may produce deleterious effects on host species through several mechanisms. These may vary with the species and virulence of the parasite, but include previous exposure of the host to the parasite, infection of the host by microorganisms carried by the parasite, inordinately heavy infective doses of organisms and the effects
of heavy infections on the host during times of specific physiological demands, such as poor nutritional condition, lactation, during breeding season or with concurrent disease.

Disease syndromes resulting from these direct or indirect effects of parasites on the host are reported in many wild species (Sweatman 1971, Dieterich 1981, Thorne et al. 1982); however, only a few species have been reported to be serious pathogens in Rocky Mountain bighorn sheep. Those most commonly associated with clinical signs or mortality in bighorns are Psoroptes spp., the scabies mites, and Protostrongylus spp., nematodes of the respiratory tract (Buechner 1960, Lange et al. 1980, Spraker 1979). Sporadic disease and mortality due to other parasites are also reported (Thorne et al. 1982). Other more subtle losses in the form of reductions in growth rate and physical condition are well documented in domestic livestock (Soulsby 1982). These effects are suspected but are as yet undocumented in free-ranging wild sheep.

2.2.3.1. Arthropods

The commonly recognized external parasites of wild sheep include several species of ticks and mites. These arthropods live for all or part of their life cycle on or within the superficial layers of the host's skin surface feeding on blood or skin debris. Hard ticks, Dermacentor andersoni and D. albipictus and the soft-bodied spinose ear tick, Otobius megnini are found on bighorn sheep as well as other big game and livestock species (Becklund and Senger 1967, Thorne et al. 1982).
Large numbers of blood-feeding hard ticks may weaken a young or debilitated animal through energy losses associated with irritation and hair loss, as well as by the volume of blood consumed. *Dermacentor albinotus*, in particular, is characterized by heavy feeding of adults during the winter months when hosts can least afford either blood or hair loss (Drew and Samuel 1985). *Otobius megnini* may cause marked irritation and possible hearing impairment in heavy infections when exudate is deposited in the ear canals of infected individuals (Thorne et al. 1982). *Dermacentor andersonii* and *O. megnini* can also secrete neurotoxins capable of causing paralysis leading to death of the host (Blood et al. 1983). Multiple host ticks such as *D. andersonii* can serve as vectors for the transfer of infectious diseases, including Rocky Mountain spotted fever, where these diseases are endemic (Thorne et al. 1982).

All three tick species have been identified throughout bighorn sheep ranges in the U.S.A. and Canada; however, no comprehensive surveys have defined the full extent and effects of tick infections in bighorn sheep. It is believed that ticks alone do not cause major health problems in wild sheep, although their effects on animals suffering from other conditions may be considerable.

Buechner (1960) and others (Becklund and Senger 1967, Lange et al. 1980, Foreyt et al. 1985) have identified various mite species of the genus *Psoroptes* on Rocky Mountain, Mexican and desert bighorn sheep throughout southwestern U.S.A., in Wyoming, Montana and Idaho. Epizootics of severe skin lesions (scabies)
in bighorn sheep caused by these mites have occurred since at least the mid-1800s. In addition, there have been many reports of animals with milder ear, head and neck infections caused by Psoroptes, rather than by O. megnini ear ticks (Ward 1915, Foreyt et al. 1985).

The introduction of domestic sheep into bighorn habitat has been linked with the outbreaks of scabies in wild sheep (Buechner 1960). Due to the difficulties in identification of mites, it is not yet clear which species of Psoroptes mites occur on wild sheep, which have caused outbreaks of scabies and whether they originated from domestic stock.

Psoroptes ovis mites, common to domestic sheep, cattle and horses (Sweatman 1971) and P. equi var cervinus are those species implicated in causing clinical scabies and ear infections in bighorn sheep. Mites taken from scabies cases in New Mexico were tentatively identified as P. ovis (Lange et al. 1980). These mites did not infect domestic sheep, cattle, elk or mule deer (Thorne et al. 1982). The mite was suspected to be specific to the bighorn sheep, but further transmission studies are required to prove this point.

In 1978, significant clinical signs of scabies were seen in large numbers of Mexican bighorn sheep (Ovis c. mexicana) for the first time in New Mexico (Lange et al. 1980). This major outbreak was the first to occur in recent times, to be well investigated and the first to be treated. Animals with alopecia and pruritus over a large percentage of the body and crusty, suppurative skin lesions of the ears were seen during the winter
months. Sheep in various stages of emaciation were observed by the early spring, and less than half the expected number of sheep were seen in the following year. Lange et al. (1980) attributed the reduction of sheep to either mortality, although few carcasses could be located in the rugged terrain, or to mass movement of sheep from traditional range. No direct evidence of dispersion was reported, but this has occurred in other areas in response to changes in feed and water availability (Watts and Schemnitz 1985). Eventually, Lange et al. (1980) determined that scabies alone was responsible for mortality of 60-70% of the New Mexico sheep. The outbreak was believed to be controlled by mass capture and medication of a majority of the population (Kinzer et al. 1983).

Sweatman (1971) suggested that the effects of mite infections were more severe during the winter months in part as a result of reduced nutritional quality of forage. His suggestion was supported by experiments with domestic sheep in which mange skin lesions caused by Sarcoptes spp. mites were less severe in sheep fed diets supplemented with trace mineralized salt and carotene. The lesions became more severe when animals were also infected with high levels of gastrointestinal parasites which were believed to decrease nutrient absorption (Sweatman 1971).

2.2.3.2 Protozoa

The most significant protozoal parasite of bighorn sheep are coccidia of the genus Eimeria. This parasite undergoes a direct life-cycle within the intestinal epithelium of the host animal. Pathogenic effects vary from inapparent to peracute
disease, or chronic villous atrophy from continual sequential
reinfections (Barker and Van Dreumel 1985). Coccidia are
considered host specific with a number of species described for
each host species.

Coccidiosis is characterized as a disease of herd animals
where most animals are infected but few develop clinical disease.
Animals are usually infected as juveniles and, once recovered
from the self limiting course of disease, are rarely seriously
affected again by the same species of Eimeria. High densities of
animals on a small area increase the concentration of infective
oocysts on contaminated forage. Under these conditions, clinical
disease may become widespread. Outbreaks in cattle and domestic
sheep occur in late fall and winter, especially in overcrowded
situations with animals stressed by cold or other diseases (Blood
et al. 1983). High mortality may also occur in animals lacking
previous exposure and infected with large numbers of organisms.

There have been a number of changes in the taxonomy of
Eimeria and confusion in species nomenclature, particularly in
the older literature, has resulted. The Eimeria species in Rocky
Mountain bighorn sheep have been identified as E. ahsata, E.
arloingi, E. crandallis, E. ovinoidalis, E. faurei, E. granulosa,
E. intricata and E. parva. These were found in surveys of
bighorn sheep in Colorado and Wyoming (Becklund and Senger 1967),
and all have been reported in Canada (Uhazy et al. 1971). The
first four species are considered pathogenic in domestic sheep
(Blood et al. 1983).
In domestic lambs typical signs of coccidiosis include weakness, inappetance and weight loss with soft or liquid feces. Bloody diarrhea is present in severe cases, but is less frequent than in bovine coccidiosis (Blood et al. 1983). Pout (1976) found that with natural multiple Eimeria species infections, the growth rate of lambs was not affected. Further experiments suggested that in lambs, other pathogenic factors such as concurrent enteric infections with bacterial or viral agents must be involved for conversion to clinical disease (Pout 1976).

Coccidiosis due to mixed infections has occurred in captive bighorn sheep ranging in age from 4 months to young adults (Thorne et al. 1982). Clinical signs ranged from inapparent infection to passage of soft to liquid feces. Severely ill sheep usually had concurrent respiratory or systemic diseases. The disease in free-ranging bighorn sheep is unlikely to be a serious health problem but may be a secondary disease in weakened, crowded animals.

Sarcocystis spp. are protozoa with an obligatory two-host life-cycle. Sporocysts passed in feces of a definitive carnivorous host contaminate forage or water. Ingested infective forms undergo merogony in endothelial cells and mature as cysts in the skeletal or heart muscle of an intermediate herbivorous host. More than one species commonly infects livestock and wildlife hosts at the same time. There is believed to be a high degree of host specificity (Thorne et al. 1982).

Many Sarcocystis spp. are considered to be non-pathogenic in the intermediate host (Dubey and Fayer 1983); however, those with
canid definitive hosts are more likely to cause clinical disease (Blood et al. 1983). Experimental infection of mule deer fawns with high numbers of sporocysts from a dog host has caused serious illness and death with widespread hemorrhage, anemia, lymphadenopathy and emaciation (Kistner et al. 1977).

Increasing levels of Sarcocystis infection were present during the fall and winter in naturally infected mule deer in Oregon (Dubey and Kistner 1985). The timing of the most severe infections coincided with the period of most severe weather and, hence, nutritional demands. Kistner et al. (1977) suggested that the poor physical condition of some individuals in the spring was due to Sarcocystis spp. infections.

A single species of Sarcocystis, Sarcocystis ferovis, has been definitively identified in Rocky Mountain bighorn sheep (Dubey and Fayer 1983). Muscle cysts were also present in Dall sheep (Dau 1981). Dubey and Fayer (1983) were unable to transmit S. ferovis to domestic sheep, goats or cattle, but proved that the coyote (Canis latrans) could serve as the definitive host. Domestic lambs may be seriously affected by S. tenella (Dubey and Fayer 1983), but nothing is known of the effects of this parasite on bighorn sheep. Muscle cysts are commonly found and, although no studies of the epizootiology or pathogenicity of these parasites in bighorn sheep have been done, they may cause similar effects on heavily infected wild sheep.

2.2.3.3. Helminths

The final major group of parasites which occur in bighorn sheep is composed of cestodes and nematodes.
As a general rule, the presence of helminths is far more common than the diseases they cause. The diagnosis of disease induced by these parasites, or helminthiasis, should be reserved for cases where sufficient numbers of parasites are present, pathological lesions which can be attributed to the parasites are present, and there are clinical symptoms associated with the pathogenic mechanism of the infection (Barker and Van Dreumel 1985).

The helminths of the gastrointestinal tract have been divided into five categories on the basis of feeding mechanism and pathogenic effects on the host tissues (Barker and Van Dreumel 1985). The first group is composed of those parasites which attach at a single point but are otherwise wholly lumen dwelling. Nutrients are absorbed passively from the host gut content. If the parasite is present in large numbers, subclinical diseases such as poor growth, diarrhea or obstruction may occur rarely. Tapeworms of the small intestine such as *Moniezia* are the best examples of this group.

A second group, composed of nematodes such as *Haemonchus* spp., damages the mucosa in order to consume blood. Clinical signs are associated with blood loss and hypoproteinemia. Secondary inflammation of the eroded mucosa may also occur.

The third group can be described as affecting the functional integrity of the intestinal mucosa and causing protein loss, malabsorption and diarrhea. This is the result of chronic irritation by parasites within crypts or within the epithelial cell layer. The response consists of the invasion of
lymphocytes, plasma cells and eosinophils, goblet cell metaplasia, crypt hyperplasia and villous atrophy. A large number of species including members of the genera *Ostertagia*, *Marshallagia*, *Trichostrongylus*, *Cooperia*, *Trichuris* and *Nematodirus* belong to this group.

Nematode species which burrow into or erode the mucosa and submucosa belong to the fourth group. *Oesophagostamum* spp. and others may incite protein losses and inflammatory lesions by this mechanism.

The final group causes lesions in tissues other than the gastrointestinal tract, usually due to normal, or aberrant, larval migration. Larval cestodes or nematode larvae in accidental hosts can cause lesions at various extraintestinal sites.

Representatives of each of the five groups may be present in a host simultaneously. The effects of each parasite species may be impossible to distinguish from one another, particularly if they belong to the same pathogenic group.

The cestodes of wild ruminants have relatively complicated life-cycles involving two or more hosts. The definitive hosts of the family *Taeniidae* are wild or domestic carnivores. Larval stages are present in wild or domestic herbivores. Two of the most common species in game animals are *T. hydatigena*, which forms hollow cysts or cysticerci in the liver and abdominal cavity, and *T. krabbei* larvae which infect skeletal or heart muscle (Leiby and Dyer 1971). Degenerating cysticerci are often present as fibrotic nodules within the liver, while larger cysts
may be attached to mesenteries. Secondary symptoms may result due to the location of cysts, expansive growth in organs or heavy infections.

Bighorn sheep in Colorado, Montana and Wyoming have been found infected with *T. krabbei* (Thorne et al. 1982). The cysticercus of *T. hydatigena* or "*Cysticercus tenuicollis*" is the most common larval tapeworm of bighorn sheep. This parasite has been reported to occur in Dall sheep, California, desert and Rocky Mountain bighorn sheep of the U.S.A. (Marsh 1938, Philip 1938, Allen 1955, Kistner et al. 1977, Dau and Barrett 1981), British Columbia and Alberta (Cowan 1951, Blood 1963). There is no direct evidence of mortality in bighorn sheep associated with these parasites, but heavy infections inflicting extensive damage to the liver and peritoneum have been found in bighorn and domestic sheep (Thorne et al. 1982, Blood et al. 1983).

A second group of cestodes, including *Moniezia*, *Thysanosoma* and *Wyominia* species, utilize wild and domestic ruminants as definitive hosts, occupying sites in the small intestine and bile ducts. Intermediate hosts are free-living mites in the case of *Moniezia* infections. The life cycles of *Thysanosoma* and *Wyominia* are poorly defined. These tapeworms have been reported to cause diarrhea and weight loss in young pronghorn antelope and mule deer fawns, but, although they have also been found in Rocky Mountain bighorn sheep, no evidence of clinical disease was present (Thorne et al. 1982). They have been identified in Montana, Colorado, Wyoming (Becklund and Senger 1967), British Columbia and Alberta (Cowan 1951).
Thysanosoma actinoides causes mild to marked fibrosis, inflammation and bile duct hyperplasia of the common bile duct in bighorn sheep. Large numbers of adult worms may partially occlude the flow of bile, but no clinical signs have been reported, even with heavy infections. Wyominia tetoni occupies the intrahepatic biliary system as well as the common bile duct, so that pathologic changes are similar, but more extensive than with T. actinoides. Wyominia tetoni is more widely distributed than T. actinoides, being reported in Dall sheep, California, desert and Rocky Mountain bighorn sheep in nine states (Becklund and Senger 1967, Colwell et al. 1975, Kistner et al. 1977, Thorne et al. 1982), British Columbia, Alberta and the Yukon (Cowan 1951, Gibbs and Fuller 1959, Blood 1963).

Nematode parasites comprise a large group of tubular organisms which inhabit various body systems of animals. The majority of nematodes are associated with the gastrointestinal tract and their pathogenic mechanisms have been outlined. Certain species occupying the respiratory system are of particular importance to wild sheep and will be discussed in detail later.

Gastrointestinal nematodes are so common that they are considered ubiquitous. Their effects on the gut and the health of the host are proportional to the number of parasites, their mechanism of feeding and other host related factors such as animal density, immune and nutritional status, climate and the presence of secondary microorganisms (Soulsby 1982).
Nematode infection of the gastrointestinal tract, or nematodiasis, is a significant clinical and economic problem in domestic livestock. The high prevalence of infection exists primarily because of intensive management practices. Clinical nematodiasis is most frequently seen in young, growing animals, but may occur in any age group where conditions favour the parasite. This same pattern of disease may also be presumed to occur in wildlife species.

A large number of gastrointestinal nematode genera have been identified in California and Rocky Mountain bighorn sheep and include Ostertagia, Marshallagia, Haemonchus, Skrjabinema, Nematodirus, Cooperia, Capillaria, Trichuris, Oesophagostamum, and Trichostrongylus (Becklund and Senger 1967, Boddicker and Huggins 1969, Uhazy et al. 1971, Kistner et al. 1977, Thorne et al. 1982). Becklund and Senger (1967) found that 70% of 51 species of bighorn nematodes were also parasites of domestic sheep and 35% of cattle. It is likely that in areas where livestock and bighorns graze on the same ranges at different times of the year, some species of nematodes are interchanged as animals move through contaminated pastures.

Clinical nematodiasis in bighorn sheep appears to be rare. Consistent pathological changes have been identified for some nematodes and are believed to cause significant effects on specific individuals and groups of bighorn sheep. Part of the third or fourth larval stage of Ostertagia and Marshallagia spp. is spent within the abomasal mucosa. Nodular inflammatory lesions are produced by the burrowing larvae to give a classic
"moroccan leather" appearance as has been described for domestic ruminants (Blood et al. 1983). Large numbers of larvae and adults may severely damage the mucosa and glandular elements. The resulting protein loss and diarrhea causes weight loss in domestic or wild ruminants alike (Soulsby 1982, Thorne et al. 1982, Blood et al. 1983).

The presence of Haemonchus, Skrjabinema, Nematodirus, Cooperia, Capillaria, Trichuris and Oesophagostomum spp. may cause microscopic and physiological changes in the intestinal tracts of affected bighorn sheep in a manner similar to that of Trichostrongylid-type outbreaks on domestic sheep (Soulsby 1982). They do not appear to be very significant in most free-ranging bighorn sheep; however, most reports of gastrointestinal nematode infections of bighorn sheep describe the occurrence, rather than the clinical and pathological effects of these parasites.

2.2.4 Lungworm

The wild sheep of North America are definitive hosts for several species of lung nematodes. Protostrongylus stilesi Dikmans, 1931, and P. rushi Dikmans, 1937, were first described by Dikmans in the lungs of Rocky Mountain bighorn sheep from Colorado in 1930, Wyoming in 1934 and in Dall sheep by Goble and Murie in 1942 (Forrester 1971). The host range has since been extended to include California and desert bighorn sheep (Cowan 1951, Blood 1963, Forrester 1971).

Dikmans (1930) found P. stilesi adults and larvae in nodules within the lung parenchyma, while those of P. rushi were larger and grossly visible within bronchioles and bronchi (Dikmans,
1943). A third species, *P. frosti* Honess, 1942, also present in parenchymal nodules, was described by Honess (1942) in bighorn sheep from Wyoming. Few specimens were available, however, and Dougherty and Goble (1946) suggested that this species was indistinguishable from *P. stilesi*. Later reviews concluded that *P. frosti* should be considered a valid species (Forrester 1971). Despite this conclusion, it has never been reported outside of Wyoming. The only other recognized definitive host for protostrongylid lungworms is the mountain goat. Both *P. stilesi* and *P. rushi* have been reported in mountain goats from western Canada (Cowan 1951) and the U.S.A. (Buechner 1960) and lesions of bacterial pneumonia with lungworms, similar to those in bighorn sheep, have been diagnosed occasionally (Joslin 1986).

The second lungworm genus found in bighorn sheep is *Muellerius*. *Muellerius capillaris* Mueller, 1889, is considered to be a mild pathogen of domestic sheep and goats (Dungworth 1985). It is not commonly found in bighorn sheep, but was reported as the only lungworm species present in Rocky Mountain bighorns from South Dakota (Pybus and Shave 1984). Larvae with dorsal spines resembling those of *Muellerius* have been found in Dall sheep (Goble and Murie 1942) and bighorn sheep of British Columbia, Alberta (Hudson et al. 1971), Montana and North Dakota (Pybus and Shave 1984), but conclusive species identification has not been made.

Demartini and Davies (1977) suggested that heavy infections of *M. capillaris* played a role similar to that of protostrongylid lungworms and predisposed a group of recently captive bighorns to
the development of bacterial pneumonia. No evidence of *P. stiledi* was found in these sheep. *Protostrongylus* spp., however, remain by far the most common and important parasite of bighorn lungs. References to lungworm in the remainder of this paper will therefore concentrate on those species.

Protostrongylid lungworms are, and have probably always been an integral element of the bighorn sheep respiratory disease complex. Buechner (1960) reviewed the literature on bighorns to that date and postulated that lungworm infections "acted as predisposing agents which condition the lungs for pneumonia". His historical references to lamb and adult bighorn mortality caused by lungworm or verminous pneumonia were extensive. He cited reports of these animals in poor physical condition with chronic pleuropneumonia and varying degrees of lungworm infection. Many early reports did not distinguish deaths as a result of bacterial pneumonia from those complicated by lungworm infections; however, it seems likely that lungworms were at least present in bighorn sheep prior to Dikman's description and contributed to pneumonia outbreaks.

Marsh (1938) cultured *Pasteurella* and *Actinomyces* bacteria in association with lungworm infections in bighorn dieoffs in Montana and Wyoming. Reports of subsequent bacterial pneumonia outbreaks in these and other areas consistently identified lungworm infections of various degrees of severity (Buechner 1960). Because of the constant relationship of bacteria and lungworms, pneumonic conditions of bighorn sheep have been referred to as the lungworm-pneumonia complex (Forrester 1971).
Clinical symptoms and mortality caused by lungworm are far less common than is the presence of the parasites. Lungworm infection, or protostrongylosis, is almost ubiquitous. Herd infection rates of over 90% have been commonly reported in Rocky Mountain and California bighorn sheep throughout western Canada (Blood 1963, Uhazy et al. 1973) and northwestern U.S.A. (Forrester and Senger 1964, Kistner et al. 1977, Thorne et al. 1982). Low and zero infection rates have only been found in herds of desert bighorn sheep from hot, semiarid regions of southwestern U.S.A. (Allen 1955), probably due to a lack of suitable habitat for the intermediate hosts of the parasite in these areas.

The two-host development of *P. stilesi* and *P. rushi* infections is ideally adapted to the maintenance of the parasite in populations of bighorn sheep. The life-cycle of both species is considered similar in respect to all but one phase. The apparent ability of *P. stilesi* to infect bighorn lambs in utero provides an extremely important mechanism for establishing early infections in neonatal sheep and initiating fatal respiratory disease in lambs less than 6 months of age. *Protostrongylus rushi* is believed to be less important in the pathogenesis of pneumonic disease since it is not known to infect the fetus and is less invasive of pulmonary tissues.

Adult *P. stilesi* lungworms are present in dorsal caudal lung lobes. Eggs are deposited in adjacent alveoli, develop to first stage larvae and migrate to small air passages. Larvae move into proximal airways, are swallowed and passed in fecal pellets. At
At this stage the larvae are extremely resistant to environmental conditions (Forrester and Senger 1963). Further development requires larval penetration into one of several species of tiny land snails. Suitable snail intermediate hosts are usually common on bighorn ranges and occupy microniches at the base of forage plants eaten by bighorn sheep.

After penetration into the intermediate host, larvae molt to the third and infective stage and remain viable, presumably for the life of the snail. Infected snails are accidentally consumed with forage, particularly when high density sheep populations graze pastures closely. Larvae penetrate the gut wall and migrate to the lungs for maturation to adults. Within 28 to 35 days after infection, first stage larvae appear in the feces (Spraker 1979).

Ingested third stage *P. stilesi* larvae also have the unique capability to lie dormant in the lungs, or possibly other body tissues, of pregnant ewes with preexisting lungworm infections (Hibler et al. 1974, Gates and Samuel 1977, Spraker 1979). No further development occurs until the last 6 weeks of gestation. The larvae then enter the maternal circulation and migrate to the uterus where they cross the placenta and enter the liver of the fetus. At the birth of the lamb, the larvae recommence migration and simultaneously reach the lungs. Adult *P. stilesi* are present in lungs by 3.5 to 4 weeks of age, with first stage larvae appearing in the feces by the time the lamb is 5 weeks of age (Spraker 1979).
The reactions of host tissues to lungworm infections and the clinical syndromes produced vary with the number of lungworms and the stage of the life-cycle that is present. Protostrongylosis of young lambs is the classic example of the pathogenic effects of the parasite. Spraker (1979) followed the development of transplacentally transmitted P. stilesi larvae to explain the high mortality of lambs in Colorado herds. He described the mass movement of third stage larvae from the liver into the lungs soon after birth. Multifocal, sometimes massive pulmonary hemorrhage was caused as large numbers of larvae left the circulation and occupied alveoli. Infective larvae reaching the lung, after ingestion in either lambs or adults, probably enter the lung more sporadically but develop in a similar manner.

The parasites concentrate in the dorsal caudal lung lobes. Grossly visible nodules are raised above the pleural surface. In acute infections the nodules are grey-yellow and translucent. The nodules become progressively more firm, opaque and white, then contract as fibrous tissue proliferates within alveolar walls and the pleura.

The initial cellular reaction to the larvae developing into adults is limited to mononuclear cells. When eggs and first stage larvae develop, intense infiltrations of lymphocytes, macrophages and eosinophils develop. Alveolar walls thicken with the mononuclear cells, increased smooth muscle and fibrous tissue hyperplasia. The overlying pleura and surrounding interlobular septae become increasingly fibrotic. Adjacent alveoli are usually atelectatic. Focal necrosis and acute inflammation with
neutrophils may be present when larvae invade airways for movement up the bronchial tree. Bronchiolar smooth muscle is often hyperplastic and the epithelium frequently becomes hyper and/or metaplastic. Peribronchiolar lymphoid tissue is usually prominent within the nodules and may be found at some distance from the nodule. A mucoid exudate, often containing larvae, is usually present in the lumen of airways.

In lambs or adult bighorn sheep, larvae in larger airways can be accidentally aspirated into more cranial, dependent lung lobes. These larvae are rapidly surrounded by inflammatory cells consisting mostly of macrophages and multinucleate giant cells. The granulomas initiated by degenerating larvae are extensive if large numbers of larvae are aspirated. This frequently appears to be the case in neonatal lamb protostrongylosis.

The pathogenesis of protostrongylosis in both lamb and adult bighorn sheep involves mild to extensive damage to the lower respiratory tract. If tissue damage is sufficient and pulmonary defenses are compromised by lungworms, viral or bacterial infections or other concurrent parasitic, nutritional or physiological stressors, opportunistic bacteria colonize the damaged lungs and cause typical lesions of the respiratory disease complex of bighorn sheep.

Since the health of an individual sheep may be so profoundly affected by the presence of large numbers of lungworms, numerous studies have attempted to assess the lungworm burdens of bighorn sheep. The number of first stage larvae in the feces has been associated with other physiological indices in order to describe the overall parasite and health condition of bighorn herds.
The degree of lungworm infection in an animal is proportional to the availability of infective intermediate hosts, the density of infected animals utilizing the range and the degree of infection in other animals in the herd, in addition to other host factors as previously discussed. Fecal larval output undergoes seasonal and annual fluctuations. The time of maximum counts varied between herds in several studies (Forrester and Senger 1964, Uhazy et al. 1973, Holmes and Samuel 1974). The highest mean number of lungworm larvae per gram of feces was usually seen during the late spring or in winter. The pattern of larval output variations has also changed in subsequent years in the same herds (Gates 1975).

Much of the variation in fecal larval output appears to be associated with changes in the availability of the intermediate host and the state of development of the larvae within the snail. Snails with third stage larvae must occupy sites used by sheep for ingestion and infection to occur. Snails prefer the high moisture and moderate temperatures which also favour the movement of larvae from fecal pellets, thereby increasing the snail infection rate (Forrester and Littell 1976). Snail numbers and activity were higher during spring months with high rainfall, and sheep fecal larval output was higher in the following fall, than in years with lower spring rainfall (Lange 1973, Gates 1975, Forrester and Littell 1976). Appropriate conditions are well provided in areas of sheep habitat that are preferentially used. These areas, such as those near bedding sites, invariably green up early in the spring, are heavily grazed and are believed to be
foci for transfer of infected intermediate hosts (Lange 1973). Samson and Holmes (1985) found that most first to third stage development of larval *Protostrongylus* spp. occurred during the warm summer months. Almost no development occurred during the winter. Infected snails containing third stage larvae on ranges during autumn and early winter are then probably responsible for a large proportion of the transmitted lungworm.

Forrester and Senger (1964) advised the consideration of larvae count fluctuations when assessing the lungworm infection levels of individuals or herds. They felt that the examination of a small number of fecal samples provided the closest estimation of infection rates. Uhazy et al. (1973) and Holmes and Samuel (1974) suggested that the proportion of heavily infected animals in a herd contributed another useful index of population condition. It appears that larval production may be governed by changes associated with the individual herd's environmental conditions, host nutrition, pregnancy or other physiological demands, as well as by the biology of the lungworm. The usage of several indices to lungworm numbers to estimate the levels in sheep herds must then be carried out over several years to be of value.

2.2.5 Miscellaneous

Several other disease conditions are reported and may contribute to sporadic mortality in bighorn sheep. None of the conditions appear to have a specific infectious etiology.

Amyloidosis is a systemic disease characterized by the extracellular deposition of amyloid, an eosinophilic, homogenous,
fibrillar protein. The cause of excess protein production is usually idiopathic; however, it may be produced by abnormal plasma cells or, in the secondary form, as a byproduct of chronic inflammation (Maxie et al. 1985). The most common sites of deposition are within renal glomeruli or visceral organs such as the spleen and liver. Severe cases in aged domestic animals may cause proteinuria and terminal renal failure.

Amyloidosis has been infrequently reported in free-ranging wild animals. It was seen in three emaciated, wild, adult Rocky Mountain bighorn sheep from Montana and Idaho (Hadlow and Jellison 1962). Two of these sheep had chronic frontal sinusitis and the third had chronic bacterial leptomeningitis. Seven of 17 bighorn sheep with chronic bacterial pneumonia from a dieoff of captive sheep had similar lesions (Woolf and Kradel 1973). The condition was also reported in desert bighorn sheep (Cosgrove and Satterfield 1982). All lesions occurred mainly in the kidney, liver, spleen and adrenal glands, and were consistent with secondary amyloidosis. Hadlow and Jellison (1962) remarked on the apparently high incidence of cases in wild sheep as compared to other game species, and speculated that bighorn sheep may be prone to develop amyloidosis.

Inflammation of the nasal passages and sinuses has been reported in desert bighorns (Cosgrove and Satterfield 1982) and bighorn sheep in Wyoming and Montana (Honess 1942). The condition is similar to sinusitis in domestic sheep and is associated with secondary bacterial invasion of tissues damaged by larvae of the nasal bot fly Oestrus ovis (Capelle 1966). Sinusitis appears to be a sporadic disease in wild sheep.
Capture myopathy is a degenerative disorder of cardiac or skeletal muscle seen commonly in exertionally stressed or recently captured wildlife. The affected animal may suffer from a variety of signs, including those related to primary muscle lesions, and to the secondary sequellae of shock or kidney failure. The condition is associated with prolonged muscle exertion, fear, handling and transportation, high environmental temperatures and low vitamin E-selenium levels (Spraker 1976, Chalmers and Barrett 1982). Myopathy may also predispose free-ranging animals to predation, since similar muscle changes have been present in animals after pursuit by predators or hunters (Harthoorn 1975, Chalmers and Barrett 1982).

Three clinical and pathological syndromes of capture myopathy were seen in adult bighorn sheep captured and transported for 18-24 hours (Spraker 1976, DeMartini and Davies 1977). Some sheep died within 12 hours with few gross lesions; others were ataxic and had myoglobinuria before death within 24 hours. Myonecrosis and nephrosis were the major lesions in these animals. The third group of sheep had bilateral ruptures of the gastrocnemius muscles due to widespread myonecrosis 48 hours after capture. Capture myopathy in wild sheep appears to be significant primarily during live capturing procedures. Wild and domestic carnivore or human related harassment may also initiate the condition, but it is probably sporadic in free-ranging animals.

The diagnosis of neoplasms in wildlife is relatively uncommon. Benign and malignant tumors are sporadic and may or
may not contribute to mortality. The effects of each tumor is proportional to the extent and organ(s) of involvement. Several types have been reported to occur in wild sheep. Dieterich (1981) found tumors of an "uncertain histologic type" in a Dall sheep in Alaska. An intestinal adenocarcinoma was present in a bighorn X mouflon hybrid, and pituitary and adrenal adenomas were found in Rocky Mountain bighorn sheep (Thorne et al. 1982). None of these appeared to have any significance other than to the affected individual.

2.3 RESPIRATORY DISEASE IN DOMESTIC ANIMALS

2.3.1 Current Concepts - Agent Synergism

Jakab (1982) and Yates (1982) reviewed the clinical and experimental literature on viral-bacterial interactions in respiratory diseases of domestic and laboratory animals. It has been demonstrated in many species that some virus-induced respiratory infections have an overwhelming suppressive influence on pulmonary defense systems and allow colonization of certain bacteria and the development of pneumonia.

In the normal lung, the mucociliary apparatus, alveolar macrophages and circulating leucocytes combine to intercept, inactivate and eliminate aspirated bacteria or foreign particles. Local and systemic immunoglobulins assist in local defense and particle ingestion. The pathogenesis of acute viral respiratory infections involves the modification of these reactions. This may include necrosis of epithelial cells of all levels of airways, enhanced susceptibility to bacterial attachment and
colonization and numerous dysfunctions of alveolar macrophages (Green et al. 1977, Warr and Jakab 1979, Yates 1982). Pulmonary responses produce an inflammatory cell invasion of peribronchiolar tissues and airway lumina, and filling of alveoli with edema fluid and leucocytes, secondarily impairing antibacterial functions at this level. The secondary infection of lung tissues by bacteria occurs only under certain conditions. Respiratory tract defenses must be affected at all levels to allow pathogenic bacteria to multiply within the upper respiratory tract, reach, invade and multiply in distal air passages and alveoli. It appears from experimental studies, that only with properly timed, large doses of virus do the pulmonary antibacterial defenses fail completely to allow the rapid growth of bacteria and the production of fatal pneumonia.

In the natural state, however, other factors must be considered to act as concomitant stressors with viral infection to reduce the efficacy of antibacterial defenses of the lung. Rarely do pure infections occur as in experimental situations. There is much evidence that other agents, such as mycoplasmas and chlamydia, other concurrent diseases and environmental stressors assist in decreasing resistance to secondary bacterial invasion.

Combined infections of viral and bacterial agents in domestic sheep and cattle are associated with initiating more severe respiratory disease than the disease caused by either pathogen alone (Friend et al. 1977, Warr and Jakab 1979, Jericho et al. 1982, Yates 1982, Trigo et al. 1984). Initially, studies

Synergistic effects of PI3 or RSV and *P. haemolytica* have been demonstrated and were highly reproducible in both immunocompromised and conventionally reared domestic lambs (Sharp et al. 1978, Rushton et al. 1979, Davies et al. 1981, Trigo et al. 1984). Pneumonia of varying severity proportional to the experimental conditions were present, i.e. more severe in immunocompromised animals. Experimental infection with an ovine isolate of RSV alone, and with *P. haemolytica*, produced only mild clinical respiratory symptoms in 3 month old domestic lambs (Trigo et al. 1984). However, lesions were more severe and there were increased numbers of inflammatory cells in the dual infection. Although the results did not indicate that this virus was a significant pathogen under the experimental conditions, Evermann et al. (1985) suggested that with intensification of management, stressful conditions or multiple bacterial or viral infections, the susceptibility of sheep to RSV may increase. These authors also speculated that, because this virus was antigenically related to bovine RSV, there was definite potential for interspecific transmission.
A great deal of time and money has been spent to elucidate the synergism of viral and bacterial agents in respiratory diseases. Despite the advances in identification of organisms and definition of immunological dysfunctions, the mechanisms linking the interactions of host, pathogen and environment to cause disease are still not completely understood in domestic species. Although extrapolations to wild species may be made and may be applicable, experimental studies are obviously needed before the mechanisms may be understood in wild sheep.

3.0 HYPOTHESES AND OBJECTIVES

As has been described, Rocky Mountain bighorn sheep undergo periodic all-age respiratory disease epizootics. The respiratory disease is characterized by a peracute to chronic bronchopneumonia. Opportunistic microorganisms are the most commonly identified pathogens, but immunosuppression by a complex of physiologic and environmental factors is believed to initiate clinical disease. Herds of bighorn sheep in the East Kootenay region of British Columbia have shown two basic patterns of herd dynamics; low elevation wintering herds which suffer periodic all-age dieoffs and high elevation wintering herds which remain stable in numbers.

The working hypotheses for this study were that:

1. Herds of bighorn sheep with differing histories of disease occurrence have differing, measurable physiological and pathological parameters.
2. Certain environmental and physiological factors can be associated with the differing parameters and these factors can be associated with the overall herd dynamics and health status.

3. Parameters can be selected and monitored to assist in the evaluation of herd status and appropriate management actions.

The study was designed to achieve the following major objectives:

1. To describe and compare the health status of individual bighorn sheep from three herds in the East Kootenay region of British Columbia.

2. To associate the health status of collected sheep with environmental and physiological factors and the overall dynamics of each herd.

3. To select parameters suitable for future monitoring of bighorn sheep herd health status.

4.0 STUDY

4.1 INTRODUCTION

Investigations into all-age dieoffs of bighorn sheep in the E.K. region of British Columbia in the 1960's and 1980's determined that these dieoffs were preceded by high population densities, heavy interspecies competition for forage, contact with domestic sheep, severe winter weather, high lungworm levels and low copper and selenium levels in some sheep (Stelfox 1971, Davidson 1982, Schwantje 1983). It was concluded that the herds in which dieoffs occurred were predisposed to pneumonia by
chronic stress conditions caused and complicated by high animal density, poor nutrition, parasitism and trace mineral deficiencies. The presence of concurrent or subclinical diseases was unknown (Schwantje 1983).

Sheep herds wintering at high elevation were not affected during either of the previous dieoffs in the E.K. region. This suggested that the absence of some of the previously mentioned factors allowed the sheep herds at high elevation to be maintained in better overall health than those which suffer from dieoffs, and that they have thus avoided disease epizootics.

A definitive study was required to evaluate the presence of these factors in E.K. herds with and without the history of dieoffs. The following project surveyed three herds of similar size which differed in herd dynamics and disease occurrence. Samples were compared and evaluated for the presence and influence of predisposing factors, to describe the overall health of each herd and to apply this information to the overall management of E.K. bighorns.

4.2 STUDY HERDS

The three herds selected for this project are designated by their winter range: Columbia Lake (CL), Wigwam (WW) and Ewin Ridge (ER) (Fig. 1).

The sheep from CL and WW herds graze summer pastures in lamb-ewe and juvenile and ram groups at elevations up to and above 2170 metres. In the fall they return along what are believed to be traditional corridors to lower elevation
Fig. 1. Study area in southeastern British Columbia
(1000-1500 m), predominantly seral ranges along the eastern boundary of the Rocky Mountain Trench (Bandy 1968). The time of arrival on winter range varies between sexual groups and herds and may vary between years, but is usually during September and October (Davidson pers. commun.). This is followed by the rut as breeding males arrive in November to December. The herds usually stay together on the winter ranges until early spring (April - May) when the pregnant ewes separate themselves and either stay or move to established lambing grounds in suitable terrain. The reverse migration occurs when snows retreat from the high country.

The winter ranges of these two herds are easily accessible by dirt road. They are heavily used during elk and deer hunting seasons, as well as for nonconsumptive recreational use, such as hiking and photography. Vehicle access to both winter ranges is possible until December 1 after which roads are closed. Although the closures are generally respected, vehicle and foot access is still possible and does occur after this date.

The CL range is situated at 50°12'N and 115°50'W. It consists of alluvial fans, parkland areas and rocky bluffs on the eastern shore of Columbia Lake. Conifer succession is very prominent around most meadows. Livestock have been absent from the range for over 20 years; however, mule deer and especially elk are abundant. The quality and amount of forage on this range has been considered poor since at least 1970 (Demarchi 1970, Davidson pers. commun.). Exclosures erected in 1983 showed obvious differences in forage growth by 1984. The CL range is
adjacent to a small community and shares a portion of the range with a growing residential subdivision. The sheep feed readily on planted grasses and wander through the subdivision to reach the lakeshore and mineral licks. Free-ranging dogs from the community are reported to frequent the winter range and cause considerable harassment to the sheep (Davidson pers. commun.).

In 1966, a dieoff reduced the CL herd from 100 to 28 animals (Bandy 1968, Demarchi 1970). In 1983 the herd consisted of approximately 150 animals. Annual lamb:ewe ratios were considered to be gradually decreasing and were 0.4–0.5 in 1983 (Davidson pers. commun.).

The WW range is made up of fire-formed alluvial plain grasslands, some natural parkland and thickly forested slopes between the Elk and Wigwam Rivers at 49°17'N and 115°5'W. Livestock are not permitted on the range but mule deer and, particularly, elk are abundant. The range condition appeared to have improved following reductions in the wildlife stocking rate, as was seen after a sheep dieoff in 1965 and subsequent increases in elk harvests (Demarchi 1970). It was considered to be improving after the 1981 sheep dieoff. The WW range is separated from a small community and a lumber mill by the Elk River. The inhabited shore is not often used by sheep. The side immediately opposite, however, has supported large groups of sheep in the past. Most of the available WW range is out of visual and acoustic contact with the community and mill.

The WW herd also suffered a 40–50% reduction in a major all-age dieoff in 1965 (Bandy 1968). The 150 animals remaining
after the dieoff had increased to at least 424 by 1981, the highest density ever reported for the range (Warkentin pers. commun.). At this time, elk and mule deer numbers were also considered high, similar to levels prior to the 1965 dieoff.

Another all-age dieoff began in December 1981 and reduced the population to fewer than 150 bighorn sheep. Animals in poor condition and/or demonstrating respiratory symptoms were still occasionally reported in the years after the dieoff. The herd contained approximately 100-150 sheep with lamb:ewe ratios of less than 0.05 in 1983.

The ER sheep winter on virtually inaccessible, windswept, west-southwestern aspect subalpine and alpine ridges up to 2700 m in elevation at 50°4'N and 114°45'W. At this high elevation both the rut and onset of winter conditions begin approximately one month earlier than on the low altitude ranges. Snow conditions closely control the movement of sheep so that they remain on winter range until a reduction of snowpack allows them to disperse. The amount of available range is reduced with heavy snowfalls or the late arrival of spring. When this occurs, intraspecific competition limits the quantity of forage available to overwintering sheep (Schuerholz 1983). A large open-pit coal mine is present on traditional ram summer range but the winter range common to both sexes is generally undisturbed. Extensive range studies demonstrated excellent range quality and a lack of forage or space competition from mule deer or elk (Schuerholz 1983, 1984).
This herd has never been reported to suffer large scale mortality, although, because of the location, we can only base our judgement on 15 years of aerial classified counts. These have remained stable at approximately 100-150 sheep since 1971 (Schuerholz 1984). The lamb:ewe ratio has averaged 0.55 since 1971 and was 0.78 in 1984.

4.3 MATERIALS AND METHODS

In 1983 and 1984, the CL and WW herds were observed by me and counted from the ground (foot and automobile). Data on the ER population were supplied by Crowsnest Resources and Transamerica Environmental Science Consultants (TAESCO), as well as through minimal direct observation during early winter. Animals in all herds were counted and differentiated as to sex and whether juvenile or adult (classified count) using fixed wing aircraft or helicopter in January or February by B.C. Wildlife Branch personnel.

Six sheep were collected from each herd. Selection criteria were designed to select for those animals which were of less than the average body condition, or that demonstrated clinical signs of disease, so that they appeared different from other sheep in the group. Criteria included body condition and clinical symptoms of disease or signs stated by wildlife biologists or experienced observers as indicative of poor condition or subclinical disease. The signs ranged from behaviour in which an animal was isolated from the group to pale or rough haircoats, loss of muscle mass, the presence of lesions of contagious
ecthyma and mild to severe respiratory symptoms. Animals were also selected from three age classes; lambs of the year, yearlings and adult ewes. The collections from the ER herd used only these latter criteria.

From late October to early December 1983 observations of major CL and WW groups were made, usually from a vehicle. The animals were observed for 0.5 to 1 hour before an individual was collected for sampling. Disturbance was considered minimal before collection. Ewin Ridge animals were observed from helicopter and collected after hazing towards a ground based hunter during late October and early November 1984.

Sheep were shot in the caudal skull or anterior cervical region. Blood was collected immediately, chilled and serum was harvested on the following day. Each animal was weighed and a necropsy was performed. Carcasses were evaluated subjectively for body condition on the basis of body fat deposits and muscling for a total body score of 100 after the method of Kistner (1980). Major organ systems were examined and representative portions were preserved in 10% neutral buffered formalin. The thoracic thymus was collected where identifiable, photographed and sectioned.

The lungs were removed intact and photographed. Lungworm affected lungs were classified as mild, moderate or severe. The mild classification had less than one quarter of the caudal lobes affected by parasitic lesions, while moderate to severely affected sheep had one-third to one-half of the caudal lung lobes involved. Lesions of lungworm or bacterial pneumonia were
recorded by drawing on a standard outline, and at least one 2 cm slice of each lobe was preserved. Portions of anterior or grossly affected lobes, as well as a retropharyngeal lymph node and main stem bronchial swabs, were obtained for bacterial and viral culture. These tissues were chilled and frozen, respectively, and transported to the Alberta Agriculture Laboratory, Edmonton, Alberta for viral and bacterial culture using standard methods employed in that laboratory during the period October 1983 to December 1984 and under the direction of Dr. J.F.C.A. Pantekoek.

Liver was frozen and assayed for trace minerals at the British Columbia Veterinary Pathology Laboratory, Abbotsford, B.C. using standard methods employed in that laboratory during 1985 and under the direction of R. Puls. The tissue was digested and analyzed by spectrophotometry for selenium levels and with atomic absorption spectrophotometry for copper, zinc and manganese. Copper and selenium levels were converted from parts per million to micromoles per litre by using conversion factors of 15.74 and 12.665 respectively.

Serum was frozen and analyzed later for the presence of antibodies to infectious bovine rhinotracheitis (IBR), parainfluenza 3 virus (PI3), bovine virus diarrhea (BVD) and bovine respiratory syncytial virus (RSV) by serum virus neutralization tests in cell culture at the Department of Veterinary Microbiology, Western College of Veterinary Medicine, Saskatoon, Saskatchewan, under the direction of Dr. P. Durham. Results were given as positive or negative. The presence of
antibodies to bovine lymphosarcoma virus (BLV) was determined for one animal from the WW herd by agar gel immunodiffusion at the Agriculture Canada Animal Disease Research Institute in Lethbridge, Alberta. Evaluations for antibody titres to bluetongue (BT), epizootic hemorrhagic disease (EHD), and Johne’s disease (Mycobacterium pseudotuberculosis) were determined by agar gel immunodiffusion tests. Complement fixation was also used on sera for titres to Johne’s disease. These tests were performed by standard methods used by the Agriculture Canada Animal Disease Research Institute, Nepean, Ontario, under the direction of Dr. J.L. Shapiro. Sera were evaluated by agar gel diffusion tests for maedi/visna virus antibodies using standard methods employed by Agriculture Canada in Sackville, New Brunswick under the direction of Dr. R.G. Stevenson.

Samples of feces were frozen and analyzed for nitrogen by the Ministry of Agriculture Soil, Feed and Tissue Testing Laboratory, Kelowna, B.C. by a modified (Hebert et al. 1984) Kjeldahl method using selenium, hydrogen peroxide and sulphuric acid. Total fecal nitrogen content was then determined colourmetrically. Gastrointestinal parasite ova counts were done with routine fecal flotations and parasite identification were performed in the diagnostic laboratory, Department of Veterinary Microbiology, W.C.V.M. by A. Gajadhar. Fecal lungworm larvae counts were performed on air dried feces by the Department of Zoology, University of Alberta, Edmonton, Alberta using a Baermann technique (Samuel and Gray 1982).
Serum blood urea nitrogen levels were determined with a Beckman BUN Analyzer II (Brea, California) by an electrical conductivity method. Glucose levels were assessed with an ABA 100 (Abbott Diagnostics, Texas) using a hexokinase method. Total serum proteins were also assessed with an ABA/100 using a biuret reaction and an A-gent reagent (Abbott Diagnostics, Texas). These tests were performed by Clinical Pathology personnel, Department of Veterinary Pathology, W.C.V.M. Cortisol concentrations in serum and urine were determined by radioimmunoassay at the Department of Veterinary Pathology, College of Veterinary Medicine, Fort Collins, Colorado, using methods employed by that laboratory in 1985 and under the supervision of Dr. T.R. Spraker.

Tissues for histology were embedded in paraffin, sectioned at 5 um thickness and stained with hematoxylin-eosin. Special stains (Masson’s trichrome, PTAH, PAS, Grocott, Brown and Brenn) (Luna 1974) were used as required. Three microscopic sections were taken from each lung slice as well as additional sections from lesions seen grossly. The preserved left kidney and adrenal from each animal were weighed and photographed.

Histopathological findings were recorded for all organs. The microscopic changes in tissues other than the lung were graded into four categories on the basis of the severity of the most significant pathologic abnormalities. The significant abnormalities were inflammatory cell infiltration, lymphoid tissue hyperplasia and infection by Sarcocystis spp. and contagious ecthyma. Each was assessed subjectively and the
degree of change ranked as normal, mild, moderate or severe.

Pulmonary disease was categorized on the basis of the most common microscopic changes. The prominent pulmonary changes were divided into seven major categories; those associated with the pleura and septae, airways, vessels, interstitium, alveoli, inflammation associated with lungworms and life cycle stages of Protostrongylus spp.. Each of these categories was further subdivided to indicate either pathologic changes, the presence of specific inflammatory cells or parasite stages. Each lung section was examined without knowledge of the sheep of origin at 40 power magnification. Ten fields of view were surveyed following the same pattern and observing the presence or absence and severity of each change, cell type or parasite stage. These changes were summarized for each herd.

Statistical analysis was by one-way analysis of variance, where significance was detected, a Neuman-Keul's Range Test was used to differentiate among the groups. Means are reported with standard deviations, significance was accepted at $p \leq 0.05$.

5.0 RESULTS

5.1 CLINICAL EVALUATION AND CONDITION

5.1.1 Columbia Lake

The sheep in this herd were usually seen in groups of 40 to 65 animals. They could be approached to within 15 m and remained in the immediate area during all procedures. On clinical inspection individual sheep appeared in good to excellent body
condition. The pelage was occasionally long and/or pale or yellow. Three sheep (CL 1, 4, 5) were selected for these haircoat changes (Table 1). One ewe was selected because of a mild dry cough (CL 6). This was the only sign noted during all observations. Some sheep had faintly depigmented areas on the oronasal mucosa, suggestive of healed contagious ecthyma lesions. A mean body condition score of 85.8 was estimated for collected sheep at necropsy (Table 2).

CL 1: This young ram was selected on the basis of a haircoat, graded as fair, which was long and patchy over the forequarters and neck. The rump appeared more angular than other sheep in the herd but body condition was graded as good. Muscling and fat deposits were, however, excellent on necropsy.

CL 2: The lamb was considered typical of this age class in the CL herd. The haircoat and body condition were considered in good to excellent on clinical and necropsy examinations.

CL 3: This lamb was smaller in size than others in the group. Haircoat and body condition appeared good to excellent; however, on necropsy, fat deposits on the rump, brisket and omentum were mildly depleted, thereby reducing the body score.

CL 4: The ewe appeared to be in fair body condition with a slightly rounded abdomen and an angular rump. The haircoat was considered fair. It was long and rough with mild hair loss and a few scabs over the forequarters and dorsum. A unilateral ocular discharge was suspected but not confirmed at necropsy due to the destruction of the orbits by gunshot. A few tick larvae present on the hide were identified as *Dermacentor albipictus*. General
Table 5.1. Selection criteria of bighorn sheep collected from three herds in the East Kootenays in 1983-84.

<table>
<thead>
<tr>
<th>Sheep number</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Body weight (kg)</th>
<th>Body condition</th>
<th>Haircoat</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>CL 1</td>
<td>2.5</td>
<td>M</td>
<td>77.3</td>
<td>G</td>
<td>long,patchy</td>
<td>---</td>
</tr>
<tr>
<td>CL 2</td>
<td>0.5</td>
<td>M</td>
<td>40.9</td>
<td>G-E</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>CL 3</td>
<td>0.5</td>
<td>M</td>
<td>32.7</td>
<td>G-E</td>
<td>---</td>
<td>small size</td>
</tr>
<tr>
<td>CL 4</td>
<td>4.5</td>
<td>F</td>
<td>72.7</td>
<td>F</td>
<td>long,thin</td>
<td>ocular discharge suspected</td>
</tr>
<tr>
<td>CL 5</td>
<td>1.5</td>
<td>M</td>
<td>64.5</td>
<td>F-G</td>
<td>long,rough,yellow</td>
<td>---</td>
</tr>
<tr>
<td>CL 6</td>
<td>3.5</td>
<td>F</td>
<td>71.4</td>
<td>G-E</td>
<td>patchy</td>
<td>few dry coughs</td>
</tr>
<tr>
<td>WW 1</td>
<td>0.5</td>
<td>M</td>
<td>29.1</td>
<td>VP-P</td>
<td>rough,yellow</td>
<td>isolated,lethargic nasal discharge, CE</td>
</tr>
<tr>
<td>WW 2</td>
<td>3.5</td>
<td>F</td>
<td>71.8</td>
<td>F</td>
<td>long,rough,pale</td>
<td>---</td>
</tr>
<tr>
<td>WW 3</td>
<td>0.5</td>
<td>F</td>
<td>14.5</td>
<td>VP-P</td>
<td>long,rough</td>
<td>isolated,lethargic weak, nasal discharge, CE, diarrhea</td>
</tr>
<tr>
<td>WW 4</td>
<td>6.5</td>
<td>F</td>
<td>64.5</td>
<td>F</td>
<td>---</td>
<td>CE</td>
</tr>
<tr>
<td>WW 5</td>
<td>3.5</td>
<td>F</td>
<td>65.5</td>
<td>F</td>
<td>yellow</td>
<td>CE, diarrhea</td>
</tr>
<tr>
<td>WW 6</td>
<td>1.5</td>
<td>M</td>
<td>53.2</td>
<td>F</td>
<td>long,pale</td>
<td>isolated</td>
</tr>
<tr>
<td>ER 1</td>
<td>0.5</td>
<td>F</td>
<td>23.6</td>
<td>G</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>ER 2</td>
<td>4.5</td>
<td>F</td>
<td>67.3</td>
<td>F-G</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>ER 3</td>
<td>3.5</td>
<td>F</td>
<td>68.2</td>
<td>G</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>ER 4</td>
<td>1.5</td>
<td>M</td>
<td>41.8</td>
<td>G</td>
<td>pale</td>
<td>small size</td>
</tr>
<tr>
<td>ER 5</td>
<td>7.5</td>
<td>F</td>
<td>81.8</td>
<td>G</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>ER 6</td>
<td>9.5</td>
<td>F</td>
<td>72.7</td>
<td>G</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

a E = excellent  
G = good  
F = fair  
P = poor  
VP = very poor  
b CE = contagious ecthyma
Table 5.2. Body condition scores of bighorn sheep collected from three herds in the East Kootenays in 1983-84.

<table>
<thead>
<tr>
<th>Sheep number</th>
<th>Muscle mass</th>
<th>Fat depots</th>
<th>Total Body Score(^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CL 1</td>
<td>10</td>
<td>90</td>
<td>100</td>
</tr>
<tr>
<td>CL 2</td>
<td>10</td>
<td>90</td>
<td>100</td>
</tr>
<tr>
<td>CL 3</td>
<td>10</td>
<td>70</td>
<td>80</td>
</tr>
<tr>
<td>CL 4</td>
<td>5</td>
<td>70</td>
<td>75</td>
</tr>
<tr>
<td>CL 5</td>
<td>10</td>
<td>65</td>
<td>75</td>
</tr>
<tr>
<td>CL 6</td>
<td>10</td>
<td>75</td>
<td>85</td>
</tr>
</tbody>
</table>

\[ x = 85.8 \pm 11.6 \]

| WW 1         | 5           | 30         | 35                     |
| WW 2         | 10          | 80         | 90                     |
| WW 3         | 5           | 10         | 15                     |
| WW 4         | 5           | 80         | 85                     |
| WW 5         | 5           | 65         | 70                     |
| WW 6         | 5           | 45         | 50                     |

\[ x = 57.5 \pm 29.5 \]

| ER 1         | 10          | 55         | 65                     |
| ER 2         | 10          | 60         | 70                     |
| ER 3         | 10          | 75         | 85                     |
| ER 4         | 10          | 75         | 85                     |
| ER 5         | 10          | 80         | 90                     |
| ER 6         | 10          | 80         | 90                     |

\[ x = 80.8 \pm 10.7 \]

\(^a\) - Body scores were not significantly different among groups.
body muscling and fat deposits of the rump, brisket and omentum were reduced.

CL 5: The body muscling of this ram was mildly decreased and there was a yellow, dry appearance to the haircoat. He was graded as fair to good for both categories. Slight hair loss and a few scabs were present over the forequarters. No ectoparasites were observed. Fat was slightly decreased in all body deposits.

CL 6: This ewe was observed in sternal recumbancy and made a series of dry coughs during the period of observation. She appeared to be in good to excellent body condition with a dark, patchy haircoat. No abnormalities of haircoat nor reduction in muscling or fat deposits were present on necropsy.

5.1.2 Wigwam

This herd was wary and was scattered as pairs or small groups over the range. Approach was possible by vehicle but sheep dispersed rapidly with disturbance. Field inspection revealed most of the sheep to be adults of poor to good body condition. Some groups were uniformly thin. Sheep selected had long pale haircoats and were thinner than average (Table 1). The two lambs collected (WW 1, 3) were very small, thin and lethargic. Both had nasal discharges and obvious oral lesions which appeared to be typical of contagious ecthyma. Oral contagious ecthyma was also severe in WW 4, mild in WW 5, with prominent nasal scarring in WW 6. Diarrhea with perineal staining was present in WW 3 and 5. All animals except WW 5 had Otobius megnini and D. albipictus ticks in light to heavy numbers. No signs of coughing were observed. The mean body condition score was 57.5 (Table 2).
WW 1: This lamb moved slowly and his ears were lowered. The poor haircoat stood on end, especially over the neck, and was dry and yellow. A crusty exudate was present around the nares and lips. The body condition was very poor to poor. Necropsy revealed no fat deposits over the rump and brisket and reduced muscling and fat deposits or serous atrophy of fat in the remainder of the carcass. The body score was lower than the clinical examination suggested due to the haircoat standing on end reducing the angularity of the body profile.

The hooves were overgrown. Circular scars and small raised papules were present beneath the nares and along the lips. A single engorged *D. albidus* was present on the muzzle.

WW 2: This ewe appeared small with an angular rump and was graded to be in fair condition. Her haircoat was also considered to be fair and was pale, long and rough. *Otobius megnini* ticks were present in one ear. Body fat deposits and muscling on necropsy appeared more abundant than determined on clinical examination.

WW 3: The lamb was thin, of extremely small size and considered to be in very poor to poor condition. It stood with a hunched back, lowered ears and moved slowly. The very poor to poor haircoat was long, rough and stood out from the body. A small amount of nasal discharge and crusting of the lips was visible. Fecal staining was present on the perineum and hocks.

A single *D. albidus* tick was found at the tail base and large numbers of *O. megnini* were within one ear. Proliferative lesions were on both lips, the coronet bands of both forefeet and
the right hind foot. Decubitus ulcers were present over the left carpus and both hind fetlocks. All hooves were overgrown. Skeletal muscles were atrophied and almost total serous atrophy of all body fat deposits was found on necropsy.

WW 4: The ewe appeared thin with a more prominent backbone than other animals in the group. She was considered to be in fair condition with a good haircoat. Both lips drooped with the weight of numerous large proliferative growths. Similar lesions were also present on the dental pad, gingiva, hard and soft palates and several rumen pillars. A single *O. megnini* nymph was found in one ear. Body muscling and rump and brisket fat deposits were only slightly reduced.

WW 5: This ewe was graded as being in fair body and good haircoat condition, appearing thin with a yellowish tinge to the coat. Body muscling and fat deposits were all slightly reduced. There were mild proliferative lesions around the left nostril. A small amount of fecal staining was present at the perineum.

WW 6: The ram appeared slightly thin, had a long pale haircoat and was graded as having fair body and coat condition. A moderate number of *O. megnini* were present in one ear. Discrete depigmented areas were present on the dark mucosa of both nostrils. There was a generalized reduction of body muscling and fat deposits.

5.1.3 **Ewin Ridge**

Clinical evaluation of these sheep was not possible but the carcasses were examined after collection (Table 1). The only noticeable abnormality was that the yearling (ER 4) had a pale
but otherwise normal haircoat and was of small size. The other sheep were in good body condition with a mean body condition score of 80.8 (Table 2).

ER 1: This lamb was in good physical and haircoat condition with no abnormalities on gross examination. Body fat deposits were moderately reduced but muscling appeared to be maintained.

ER 2: The rump of this ewe was slightly angular but she appeared in good body condition. The haircoat was in good condition. Body fat deposits were moderately reduced in all but the perirenal location. She was the dam of ER 1.

ER 3: The ewe was in good body and haircoat condition and only a slight decrease of body fat deposits was seen.

ER 4: This yearling ram was of small size, but was in good physical condition. The haircoat was pale in colour but in good condition. Body fat deposits were only slightly reduced.

ER 5: This large ewe was in good physical condition with good haircoat. There was little reduction in body fat deposits.

ER 6: The ewe was large and in good physical and haircoat condition. Little change in body fat deposits was noted.

5.2 GROSS NECROPSY

5.2.1 Columbia Lake

Five of the six CL sheep were classified as having moderate to severe verminous pneumonia. Animal CL 1 was classified to have mild lesions. All pneumonic lesions were typical of those described by Spraker (1979) and others as resulting from infection by the lungworm Protostrongylus stilesi. Infected
lungs had pale, firm, rounded nodules protruding from the surface of the lung. Nodules were always concentrated in the dorsal caudal lung lobes or the dorsal aspect of other lobes, but individual small nodules were occasionally present in distal cranial or middle lobes. Nodules were frequently fused in the caudal lobe and covered by thickened opaque pleura. Smaller nodules were translucent with yellow-grey mottling. Airways adjacent to lungworm nodules were usually surrounded by glistening white cuffs.

*Protostrongylus rushi* adults were present in the airways of all six sheep. A single small focus of consolidated parenchyma was present in the right cranial lung lobe in CL 6. This was later determined to be a sterile focus of bronchopneumonia, possibly induced by larval aspiration.

*Wyominia tetoni* tapeworms were found in four sheep (CL 2, 4, 5, 6). Heavy infections were accompanied by bile duct and gall bladder dilation and fibrosis. Sheep CL 4 had two 1 and 2 cm green laminated abcesses adjacent to these structures. Multiple 0.2 cm nodules on the abomasal rugae and tiny white foci over cecal and colonic serosae and mucosae were common and were considered to indicate gastrointestinal parasitism due to various nematode species, including *Ostertagia* and *Marshallagia* spp. The kidney: adrenal weight ratios ranged from 24.6 to 37.3 with a mean of 30.4 (Table 3).

CL 1: There were a few 0.5 - 1 cm diameter raised, pale nodules on the pleural surface of the dorsal caudal lung lobes. Large airways contained *P. rushi* adults. No other abnormal findings were present in other tissues.
Table 5.3. Preserved kidney and adrenal gland weight ratios of bighorn sheep collected from the East Kootenays in 1983–84.

<table>
<thead>
<tr>
<th>Sheep number</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Body Weight (kg)</th>
<th>Kidney:Adrenal ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>CL 1</td>
<td>2.5</td>
<td>M</td>
<td>77.3</td>
<td>= 29.4</td>
</tr>
<tr>
<td>CL 2</td>
<td>0.5</td>
<td>M</td>
<td>40.9</td>
<td>= 37.3</td>
</tr>
<tr>
<td>CL 3</td>
<td>0.5</td>
<td>M</td>
<td>32.7</td>
<td>= 33.0</td>
</tr>
<tr>
<td>CL 4</td>
<td>4.5</td>
<td>F</td>
<td>72.7</td>
<td>= 24.6</td>
</tr>
<tr>
<td>CL 5</td>
<td>1.5</td>
<td>M</td>
<td>64.5</td>
<td>= 26.5</td>
</tr>
<tr>
<td>CL 6</td>
<td>3.5</td>
<td>F</td>
<td>71.4</td>
<td>= 31.7</td>
</tr>
<tr>
<td>WW 1</td>
<td>0.5</td>
<td>M</td>
<td>29.1</td>
<td>= 23.5</td>
</tr>
<tr>
<td>WW 2</td>
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<td>F</td>
<td>71.8</td>
<td>= 23.8</td>
</tr>
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<td>WW 3</td>
<td>0.5</td>
<td>F</td>
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<td>= 9.4</td>
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<td>WW 4</td>
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<tr>
<td>WW 6</td>
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<td>M</td>
<td>53.2</td>
<td>= 25.5</td>
</tr>
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<td>ER 1</td>
<td>0.5</td>
<td>F</td>
<td>23.6</td>
<td>= 34.2</td>
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<tr>
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<td>F</td>
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<td>ER 3</td>
<td>3.5</td>
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<td>68.2</td>
<td>= 26.1</td>
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<tr>
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<td>81.8</td>
<td>= 26.7</td>
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<tr>
<td>ER 6</td>
<td>9.5</td>
<td>F</td>
<td>72.7</td>
<td>= 26.7</td>
</tr>
</tbody>
</table>

\[ x = 30.4 \pm 4.6^a \]
\[ x = 21.8 \pm 6.9^b \]

\[ x = 28.0 \pm 3.1^{a,b} \]

\[ a, b \] – values with different superscript letters are significantly different
CL 2: *Protostrongylus rushi* were present in large airways. Nodules of *P. stilesi* were diffusely scattered throughout all lung lobes and varied in size from 0.2 to 2 cm. Nodules in the distal lobes were small, mottled and translucent while the dorsal aspects and the dorsal caudal lobes contained large and often confluent nodules. Full thickness areas of the dorsal caudal lobes measuring 3 x 4 cm were firm, pale and swollen. The overlying pleura was opaque. Small airways on cross section were surrounded by white glistening cuffs. Retropharyngeal, bronchial and mediastinal lymph nodes were enlarged 2-3 times normal size.

The common bile duct was dilated and the wall was thickened. It contained a yellowish mucoid exudate and many mature *W. tetoni*. There were multiple discrete nodules present on the mucosal surface of the abomasum and tiny red nematodes, identified as *Marshallagia* spp. were within the ingesta.

CL 3: *Protostrongylus rushi* adults were also visible within large airways. There were a few 1-1.5 cm nodules along the dorsal aspect of the middle lung lobes. Both caudal lung lobe tips were swollen and white and were well demarcated from normal lung tissue. *Marshallagia* spp. were present in the abomasum in small numbers.

CL 4: Lungworm nodules were present in all lung lobes with heavy concentrations and pleural thickening in the dorsal caudal lobes. Adult *P. rushi* were located in large airways.

The gall bladder was small and the wall thickened. The common bile duct was dilated and the wall was very thickened. Two abscesses measuring 1 and 2 cm and containing layered,
greenish, inspissated exudate were within the liver parenchyma adjacent to the gall bladder and bile duct. A large number of *W. tetoni* filled the bile ducts and the proximal duodenum. A few *Marshallagia* spp. were within the abomasal ingesta and the abomasal mucosa was slightly roughened. Multiple 0.1 - 0.2 cm round, white foci were present on the serosal and mucosal surfaces of the cecum and colon. Two 2-3 cm fluid filled cysts, identified as cysticerci, were present on the serosa of the cervix.

**CL 5:** A large number of *P. rushi* adults were present in airways. Lungworm nodules were almost entirely limited to the dorsal caudal lung lobes and many were fused with thickening of the overlying pleura.

A few *W. tetoni* were present in hepatic bile ducts and several *Moniezia* spp. were in the proximal duodenum. Moderate numbers of *Marshallagia* spp. and small numbers of *Trichuris* spp. were in the abomasum and cecum, respectively. Discrete white, linear foci were present on the cecal and colonic serosae. The adrenal glands appeared slightly enlarged.

**CL 6:** Adult *P. rushi* were present in airways. There were lungworm nodules in all lung lobes, with concentrations along the dorsal aspect and the dorsal caudal lobes. A small focus in the cranial right lobe was atelectatic with multiple tiny white foci.

Large numbers of *W. tetoni* filled the bile duct. The duct was greatly thickened and dilated. Several white foci were scattered over the cecal serosa.
5.2.2 Wigwam

Mild lesions of verminous pneumonia were present in five sheep, with WW 5 classified as moderately affected. Opaque, white, individual nodules rather than diffuse swelling of caudal lobes were more common in these animals. Sheep WW 6 was the only animal in which P. ruthi adults were identified.

Chronic bronchopneumonia and fibrous pleuritis were noted in four sheep (WW 1, 3, 4, 5). These ranged from a small area of consolidation of the cranial lobe in WW 5 to bilateral fibrous adhesions and areas of consolidation in the ventral portions of all lobes of WW 4. Consolidation was extensive in both lambs (WW 1 and 3) with sharp demarcation of affected ventral cranial and ventral middle and caudal lobes from the remainder of the lung. The firm, plum-coloured tissue had a cobblestone texture with scattered white foci and a mucoid exudate in the airways. Extensive fibrous pleuritis and pericarditis were also present in both lambs.

Gastrointestinal parasitism lesions were present in all WW sheep and were more severe in those with chronic pulmonary lesions. Sheep WW 4 and 5 had generalized enlargement of lymphoid organs, single white myocardial foci and multifocal pale renal streaking. Contagious eczema lesions were very severe throughout the oral cavity and rumen of WW 4 and mild in WW 5. The lambs had thymic atrophy, serous atrophy of fat, lesions of contagious eczema and those associated with large numbers of gastrointestinal parasites. Lamb WW 3 was more seriously affected with fibrinous peritonitis, serous polyarthritis,
decubitus ulcers and contagious eczema lesions of the coronet bands. The kidney: adrenal ratio ranged from 9.4 to 29.2, with a mean of 21.8. (Table 3). The lowest ratio, or largest adrenal size was in WW 3.

WW 1: Dependent areas of both anterior, middle and anterior caudal lung lobes were collapsed, plum coloured, firm and sharply demarcated from normal tissue. The affected lung had a cobblestone pattern of pale areas with a redder background. There were small areas of suppuration in a distal portion of one lung lobe. Airways were surrounded by shiny white cuffs and contained a mucoid exudate. Fibrous adhesions fixed most of these areas to the parietal pleura and the pericardium.

Small lungworm nodules were present in the most dorsal area of the caudal lobes and singly in two other lobes. Retropharyngeal, bronchial and mediastinal lymph nodes were twice normal size.

The adrenal glands were twice normal size and the thymus was extremely small. The right kidney contained a firm, depressed, demarcated area which extended deep into the cortex.

The bile duct was moderately thickened and dilated and contained mature *W. tetoni*. The abomasal wall was moderately thickened by multiple pale nodules. Numerous *Trichuris* spp. were present in the cecum. The feces were soft and pasty.

WW 2: Small discrete lungworm nodules were present primarily in the dorsal caudal lung lobes. One small area showed fusion of nodules and pleural thickening.
There were moderate numbers of *W. tetoni* in the bile duct. *Marshallagia* spp. were present in large numbers in the abomasum.

**WW 3:** The lungs were similar to those of WW 1 except that the consolidation involved a larger proportion of the lung and there was a less abrupt demarcation from normal tissue. Very few lungworm nodules were present and these were restricted to the dorsal caudal lobes.

The adrenal glands were 4-5 times normal size and the thymus was barely identifiable. The joints under the decubitus ulcers contained increased amounts of thickened joint fluid and the synovia were hyperemic. There were fibrinous and fibrous tags over peritoneal surfaces with an increased volume of serous fluid in the abdomen. The abomasal mucosa was moderately to severely thickened by nodules and *Marshallagia* spp. were in moderate numbers. The feces were soft with a foul odor.

**WW 4:** A small amount of omentum was adherent to the peritoneum and subcutaneous tissue in the middle of the right flank. There were corresponding fibrotic lesions of the abdominal musculature and skin.

There were irregular areas of deep purple, firm, shrunken parenchyma in the dependent portions of all lung lobes. The right lobes were more extensively involved and the right cranial lobe was adherent to the parietal pleura. Small lungworm nodules were scattered over the dorsal caudal lobes.

All visceral and retropharyngeal lymph nodes were enlarged from 2 to 5 times normal size. The spleen was diffusely enlarged to 3 times normal size with prominent, fine, white parenchymal
nodules. There was a 2 cm, firm, round, white swelling at the apex of the left ventricle. The renal cortex contained white linear streaks. There was moderate nodular thickening of the abomasal mucosa. The adrenal glands appeared slightly enlarged.

WW 5: There were scattered small lungworm nodules in most lung lobes. Fibrous pleural adhesions were attached to the distal portions of most lobes. A central area of the left cranial lobe was shrunken, red and firm. The bronchial and mediastinal lymph nodes were enlarged.

An area of myocardium at the apex of the heart was pale. There were multiple pale streaks in the renal cortex which were depressed from the surface. Splenic lymphoid follicles were prominent. An encrusted larval tapeworm was located adjacent to the duodenum and liver. The abomasum contained a trichobezoar and the mucosa was thickened and corrugated. The adrenal glands were enlarged slightly.

WW 6: The dorsal caudal lobes had small areas of coalescing nodules with pleural thickening. Adult *P. rushi* were present within medium-sized airways. There was a moderate number of *W. tetoni* within a slightly dilated bile duct. A few *Marshallagia* spp. were found in the abomasum.

5.2.3 Ewin Ridge

Mild verminous pneumonia was diagnosed in three sheep (ER 1,3,4) and the remainder had mild-moderate (ER 5,6) or moderate (ER 2) lesions. Nodules were mostly small and transparent. The most distal area of the caudal lobes was frequently contracted with pleural fibrosis. Two ewes (ER 5,6) had small, contracted,
consolidated areas in the middle lobes. *Protostrongylus rushi* adults were present within airways in ER 2 and 3.

Gastrointestinal parasitism lesions were restricted to the abomasum in ER 1, 2 and 4 and were moderate in number and severity. No depigmented areas or active contagious eczema lesions were present. Kidney: adrenal ratios varied from 26.1 to 34.2 with a mean of 28.0 (Table 3).

ER 1: Single or small fused lungworm nodules were present only in small areas of the dorsal caudal lung lobes of this lamb. Portions of both lungs were severely damaged from the track of the bullet and were not evaluated.

The abomasal mucosa was moderately nodular. The right ovary was absent and approximately one third of the right uterine horn was present only as a ligamentous structure.

ER 2: A few *P. rushi* adults were found within airways of the caudal lobes of the lung. Fused lungworm nodules covered by thickened contracted pleura were in the distal caudal lobes. Larger discrete nodules were more proximally located with a few small nodules over the dorsal aspect of more cranial lobes. The abomasal mucosa was only slightly nodular.

ER 3: There was a healed wound on the neck over the left prescapular lymph node of this ewe. The lymph node below was enlarged and contained a yellow-white caseous exudate. A few discrete lungworm nodules were present in the caudal lobes only.

ER 4: Lungworm nodules in the young ram were restricted to the distal aspect of the caudal lobes and were fused. The overlying pleura was thickened. Nodular lesions of the abomasal mucosa were prominent.
ER 5: The ewe had linear contracted areas several centimeters from the apex of both middle lung lobes. These areas were firm and red. The pleura of the dorsal caudal lobes was thickened and slightly reddened. It covered diffuse, confluent lungworm lesions. Mild adhesions were present between the left distal caudal lobe and the diaphragm.

ER 6: The left middle lung lobe of the last ewe had an area which was partially contracted, red and firm. Several discrete lungworm nodules were present over the dorsal middle and caudal lung lobes. The dorsal caudal lobes were covered with very thickened pleura and the right distal aspect was markedly contracted.

5.3 HISTOPATHOLOGY

The most consistent histological findings in all sheep from the three herds were lesions caused by hepatic and gastrointestinal cestode and nematode parasitism. The major hepatic changes were fibrosis, mononuclear cell infiltration and hyperplasia of bile ducts in portal regions, as well as small, cellular foci scattered throughout liver lobules (Figure 2). These small foci were often associated with portal areas but were also randomly distributed. The cell type was primarily mononuclear with occasional eosinophils and individual degenerating hepatocytes.

Abomasal inflammation consisted of nodular and diffuse mononuclear cell infiltrates. This lesion was less common in CL sheep. The small intestinal changes were diffuse mononuclear and
eosinophilic infiltrations of the lamina propria. The more severely affected sections also had mucosal hyperplasia. Areas with superficial necrosis were associated with intracellular stages of coccidial organisms, subsequently identified as *Eimeria* spp.. Lesions in the large intestine were less severe than those in the small intestine with less evidence of hyperplasia. Eosinophilic granulomas and focal chronic inflammation, suggestive of parasite migration, were occasionally present in the intestines, liver, lymph nodes and abomasum in sheep from all herds. The cardiac and skeletal muscles of sheep from WW and ER contained cysts of *Sarcocystis* spp..

Kidney changes primarily involved glomeruli or were interstitial with secondary glomerular and tubular effects. Glomerular basement membranes appeared thickened and the mesangium was hypercellular. Severely affected glomeruli had adhesions and were shrunken. None of these lesions were considered to be extensive nor significant enough to cause clinical signs. The most severe kidney lesions were in two sheep from WW and were associated with a neoplasm in one sheep and granulomatous disease in another. Heart lesions noted grossly in these sheep were caused by the same generalized conditions.

Lymphoid tissue was hyperplastic in most animals. Severe systemic illness in a lamb from WW was accompanied by lymphoid atrophy. Systemic disease accompanied lymphadenopathy in two other sheep from WW. The epithelium overlying the pharyngeal lymphoid aggregates was altered in most sheep. These changes varied from hyperplasia and metaplasia to focal necrosis.
nasopharyngeal changes were generally mild or moderate and did not appear to have any relationship to other respiratory pathology.

The pulmonary histopathological changes differed primarily among sheep from the different groups in the amount of lung affected by lungworm activity, the types of tissue response to parasitic life stages and bacteria and the degree and chronicity of bacterial bronchopneumonia. The major changes involved small airways, interstitial tissue and the locally diffuse inflammatory reactions to *P. stilesi* reproduction.

Lesions of bacterial bronchopneumonia in sheep from the three herds were similar. They were characterized by a lobular consolidation and atelectasis and diffuse interstitial thickening. Bronchioles were often distended with mucoid material, sometimes containing neutrophils (Figure 3). A similar material with macrophages was also often within partially collapsed alveoli. Neutrophils were rare, but when present formed small aggregates in airways and alveoli (Figure 4). The bronchiolar submucosa and peribronchiolar tissues contained mononuclear cells in varying numbers. Pleural fibrosis was usually present.

5.3.1. Columbia Lake

Bighorn sheep from the CL herd had histological changes caused by hepatic and intestinal parasitism to a degree similar to those in the other herds (Table 4). Coccidial organisms were present in only one lamb. Renal and abomasal inflammation was mild and rare. There was mild mononuclear cell infiltration of
PLATE 1

Figure 2. Hepatic lesion including portal fibrosis, bile duct hyperplasia (arrows) and mononuclear cell infiltration typical of that found in many sheep. x 16. H & E.

Figure 3. Focal area of bronchopneumonia characterized by lobular consolidation, diffuse interstitial thickening, mononuclear peribronchiolar infiltrations and bronchiolar exudate. x 6.3. H & E.

Figure 4. Small intraalveolar aggregates of neutrophils mixed with mucus and macrophages in bronchopneumonia caused by Pasteurella haemolytica. x 16. H & E.
Table 5.4. Severity of histopathologic changes in the tissues of six bighorn sheep from the Columbia Lake herd in southeastern British Columbia in 1983.

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Sheep Number</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CL 1</td>
</tr>
<tr>
<td><strong>Hepatitis</strong></td>
<td></td>
</tr>
<tr>
<td>- periportal</td>
<td>++</td>
</tr>
<tr>
<td>- focal</td>
<td>+</td>
</tr>
<tr>
<td><strong>Renal</strong></td>
<td></td>
</tr>
<tr>
<td>- glomerulitis</td>
<td>---</td>
</tr>
<tr>
<td>- nephritis</td>
<td>---</td>
</tr>
<tr>
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<td></td>
</tr>
<tr>
<td>- retropharyngeal node</td>
<td>---</td>
</tr>
<tr>
<td>- mediastinal node</td>
<td>---</td>
</tr>
<tr>
<td>- mesenteric node</td>
<td>---</td>
</tr>
<tr>
<td>- spleen</td>
<td>---</td>
</tr>
<tr>
<td>- Feyer’s patches</td>
<td>---</td>
</tr>
<tr>
<td>- nasopharynx</td>
<td>---</td>
</tr>
<tr>
<td>- epithelial changes</td>
<td>---</td>
</tr>
<tr>
<td><strong>Tracheitis</strong></td>
<td>---</td>
</tr>
<tr>
<td><strong>Abomasitis</strong></td>
<td>---</td>
</tr>
<tr>
<td><strong>Parasitic enteritis</strong></td>
<td></td>
</tr>
<tr>
<td>- small intestine</td>
<td>++</td>
</tr>
<tr>
<td>- large intestine</td>
<td>++</td>
</tr>
<tr>
<td><strong>Sarcocystis spp. infection</strong></td>
<td></td>
</tr>
<tr>
<td>- muscle</td>
<td>---</td>
</tr>
<tr>
<td>- heart</td>
<td>---</td>
</tr>
<tr>
<td><strong>Contagious eczema</strong></td>
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</table>

a coccidial organisms present
--- normal
+ mild
++ moderate
+++ severe
the tracheal submucosa in CL 2. Tiny foci of epithelial necrosis were present. There did not appear to be any association of the lesion with other respiratory tract changes or clinical signs in this animal.

There was no evidence of infection by Sarcocystis spp. or lesions suggestive of active contagious ecthyma in this herd. Lymphoid hyperplasia was usually present to a moderate degree.

The lungs of the six CL sheep examined had many small airways which had marked fibromuscular hyperplasia of the muscularis mucosae. The epithelium had frequently undergone squamous metaplasia and the lumen sometimes contained a mucoid exudate, with or without larvae of Protostrongylus spp. nematodes (Figure 5). Mucosal hyperplasia was a relatively uncommon change compared to lung sections from the other herds. Cuffs of mixed lymphocytes and plasma cells frequently surrounded the airways and adjacent vasculature. Activated lymphoid follicles were prominent around airways, vessels and especially within the interstitium.

The interstitial tissue was often thickened, primarily by hyperplasia of fibromuscular elements and considerable fibrosis. Mixed mononuclear cells infiltrated the expanded interstitium.

Acute inflammatory changes were rarely seen in alveolar spaces. Alveoli usually contained differentiated macrophages. The alveolar lining cells were commonly cuboidal, probably type II pneumocytes.

Large numbers of adults, larvae and ova of P. stilesi were present in the CL sheep. The granulomatous inflammatory response
to these life stages consisted of large numbers of eosinophils, lymphocytes and plasma cells in equal proportions, with fewer giant cells (Figure 6). The cell types associated with each life stage did vary but this aspect was not considered separately in this study.

A mild, focal bronchopneumonia was present in CL 6. An identifiable protostrongylid larva was located within the area of reaction.

5.3.2. Wigwam

Parasitic lesions of the liver, abomasum, intestines and muscles were present in all sheep from the WW range (Table 5). Five of six sheep had coccidial organisms visible in sections of small intestine. Renal lesions were present in all sheep; however, the most severe and extensive were in WW 1, 4 and 5 and were associated with primary disease in other organs. Areas of chronic inflammation were noted on gross examination of the kidneys of WW 1. Histologically these were severe infiltrations of mononuclear cells and fibrous connective tissue extending through the cortex. Glomeruli and tubules were obliterated locally. The remaining areas appeared normal except for tubular casts in some tubules.

There was mild mononuclear cell infiltration of the tracheal submucosa in three of five sheep examined. Moderate inflammation of the trachea was present in two sheep. One of these, the lamb WW 1, had an active suppurative tracheitis with epithelial hyperplasia and metaplasia as well as neutrophil and lymphocyte infiltration.
Table 5.5 Severity of histopathologic changes in the tissues of six bighorn sheep from the Wigwam herd in southeastern British Columbia in 1983.

<table>
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<tr>
<th>Lesion</th>
<th>Sheep Number</th>
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<tr>
<td></td>
<td>WW 1</td>
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<tr>
<td>Hepatitis</td>
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<td>- muscle</td>
<td>---</td>
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<td>- heart</td>
<td>+</td>
</tr>
<tr>
<td>Contagious ecthyma</td>
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</table>

\(a\) coccidial organisms present  
\(b\) lymphoid tumor  
\(c\) granulomatous reaction  
\(d\) lymphoid atrophy  
NE not examined  
--- normal  
+ mild  
++ moderate  
+++ severe
Lesions characteristic of CE were identified in four sheep. All lesions examined had a similar histologic appearance, regardless of location on the mucocutaneous junctions of either the mouth or coronets, or the mucosal surfaces of the oral cavity and rumen. The lesions of keratinized epithelium were covered by a crust of cellular debris consisting of epithelial cells, necrotic neutrophils and occasional bacterial colonies. Focal areas were eroded or ulcerated. Hyperkeratosis, parakeratosis and severe ballooning degeneration of the superficial cell layers were the predominant cellular changes. Occasional large irregular eosinophilic intracytoplasmic inclusion bodies were present. Bullous-like clefts within the epidermis were filled with serum. Neutrophils were often present within the epidermis and dermis, and were occasionally aggregated into intraepithelial microabscesses. The dermal papillae and basal epidermal cell layers were usually edematous. Nonkeratinized epithelial lesions were also proliferative in nature, but usually lacked the acute inflammatory changes. Varying numbers of mononuclear inflammatory cells surrounded the adjacent microvasculature and adnexal structures.

Lymphoid hyperplasia was generalized in five sheep. Lymphoid atrophy was noted in all locations except the nasopharynx in WW 3. There was a replacement of normal lymph node architecture by a monotonous population of mononuclear cells with large round nuclei in WW 4. Infiltrates of similar cells were present in the kidney, spleen, heart, abomasum, retropharyngeal, mediastinal and mesenteric lymph nodes (Figure
The nuclei contained reticulated chromatin and prominent multiple nucleoli were frequent. Many of these cells were very anaplastic. There was an average of 2-3 mitoses per high power field. Since these cells were present in vessels in numerous organs, the condition was diagnosed as leukemic lymphosarcoma.

There were multiple areas of granulomatous inflammation present in the heart, liver, kidney, spleen, small intestine and retropharyngeal lymph node of WW 5 (Figure 8). All areas were similar with accumulations of epithelioid macrophages interspersed with lymphocytes and plasma cells. Some areas had centers of necrotic, often mineralized material. Multinucleate giant cells were occasionally present. Small arteries were sometimes surrounded by mononuclear cell cuffs and there were fibrinoid changes in the walls of several vessels of the renal pelvis.

The cytoplasm of some macrophages and giant cells was granular and multiple, gram positive, mildly refractile organisms measuring 1 x 2 um were present (Figure 9). They were more obvious when viewed with phase contrast microscopy (Figure 10). The size, staining and refractile nature of the organisms and the accompanying histological changes caused them to be tentatively identified as microsporidia, possibly Encephalitozoon cuniculi; however, ultrastructural morphology and cross-infection trials would likely be necessary for confirmation.

Chronic moderate to severe bronchopneumonia with fibrous pleuritis were present in WW 1, 3, 4 and 5. Interstitial tissue was thicker in WW sheep, but chronic inflammatory changes of
alveolar cell hyperplasia and the accumulation of macrophages were decreased. Acute inflammation with necrosis and neutrophil accumulation was rare.

5.3.3. Ewin Ridge

There were parasitic lesions in the liver, abomasum, intestines and muscles of most of the sheep from the ER herd (Table 6). The small intestines of four sheep contained coccidial organisms. Inflammatory changes in the kidney and trachea were mild and sporadic. There were no lesions of active contagious eczema. All sheep had some degree of generalized lymphoid hyperplasia.

Protozoa were found within the pharyngeal submucosa or within an artery adjacent to the retropharyngeal lymph node of ER 4, 5 and 6 (Figure 11). They appeared to be identical to organisms present within dilated abomasal glands of ER 4 (Figure 12). The protozoa were oval ciliates with a large, prominent macro-nucleus. They were usually accompanied by focal epithelial and lymphoid tissue necrosis. I am unaware of a similar tissue invasive protozoan spp. and feel that the localized inflammatory reactions rule out the possibility of the presence of the protozoa being an artifact.

Lungs from ER sheep were most similar to those of CL bighorns. Airway pathology also included a large amount of fibromuscular hyperplasia of the muscularis mucosae. Epithelial hyperplasia and metaplasia of the airway mucosa was a more common change than in lungs from CL sheep. Lymphoid cuffs and follicles also surrounded vessels and airways but were less common in the
Figure 5. Fibromuscular hyperplasia of small airway in lung infected with Protostrongylus stilesi. Squamous metaplasia of airway mucosal cells was common in these lungs. x 16. H & E.

Figure 6. Chronic inflammatory response to various life stages of Protostrongylus stilesi. x 6.3. H & E.

Figure 7. Diffuse infiltration of myocardium of sheep WW4 by large atypical lymphoid cells. x 16. H & E.

Figure 8. Granulomatous foci in renal cortex of sheep WW5. Central mineralized areas are surrounded by giant cells, epithelioid cells, lymphocytes and plasma cells. x 6.3. H & E.
Table 5.6. Severity of histopathologic changes in the tissues of six bighorn sheep from the Ewin Ridge herd in southeastern British Columbia in 1984.

<table>
<thead>
<tr>
<th>Lesion</th>
<th>ER 1</th>
<th>ER 2</th>
<th>ER 3</th>
<th>ER 4</th>
<th>ER 5</th>
<th>ER 6</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hepatitis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- periportal</td>
<td>++</td>
<td>++</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>- focal</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td><strong>Renal</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- glomerulitis</td>
<td>---</td>
<td>+</td>
<td>---</td>
<td>---</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>- nephritis</td>
<td>---</td>
<td>---</td>
<td>+</td>
<td>---</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Lymphoid hyperplasia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- retropharyngeal node</td>
<td>++</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>+</td>
<td>b</td>
</tr>
<tr>
<td>- mediastinal node</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>- mesenteric node</td>
<td>++</td>
<td>---</td>
<td>+</td>
<td>++</td>
<td>+</td>
<td>---</td>
</tr>
<tr>
<td>- spleen</td>
<td>++</td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td>++</td>
<td>---</td>
</tr>
<tr>
<td>- Peyer's patches</td>
<td>++</td>
<td>---</td>
<td>++</td>
<td>++</td>
<td>+</td>
<td>---</td>
</tr>
<tr>
<td>- nasopharynx</td>
<td>---</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>- epithelial changes</td>
<td>---</td>
<td>+</td>
<td>---</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td><strong>Tracheitis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Abomasitis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Parasitic enteritis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- small intestine</td>
<td>+++a</td>
<td>+++a</td>
<td>+++a</td>
<td>+++a</td>
<td>+++a</td>
<td>+++a</td>
</tr>
<tr>
<td>- large intestine</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td><strong>Sarcocystis spp. infection</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- muscle</td>
<td>---</td>
<td>+++</td>
<td>---</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>- heart</td>
<td>---</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td><strong>Contagious eczthyma</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*a* coccidial organisms present

*b* invasive protozoa present

--- normal

+ mild

++ moderate

+++ severe
interstitium. The interstitial tissue changes in ER lungs were very similar to CL sheep except for a much lower concentration of lymphoid cells and follicular development within the interstitium. The alveoli contained even larger numbers of macrophages and were more often lined by metaplastic epithelium. The number of lungworm adults seen in ER lungworm nodules was half that found in CL sheep, with similar numbers of larvae and ova. The inflammatory cell types within lungworm granulomas showed a slightly higher proportion of giant cells, but was otherwise of equal numbers of eosinophils, lymphocytes and plasma cells.

Resolved focal bronchopneumonia was present in ER 5 and 6. There was no involvement of the pleura in either case and few remaining inflammatory cells.

5.4 LABORATORY ANALYSES

5.4.1 Microbiology

No significant pathogenic organisms were cultured from lymph nodes, respiratory tissues or swabs from CL sheep. A pure culture of low numbers of *Escherichia coli* was recovered from the hepatic abscess in CL 4. *Pasteurella haemolytica* biotype T of a nonhaemolytic variety was recovered from the bronchial swabs and right cranial lung of WW 1 and the retropharyngeal lymph node of WW 4. A haemolytic non T biotype of *P. haemolytica* was cultured from the bronchial swabs, mediastinal lymph node and left cranial lung of WW 4. *Staphylococcus aureus* was recovered from the mediastinal
lymph nodes and bronchial swabs of WW 4, retropharyngeal lymph nodes of WW 3, 4 and 5 and joint swabs of WW 3. All other lung cultures from WW sheep were negative or produced insignificant contaminants.

The retropharyngeal lymph node and right and left middle lung lobes of ER 5 contained a nonhaemolytic P. haemolytica biotype T, biochemically similar to the WW1 and WW4 isolates. There were no other significant organisms recovered from ER sheep.

No viruses were recovered from any of the sheep tissues cultured. Positive titres to PI3 virus were found in serum from WW 2 and 6 and ER 2, 5 and 6. Sheep ER 6 also had a weakly positive titre to RSV virus. All sera were negative to IBR, BVD, Johne’s disease, maedi/visna, EHD and BT viruses. There was no antibody to bovine lymphosarcoma virus in sera from WW 4.

5.4.2 Parasitology

Fecal flotations indicated negative to very high levels of nematode and/or protozoal gastrointestinal parasitism (Table 7). Ova of Marshallagia, Nematodirus and Trichuris spp. and oocysts of Eimeria spp. were recovered, often in mixed infections. The levels were higher in younger animals, particularly those with systemic illnesses (WW 1 and 3).

The number of Protostrongylus spp. larvae present in feces was higher in CL sheep than in WW or ER sheep, with average numbers of larvae per gram of feces of 799 (CL), 162 (WW) and 166 (ER). Lambs and yearling bighorns from CL had the highest larval outputs, while the lambs from WW and the lamb and yearling from
ER had very few larvae in their feces. The WW yearling had moderate numbers of larvae.

5.4.3 Trace Minerals

The selenium level in liver tissues from all CL sheep was less than or equal to 0.11 nmoles/g, levels that are considered deficient in domestic sheep (Table 7). Liver of sheep WW 1, 4 and 5 contained selenium in levels considered marginal. All ER bighorn sheep appeared to have adequate levels of selenium (Table 7).

Tissue copper was at a marginal level in CL 2 and 5, WW 2, 3 and 4 and ER 1, 2, 5 and 6 (Table 7). Zinc levels in liver were marginal in CL 6, WW 1, 3, 5 and 6 (21-29 umoles/g).

5.4.4 Chemistry

Blood urea nitrogen values were within normal domestic sheep levels in only two CL (CL 2 and 3) and two ER sheep (ER 1 and 6) (Table 8). Values were higher than normal in WW 1, 3 and 4. The remainder of bighorn sheep in all three herds had lower than normal values. Serum glucose was mildly to moderately increased in five CL, four WW and four ER sheep. Two ER sheep had extremely high glucose levels.

Total serum protein levels were reduced in four CL, four WW and two ER bighorn sheep. The average values were highest in the ER herd and lowest in the CL herd. Fecal nitrogen was very consistent in the CL and ER sheep herds with average values of 1.0 and 1.8% respectively. The fecal nitrogen content varied more among WW sheep, especially in those with clinical diseases (WW 1 and 3). The average level for this herd was 1.1%.
Figure 9. Gram positive organisms (1 x 2 um) (arrow) within renal tubular cells of sheep WW5. Brown and Brenn stain.

Figure 10. Same section under polarized light. The organisms are slightly birefringent. x 60. Brown and Brenn stain.

Figure 11. Ciliated protozoa (arrows) within the pharyngeal submucosa of sheep from Ewin Ridge. These parasites were usually associated with focal epithelial and lymphoid cell necrosis. x 40. H & E.

Figure 12. Ciliated protozoan within dilated abomasal gland of sheep ER 4. x 100. H & E.
Table 5.7. Number of lungworm larvae per gram of feces and copper and selenium concentrations (wet weight basis) in liver of bighorn sheep collected from the East Kootenay region in 1983-84.

<table>
<thead>
<tr>
<th>Sheep</th>
<th>Larvae/g of feces</th>
<th>Copper (umole/g)</th>
<th>Selenium (nmol/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CL 1</td>
<td>172</td>
<td>1.21</td>
<td>1.27</td>
</tr>
<tr>
<td>CL 2</td>
<td>665</td>
<td>0.22</td>
<td>1.27</td>
</tr>
<tr>
<td>CL 3</td>
<td>1971</td>
<td>1.05</td>
<td>0.76</td>
</tr>
<tr>
<td>CL 4</td>
<td>543</td>
<td>0.68</td>
<td>0.89</td>
</tr>
<tr>
<td>CL 5</td>
<td>959</td>
<td>0.31</td>
<td>1.39</td>
</tr>
<tr>
<td>CL 6</td>
<td>484</td>
<td>1.15</td>
<td>0.76</td>
</tr>
<tr>
<td>x</td>
<td>799 + 628&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.77 ± 0.50</td>
<td>1.06 ± 0.26&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>WW 1</td>
<td>9</td>
<td>1.43</td>
<td>2.03</td>
</tr>
<tr>
<td>WW 2</td>
<td>58</td>
<td>0.25</td>
<td>4.05</td>
</tr>
<tr>
<td>WW 3</td>
<td>3.5</td>
<td>0.27</td>
<td>3.67</td>
</tr>
<tr>
<td>WW 4</td>
<td>167</td>
<td>0.10</td>
<td>3.17</td>
</tr>
<tr>
<td>WW 5</td>
<td>321</td>
<td>0.58</td>
<td>2.41</td>
</tr>
<tr>
<td>WW 6</td>
<td>414</td>
<td>0.99</td>
<td>7.73</td>
</tr>
<tr>
<td>x</td>
<td>162 + 172&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.60 ± 0.66</td>
<td>3.84 ± 2.85&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>ER 1</td>
<td>13</td>
<td>0.10</td>
<td>3.80</td>
</tr>
<tr>
<td>ER 2</td>
<td>347</td>
<td>0.13</td>
<td>3.42</td>
</tr>
<tr>
<td>ER 3</td>
<td>142</td>
<td>0.50</td>
<td>4.05</td>
</tr>
<tr>
<td>ER 4</td>
<td>51</td>
<td>0.37</td>
<td>3.80</td>
</tr>
<tr>
<td>ER 5</td>
<td>109</td>
<td>0.13</td>
<td>3.67</td>
</tr>
<tr>
<td>ER 6</td>
<td>336</td>
<td>0.09</td>
<td>3.67</td>
</tr>
<tr>
<td>x</td>
<td>166 + 143&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.22 ± 0.20</td>
<td>3.73 ± 0.32&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Normal<sup>a</sup> deficient 0.01 - 0.06  0.06 - 1.27
marginal 0.08 - 0.31  1.90 - 3.17

<sup>a</sup> Concentration of copper and selenium considered marginal and deficient in liver of domestic sheep (R. Puls 1981)
<sup>a, b</sup> Values with different superscript letters are significantly different
Table 5.8. Serum, fecal and urine chemistry values in bighorn sheep collected from the East Kootenay region in 1983-84.

<table>
<thead>
<tr>
<th>Sheep number</th>
<th>Blood Urea Nitrogen (mmol/L)</th>
<th>Total Protein (g/L)</th>
<th>Fecal Nitrogen (%)</th>
<th>Glucose (mmol/L)</th>
<th>Cortisol (ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>serum</td>
</tr>
<tr>
<td>CL 1</td>
<td>1.0</td>
<td>50</td>
<td>0.9</td>
<td>4.3</td>
<td>3.6</td>
</tr>
<tr>
<td>CL 2</td>
<td>3.9</td>
<td>57</td>
<td>1.0</td>
<td>4.3</td>
<td>7.2</td>
</tr>
<tr>
<td>CL 3</td>
<td>4.1</td>
<td>46</td>
<td>1.0</td>
<td>3.9</td>
<td>&lt; 1</td>
</tr>
<tr>
<td>CL 4</td>
<td>1.4</td>
<td>61</td>
<td>1.0</td>
<td>3.9</td>
<td>&lt; 1</td>
</tr>
<tr>
<td>CL 5</td>
<td>1.0</td>
<td>42</td>
<td>1.1</td>
<td>3.6</td>
<td>9.5</td>
</tr>
<tr>
<td>CL 6</td>
<td>0.4</td>
<td>64</td>
<td>1.1</td>
<td>2.9</td>
<td>12.8</td>
</tr>
<tr>
<td>x</td>
<td>1.9+1.6</td>
<td>53.3+8.7</td>
<td>1.0+0.1b</td>
<td>3.8+0.7</td>
<td>ND</td>
</tr>
<tr>
<td>WW 1</td>
<td>8.9</td>
<td>57</td>
<td>1.8</td>
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<td>15.6</td>
</tr>
<tr>
<td>WW 2</td>
<td>2.7</td>
<td>60</td>
<td>0.8</td>
<td>4.0</td>
<td>3.1</td>
</tr>
<tr>
<td>WW 3</td>
<td>6.9</td>
<td>61</td>
<td>0.5</td>
<td>2.9</td>
<td>23.8</td>
</tr>
<tr>
<td>WW 4</td>
<td>10.2</td>
<td>46</td>
<td>1.4</td>
<td>4.5</td>
<td>0.3</td>
</tr>
<tr>
<td>WW 5</td>
<td>1.2</td>
<td>58</td>
<td>1.1</td>
<td>3.3</td>
<td>1.0</td>
</tr>
<tr>
<td>WW 6</td>
<td>1.2</td>
<td>58</td>
<td>0.9</td>
<td>6.0</td>
<td>1.2</td>
</tr>
<tr>
<td>x</td>
<td>5.2+4.5</td>
<td>56.7+5.4</td>
<td>1.1+0.5b</td>
<td>4.0+1.3</td>
<td>ND</td>
</tr>
<tr>
<td>ER 1</td>
<td>3.5</td>
<td>78</td>
<td>2.0</td>
<td>3.3</td>
<td>5.9</td>
</tr>
<tr>
<td>ER 2</td>
<td>1.3</td>
<td>66</td>
<td>1.8</td>
<td>6.0</td>
<td>22.4</td>
</tr>
<tr>
<td>ER 3</td>
<td>0.7</td>
<td>63</td>
<td>1.7</td>
<td>34.7</td>
<td>2.6</td>
</tr>
<tr>
<td>ER 4</td>
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<td>56</td>
<td>1.8</td>
<td>2.6</td>
<td>16.9</td>
</tr>
<tr>
<td>ER 6</td>
<td>3.8</td>
<td>57</td>
<td>1.8</td>
<td>18.1</td>
<td>32.1</td>
</tr>
<tr>
<td>x</td>
<td>2.1+1.5</td>
<td>63.5+8.0</td>
<td>1.8+0.2a</td>
<td>11.6+16.1</td>
<td>ND</td>
</tr>
</tbody>
</table>

NE - not examined
ND - not determined
a,b - values with different superscript letters are significantly different
Serum and urine cortisol values varied greatly within herds without a consistent pattern. Kidney : adrenal ratios were similar in CL and ER bighorn sheep but were lower on average in WW sheep, indicating larger adrenal size in sheep from the WW range (Table 3).

6.0 DISCUSSION

The CL herd was expected to be present in large numbers on its small traditional range during the winter of 1983. There were reports of respiratory disease and reduced numbers of bighorn sheep in other herds to the southeast of Columbia Lake during the previous winter. It was believed that some of these herds shared summer ranges or migration routes with CL sheep, and that contact and transmission of respiratory pathogens between herds was very likely to occur. In addition, the CL winter range was considered to have been badly overgrazed for many years; however, individual sheep had shown no signs of inadequate nutrition. Annual lamb:ewe ratios had been decreasing, but lambs were of large size and ratios remained higher than those of other E.K. herds. Despite the suspicion that these factors could predispose the CL herd to a disease outbreak in 1982 or 1983, there was no indication of clinical or subclinical disease in these animals.

In this study, the CL bighorn sheep occupied their winter range at a high density. Animals were in large groups and appeared to remain calm, moving little even immediately following
disturbances. This behaviour had been typical of bighorn sheep in this location. Clinical signs expected to distinguish animals with disease or decreasing body condition were restricted to slight variations in the degree of body muscling, fat deposition and coat colour. A single ewe had a mild cough. The field observations alone suggested that the animals in the CL herd were in good health.

In contrast, there appeared to have been little increase in the WW population following the dieoff. Occasional clinical disease had been reported prior to the study observations and very few lambs were identified during the spring counts. The winter range had appeared to improve with the reduced density of both sheep and competitive ungulate species. The WW herd was suspected to have experienced the most severe effects of the pneumonia dieoff, and it was believed that the herd was in, or about to enter a phase of increased population growth with minimal danger of further health problems.

The WW sheep were present in scattered small groups on the large open winter range when evaluated for the study. They were flighty and difficult to observe and approach, even without disturbance. Prior to the 1981-82 dieoff, observations made of this herd confirmed that they grazed as large groups which tolerated human approach in a manner similar to the CL sheep. There appeared then, to have been a radical change in the behaviour of sheep on the WW range. It is possible that the reduction in the total number of WW sheep increased both the nervousness and flight distance, and that the difference in
behaviour between the two herds was a simple reflection of herd density and range topography.

The clinical signs in sheep sampled from WW included pale coats, tick infestation, obvious loss of condition and weakness, nasal discharge, diarrhea and active contagious ecchyma lesions in all ages. The only lambs observed were collected because of combinations of all of the above signs. It was concluded that there was a high proportion of WW sheep in obvious poor health, and that lamb mortality was close to 100% by the time the lambs were 6 months of age. The WW herd appeared to be suffering considerable residual effect of the dieoff, and it appeared that any recovery growth phase would be delayed.

Ewin Ridge bighorn sheep had remained at a relatively stable population level for at least the past 13 years. They primarily utilized accessible snow free areas as traditional winter range. Animals were grouped on slopes where dispersal was limited by the amount of surrounding snow accumulation. Previous observations had not described large scale mortality, clinical disease or any signs of reduced body condition. This herd did not suffer from the periodic pneumonia dieoffs seen in the other E. K. sheep herds, but there was no further information available on their health.

The use of a helicopter in the study prevented the visualization of natural groupings and clinical signs at the time of collection. The lamb and yearling examined were small compared with their age classes in the other herds, but the carcasses were in good condition. No other clinical or external
signs were apparent and no conclusions were made from the data available on the ER sheep prior to necropsy. The ER herd did not appear to exhibit any obvious health problems.

The necropsies of study sheep allowed more accurate estimations of body condition than external examination of live animals or unopened carcasses. This will be discussed below. It also allowed assessment of the degree of pulmonary involvement by both parasitic and bacterial pneumonic processes and the presence of other systemic diseases.

The most common gross pathologic lesions in sheep collected from all three herds were parasite-induced focal granulomatous pneumonia, diffuse chronic gastroenteritis, miliary hepatitis and biliary cestodiasis. Microscopic parasitic lesions in the gastrointestinal tract and liver were similar in type and varied only in degree in all sheep. Lesions and parasitic species were found by histopathology but not always identified during the gross examinations and fecal evaluation of each animal. There appeared to be little correlation between the gross appearance and the severity of histologic lesions. These widespread changes in the gastrointestinal tract and liver appeared to have minor effects on animal condition.

Gross examination indicated that the lungs of CL sheep of all ages were the most severely affected by lungworm infection. The lesions associated with P. stilesi in ER sheep lungs were not as extensive and WW sheep were little affected by this parasite.

The histopathology of lungs from sheep from ER demonstrated more airway epithelial hyperplasia and metaplasia, increased
alveolar epithelial metaplasia and higher numbers of alveolar macrophages than lungs of sheep from CL. In addition, less lymphoid tissue and follicular development was present around airways and within the lung interstitium of sheep from ER. Pulmonary granulomas in these sheep also contained adult lungworms and more giant cells. Sheep from WW had markedly fewer lungworms and accompanying inflammation, as well as fewer chronic changes associated with the parasite (i.e. fibromuscular hyperplasia).

The most remarkable difference between the histopathology of CL and ER sheep lungs and that of WW sheep was the paucity of lungworm stages and accompanying inflammation in the latter herd. No adult *P. stilesi* were present in sections examined from sheep from WW. Fewer inflammatory cells were present in lungworm granulomas in this herd, compared to CL and ER. These lungs had a markedly reduced amount of fibromuscular hyperplasia, particularly surrounding airways, but also within the interstitium. Less interstitial fibrosis was also noted.

Airway epithelium in WW sheep was markedly more hyperplastic and more often contained exudate than in CL or ER sheep. Mucosal metaplasia, however, was slightly less common than in CL sheep, and only half as common as lungs from ER bighorns. Lymphoid tissue around airways and in the interstitium was as common as in CL sheep, but activated follicles in all locations were rare.

The increased degree of histopathological epithelial changes in the airways and alveoli of lungs from ER sheep was indicative of chronic irritation of these surfaces. This type of injury
could result from the presence of large numbers of protostrongylid larvae moving proximally from more distal air passages, as would occur following their maturation in the pulmonary parenchyma. Together with the reduced amount of activated lymphoid tissue and large amount of grossly visible pleural fibrosis, these changes suggest that the ER sheep were in a resolving phase of prior parasitic lung injury. The gross and histologic changes in the CL sheep were believed to indicate chronic but active parasitic injury.

These microscopic pulmonary changes together with information on fecal larvae numbers were considered to support the gross estimations of lungworm infection severity and chronicity in the three herds. The assessment of the degree of lungworm involvement also matched the clinical picture in each herd. Columbia Lake sheep undoubtedly harboured *P. stilesi* infections year round; however, they may also have been subject to heavy reinfection when concentrated on the small, shortcropped winter pasture. Ewin Ridge sheep may also suffer from high levels of lungworm at certain times of the year, but are less highly concentrated on a winter range of better quality. Less than ideal conditions for larval survival at this altitude, less contamination of the range because of lower sheep density and lower availability of larvae due to better forage condition are likely all responsible for reducing the level of infection at this season and location. At the time of this study, WW bighorn sheep were widely dispersed on a range of improving quality. The sheep in this herd with heavy lungworm burdens probably succumbed
during the previous dieoff. These factors may account for the low lungworm infection rate on this range.

Gross and microscopic tissue changes caused by chronic bacterial pneumonias were most severe and extensive in WW lambs. The chronic bacterial pneumonias of adult sheep from all three ranges varied in severity and extent. There was localization to single lung lobes in CL and ER ewes. The mild chronic bacterial pneumonia seen in ewes from ER appeared extremely well localized without secondary lesions elsewhere, and were present in what appeared to be clinically normal animals. The focal pneumonia in the single CL ewe was acute, but was also well localized and the animal appeared clinically normal.

The localized pneumonias in CL and ER ewes suggested an association with focal injury and inflammation. This type of lesion can result from the aspiration of protostrongylid larvae produced from established lungworm nodules in more distal areas of the lungs. Fragments of larvae surrounded by focal granulomas or secondarily invaded by opportunistic bacteria have been present, usually in the anterior lung lobes, as clinical or incidental lesions in many bighorns at necropsy (Spraker 1979). The presence of lungworms has historically been considered to predispose the sheep to the colonization of respiratory pathogens (Marsh 1938, Post 1962, Forrester 1971). Focal granulomas presumed to be caused by larvae were occasionally seen in this study; however, the foci were small and usually without changes related to secondary bacterial infection. The focal pneumonia in CL6 was the only such lesion visible grossly and it did contain larval remnants. No bacteria were cultured from this area.
In contrast, pneumonia in WW ewes, when present, affected more than one lung lobe, extended into the pleural cavity and appeared to be of longer duration. These animals were in poor condition or had signs of disease in other body systems. Microscopic changes in chronic bacterial pneumonias were, however, similar in all herds and varied only in the amount of fibrous tissue present or the age of the lesion.

Bighorn feces have been examined for lungworm LPG to determine parameters such as mean LPG, LPG seasonal and annual trends and the proportion of heavily infected sheep per herd to estimate herd infection levels (Uhazy et al. 1973, Holmes and Samuel 1974). These studies and others (Forrester and Senger 1964, Samson and Holmes 1985) have concluded that individual and herd lungworm larvae output are dynamic and undergo seasonal and annual trends. Single fecal LPG counts can only give rough estimates of herd infection levels.

_Pasteurella haemolytica_ type T, a bacterium strongly associated with the recent B.C. and Alberta dieoffs (Onderka and Wishart 1984), was present in a lamb from WW with active pneumonia and was cultured from lymphoid and lung tissues of WW 4 and ER 5, both of which were ewes with chronic pneumonia. The presence of the organism in the lung of WW 4, in association with _P. haemolytica_ and _Staphylococcus aureus_, also an opportunistic pathogenic bacterium, may be incidental since the functional capacity of the immune system of this ewe may have been compromised by a systemic lymphocytic tumor. The presence of the unique T biotype in the focal, mild, resolving lesion in an
otherwise healthy ER ewe, supports the hypothesis that *P. haemolytica* type T is an opportunistic pathogen (Onderka and Wishart 1984), and that pneumonia can occur and resolve in individual bighorn sheep without outbreaks of disease.

The high prevalence of chronic diseases, secondary infections, contagious ecthyma, and the continued presence of bacterial lesions within the lungs of adult and juvenile WW sheep in 1983 strongly suggests residual effects of stressors associated with the acute dieoff in 1982. Spraker et al. (1984) described the mortality of bighorn lambs in captive and free-ranging herds during years following all-age dieoffs. Lambs less than 6 months of age in their study died with bronchopneumonia caused by opportunistic bacteria and showed marked atrophy of the thymus gland. The presence of thymic and lymphoid atrophy, secondary infections, contagious ecthyma and high gastrointestinal parasitism led to the diagnosis of immunosuppression in the young WW animals in this study. Spraker et al. (1984) suggested that stressors similar to those which precipitated all-age dieoffs may continue to affect pregnant ewes. Continuous high levels of glucocorticoids secreted by an highly stimulated adrenal gland may inhibit the development of the thymus and immune system of the fetus. In addition, the steroids may restrict the production of colostral antibodies. Without a fully functional immune system and the passive immunity provided by the colostrum, the neonatal lamb would be extremely vulnerable to opportunistic microorganisms. Adult sheep harbouring chronic respiratory or gastrointestinal infections
could easily transmit large doses of pathogens to susceptible lambs. The active contagious ecchyma in combination with systemic conditions involving the immune system in two ewes from WW may indicate immunosuppression in these sheep as well.

Longterm stimulation by physical and environmental stresses can cause hyperplasia of the adrenal cortex and enlargement of the gland. In this study, estimates of adrenal gland size were made using kidney:adrenal weight ratios. Since kidney weight varies little with body condition, variations in adrenal size present with age, sex and physical condition were reduced with the use of the ratio. Adrenal glands of WW sheep were significantly larger than those from CL sheep suggesting that these sheep were subject to greater degrees of stress factors. The pattern of lamb and adult bighorn disease and mortality in the WW herd then, appears to fit the pathogenesis of disease as suggested by Spraker et al. (1984) for bighorn herds in a post-dieoff situation.

Serological results and the lack of diagnostic histological evidence in this study suggested sporadic or no exposure of the E.K. bighorn sheep to PI3, IBR, RSV, BT, EHD, BVD, maedi/visna or Johne’s disease. It was of note that a ewe (ER 5) from ER with a resolved pneumonia and a ewe (WW 2) and a yearling (WW 6) from WW without lesions of bronchopneumonia not only had antibodies to PI 3, but also had lesions of nonsuppurative tracheitis. Three other sheep from WW with chronic bronchopneumonia and one from CL without bacterial pneumonia demonstrated tracheitis without positive titres to PI 3. In the cases from WW the tracheitis
likely resulted from extension of the bacterial pneumonias. Other causes of tracheal irritation, such as inhaled dust particles or heavy lungworm larvae activity may explain the tracheitis in CL 2. On this evidence, we cannot totally rule out viral agents as contributing to mild upper respiratory tract damage.

It is recognized that the soils of the E. K. are generally deficient in selenium and that deficiency syndromes occur in domestic animals grazing on pasture in this area (Puls pers. commun.). Hebert and Cowan (1971) examined mountain goats, forages and mineral licks from the E.K. area and suggested that animals were predisposed to myopathy (white muscle disease) by low levels of dietary selenium. Eastman et al. (1971) determined that copper levels in forage plants common on low elevation winter ranges in the E.K. were especially low.

The nutritional requirements of bighorn sheep for trace minerals is unknown and may not be identical to those of domestic sheep, however, values for the latter were used in this study as normals in the absence of specific information.

Copper levels in liver were judged to be marginal in some animals in all three herds, but not in all sheep from any herd. Marginal selenium levels in some WW sheep and deficient levels in all CL sheep suggest that selenium deficiency related disorders may occur in these two herds although no clinical signs of deficiency were observed. Similar levels of copper and selenium were present in tissues from some WW bighorn sheep which died in the E.K. dieoff in 1981 (Davidson 1982). Since that time,
mineral licks in WW have been supplemented with a domestic sheep trace mineral mix. It is possible that those sheep collected from the WW range had artificially elevated levels of trace minerals and that deficiencies may have played a more important role in the herd prior to supplementation. The consistently higher levels of selenium present in ER bighorn sheep supports the findings of Hebert (1973) that alpine forages contain higher levels of trace minerals than do lower elevation species and suggests that these sheep do not suffer from selenium deficiency.

Serum chemistry profiles for healthy captive and wild bighorn sheep (Davies 1976, Franzmann 1971a, Franzmann and Thorne 1970, Woolf and Kradel 1970), stone sheep (Franzmann 1971b), Dall sheep (Foreyt et al. 1983) and Nelson desert sheep (McDonald et al. 1981), as well as for sick captive and wild bighorn sheep (Woolf and Kradel 1973, Spraker et al. 1984) have been examined. In this study sheep were shot, usually without prior disturbance. Samples were expected to reflect actual physiologic values in CL and WW sheep, while the use of a helicopter at ER was expected to cause increases in parameters affected by acute stress, such as serum cortisol and glucose (Franzmann and Thorne 1970), but not total serum proteins and blood urea nitrogen. The chemistry values obtained during the study were compared to the previously reported levels for wild sheep species as well as to those for domestic sheep.

Glucose values in bighorn sheep from the E.K. were similar to those reported in bighorn sheep elsewhere as well as to levels in domestic sheep. Mildly to extremely increased levels were
present in some sheep, and were probably caused by excitement prior to collection. The highest levels occurred in sheep from ER herded with a helicopter. Glucose levels also vary with habitat type; however, without forage evaluation, no habitat quality assumptions may be made from this data (Franzmann 1972).

Blood and urine cortisol values varied too greatly for accurate interpretation. Serum cortisol levels were high in some disturbed, clinically ill and chased sheep, although these findings were not consistent and no relationship to the size of adrenal glands in individual animals was evident. Urine cortisol levels should not have been affected by acute stresses; however, they were not available from all animals and the values obtained had not been corrected for urine concentration. There was a trend towards higher cortisol values in the order WW > CL > ER which does fit the general assessments of this study; however, the diagnosis of stress levels by this method requires much more rigorous sampling technique, larger sample sizes and further research before proper assessments can be made.

The TP of the sheep from ER was within normal limits for domestic and bighorn sheep. It was decreased in CL juveniles and low normal in CL adults. The sick WW lambs had normal TP in spite of their poor body condition, but the TP in these lambs may have been artificially elevated due to dehydration and/or high circulating immunoglobulin levels in response to systemic infections. Other WW sheep had levels of TP in the low normal range, except for low levels in the ewe with lymphosarcoma.
The levels of BUN were low in the majority of the study sheep. Mild increases were present in sick WW sheep, likely due to body protein catabolism. Levels similar to those in CL, WW and ER sheep have been reported in healthy and sick bighorns (Franzmann and Thorne 1970, Spraker et al. 1984) and it was suggested that the levels reflected the low protein content of forages. Many of the apparently healthy sheep in good condition from CL and ER ranges had BUN values far below those previously reported. No explanation was apparent for this.

The FN values obtained from bighorn sheep in this study were comparable to the range of values for bighorn sheep on native forage during October to December (Hebert et al. 1984). The FN was significantly higher in ER sheep than in the other groups and corresponded with levels in migratory sheep seen in that study. Values in WW and CL sheep were similar and matched the lowest levels that were found during winter months in sheep evaluated by Hebert et al. (1984). Variations in FN of sick WW animals were likely due to nitrogen losses from systemic illness.

Body condition scores at necropsy were high for both CL and ER sheep but were low for WW sheep. The scores gave only a rough indication of overall animal condition and probably reflected the condition of summer range prior to the time of collection. Total protein, BUN and FN appeared to be more sensitive indicators of animal protein stores. The low values of these parameters in CL sheep suggested inadequate protein content in late summer to winter range forage. This substantiates visual estimates of poor quality winter range for this herd (Davidson 1982). Similar
findings in WW sheep may reflect variations seen in chronically ill animals, but further investigation of forage protein content is required to substantiate any estimates of range quality by this method. In contrast, low BUN values of ER sheep were present with normal TP and higher FN content than either of the other herds. Hebert (1972) compared high and low altitude forages in the E.K. and found those from high altitude pastures to be of higher nutrient quality. Stelfox (1976) and Schuerholz (1983) assessed the mountain grassland bighorn habitats within the national parks and the E.K. bighorn habitats and found them to be of high productivity and quality. They were also easily available due to the preference of sheep for south facing, snow free slopes. Schuerholz (1983) also noted a lack of interspecific competition for forage at this altitude. These findings support the hypothesis that sheep grazing on high altitude pastures have year round access to better quality nutrition. The low BUN values and marginal tissue copper levels do not completely support the presence of higher quality of feed on the ER winter range; however, sample numbers were small without analysis of their particular forage quality. Larger numbers of coordinated samples of forages, soils and animals over time would be required to fully interpret these results, but the study results do suggest differences in the nutritive status of the three herds.

The study was designed to describe and compare the health status of individual bighorn sheep from three herds, to associate the health status with environmental and physiological factors
and the overall dynamics of each herd and to select parameters most suitable for future monitoring of bighorn sheep herd health status. Alternatives to the study design involving more controlled protocols may have provided data more amenable to statistical analysis, but were felt to be impractical for the field situation. I believe that the observational approach was the most useful for this situation because of the broadness of the study objectives, the restricted sample size and our aim to keep the methods as close as possible to techniques available to wildlife managers in the field.

The results of this study demonstrated measurable differences between the low and high elevation wintering bighorn sheep herds of the E.K. region of British Columbia. Despite the small sample size, variations in lungworm levels, TP, FN, selenium status and levels of chronic disease were present. These variations appeared to support the assessments of differences in overall herd dynamics and health status. The differences were primarily related to the factors which have been suggested to have predisposed the E.K. bighorn sheep to an all-age dieoff in 1981. These were high animal density, poor nutrition, parasitism and trace mineral deficiencies. High lungworm levels, reduced nutritional indices and lower selenium levels occurred in the CL herd at high animal density. Sheep wintering in lower density at high elevation on the ER range appeared to have access to better quality nutrition and maintained lower levels of lungworm infection. The effects of the 1981 dieoff and the factors believed to predispose the WW
herd to the disease outbreak had a more longterm influence than expected. There was residual chronic disease in adult bighorn sheep, a marked reduction in the viability of lambs, and evidence of immunosuppression in all age groups. This profile had persisted for at least 3 years post-dieoff, in spite of reduced animal density on winter range and low levels of lungworm infection.

The status of the study herds appeared to be best determined by the use of a group of indices. This study indicates that LPG, FN and liver selenium levels are parameters which can be measured and differ significantly between bighorn herds in the E.K. region. Since LPG and FN can be obtained from noninvasive fecal collections, these parameters are recommended to be assessed in specific herds over time, with the sampling of blood, tissues and trace mineral assessment reserved for when circumstances allow or indepth investigations are desired (see section 7.0). Hanks (1981) advised the characterization of population condition by the assessment of physiological parameters together with reproductive capacity, habitat condition and trend. Comparison of the parameters of nutritional condition, parasite levels, reproduction status and habitat condition between herds of bighorn over time may then indicate and be used to predict when conditions predisposing to health problems and sheep density move into the critical range. If educated predictions can be made before dieoffs occur, remedial actions can be carried out to maintain and increase wild sheep populations.
7.0 MANAGEMENT RECOMMENDATIONS

7.1 Health Status Monitoring

The evaluation and monitoring of the health status of any animal herd, whether domestic or wild, must follow a specific program. The livestock industry and veterinarians have developed such programs over the past century to integrate the maintenance and improvement of animal health, production and management. The clinical and subclinical disease problems of bighorn sheep have been shown to have similarities with certain livestock situations. It appears that this species may be suited to the development of basic herd health programs. The following guidelines may be considered and critically assessed over time for desired results to assist the wildlife manager in the maintenance of a healthy and productive bighorn sheep population.

The bighorn sheep population must be evaluated by herd since individual animals are impractical to treat. Wildlife managers should first assess each herd and range separately and group them on the basis of similar animal and environmental factors (ie. sheep density, range quality). Management actions can then be tailored to suit similar types of herds. A management plan with goals outlining desired sheep numbers, herd use (ie. hunting, nonconsumptive) and intensity of management desirable and possible should be established. Finally, it may be necessary to prioritize management actions and herds in order of importance to maximize benefits.
Baseline population statistics are vital for the proper identification of each herd. This must include total numbers, classified counts and reproductive performance. The critical ranges, migration routes and the location of mineral licks used during all seasons should be defined. Ranges of unknown quality of high priority herds should be assessed.

The evaluation of the health status of a bighorn herd involves at least three separate stages:

1. Determine an accurate history of range quantity and quality and bighorn production and disease occurrence for each herd.

2. Establish present status.

3. Follow seasonal and annual protocol to observe trends in range and animal parameters.

These may be best accomplished by:

1. Intensive management herd
   - collection of animals - use protocol similar to this study
   - monitor seasonal and annual trends
     a. range - forage quantity and quality
     b. animal - density
        - lamb:ewe ratio
        - lamb survival
        - feces - fecal nitrogen
        - larvae per gram
   - consider treatment with anthelmintics, trace minerals and/or density reduction
2. Moderately intensive
   - monitor seasonal and annual trends
     a. range - forage quantity and quality
     b. animal - density
       - lamb:ewe ratio
       - lamb survival

3. Extensive
   - monitor seasonal and annual trends
     a. range - forage quantity
     b. animal - density
     - lamb:ewe ratio

The method of sampling used in this study may not be indicated for some management or monitoring schemes. The results, however, underline the value of both invasive and noninvasive techniques. Of these, we believe the collection of feces from sheep herds over successive seasons and years remains the most practical and economical method of monitoring the health status of bighorn sheep. Serial fecal LPG will allow the determination of lungworm infection trends. Fecal nitrogen levels have proven useful in determining diet and habitat quality, but require further studies in field situations, again over time. Although blood and urine cortisol levels were not particularly useful in this study, further studies with these, and a potential method for analyzing fecal cortisol metabolites (Spraker pers. commun.), may provide values for baseline cortisol secretion and allow the evaluation of levels which indicate
increased herd stress. Necropsy evaluation of freshly dead animals in healthy and diseased condition by collection or hunter kills, the analysis of liver tissue for trace mineral levels and serum collection for more intensive analyses are strongly advised whenever possible. These sampling procedures must, however, be considered only as corollaries to the observation and study of stable and fluctuating sheep herds, their dynamics and range conditions for trends over time.

7.2 Columbia Lake

The B.C. Wildlife Branch was advised to treat CL sheep on the basis of high levels of lungworm infection and trace mineral deficiencies found in this study. Decreasing the density of animals on winter range was considered to be an integral part of this action to reduce grazing pressure and in anticipation of increases in lamb:ewe ratios in subsequent years (Spraker pers. commun.). Sheep on CL winter range were conditioned to eat fermented apple mash during the late winter of 1984. Fenbendazole anthelmintic was added to treat each sheep three times at weekly intervals. Mineral licks had been supplemented with domestic sheep trace minerals since the fall of 1983. Twenty five bighorns of mixed age and sex were live-captured after the treatment and moved to a suitable unoccupied winter range. Subsequent transplants have since occurred and a second herd has been established.
An additional three CL sheep were collected and a poached animal was examined in the fall of 1984. Similar procedures were performed as described for project sheep. Fecal samples were taken from transplanted sheep and the CL range for lungworm larvae counts.

Three of the CL sheep collected in 1984 had deficient and one had marginal selenium levels. One of the four had marginal copper values. Lungworm larvae numbers were very low in an adult ewe and a yearling (11,91), moderate in one lamb (344) and high in a second (724). Larvae levels in range collected or trapped sheep feces were zero after spring anthelmintic treatment, but returned to near previous levels in some animals by the fall.

7.3 Wigwam

Anthelmintic therapy of the WW herd was given low priority due to the small numbers of lungworms present in the study sheep. Trace mineral supplementation of mineral licks was advised. It was suggested that animals in poor condition or demonstrating obvious clinical signs of disease be removed, when observed, to reduce the proportion of chronic disease carriers in the herd. Since it was believed that the majority of ewes were in an upper age class of reducing productivity, it was suggested that transplantation of sheep onto this range be considered. Experience with the introduction of sheep into existing herds in the U.S.A. (Spraker pers. commun.) and Canada (Davidson 1982) has previously resulted in mortality in the native herds. This
alternative to natural population regrowth was advised to be used at the WW with caution.

7.4 Ewin Ridge

Management recommendations for the ER herd were restricted to the maintenance of present or slightly lower herd numbers. Since intensive procedures such as live-capturing were completely impractical, it was suggested that sheep numbers be controlled by harvest strategies as required. In addition to the established full curl or 8 year old and over ram regulation, a ewe-lamb harvest was suggested.

The adoption of these recommendations into the E.K. bighorn sheep management plan has accomplished some major achievements. The parasite burden of CL bighorn sheep has been reduced, if not absolutely, at least annually at the time most critical for the production of healthy lambs. Anthelmintic treatment in the late winter not only reduces the number of larvae transmitted to the fetus in utero, but also reduces the number of parasites carried to lambing and summer ranges. Lamb:ewe ratios at Columbia Lake increased after treatment of the herd (Davidson pers. commun.), but it appears that therapy must continue in the face of constant reinfection of sheep from the range and lack of significant changes in selenium and copper levels. At the time of writing, the two new herds created with sheep transplanted from CL after anthelmintic treatment are reported to be extremely successful. The management actions suggested for WW and ER have been undertaken but no marked positive or negative results have been identified.
There is a serious danger that management practices focus excessively on one area. The manipulation of bighorn sheep herds must be coordinated with the management of their habitat. It has been previously stated that habitat loss and deterioration are considered to be primarily responsible for the reduction in bighorn sheep numbers in the E.K. region. If bighorn sheep are to remain as viable populations, major efforts must be directed towards maintaining and improving the lands available to their use on a year round basis. This includes the protection of existing ranges by limiting access, road closures, the acquisition of prime range as reserves, coordinated resource planning with industrial, agricultural, residential and recreational developers and the harvest of competitive ungulate species. The decreased quantity and quality of E.K. sheep winter range can and has been improved since range enhancement techniques such as controlled burning, selective logging and clearing and forage seeding were instituted after the 1960’s dieoff and were considerably intensified since 1981. It is imperative that these practises be continued as required to prevent further habitat losses and deterioration. I believe that the intensive management practises used on the E.K. bighorn sheep and their winter ranges have been responsible for assisting in the recovery of the population from approximately 1500 sheep in 1983 to over 2000 in the spring of 1986 (Wolterson-Strauss pers. commun.). It is vital that the committment to the recovery of the bighorn herds continue and be coordinated with the protection and enhancement of their habitat.


PERSONAL COMMUNICATIONS


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