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THEORETICAL CONCEPTS OF DISEASE VERSUS NUTRITION AS PRIMARY FACTORS IN POPULATION REGULATION OF WILD SHEEP

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Abstract: Concepts concerning population regulation, including carrying capacity as it relates to nutrition as the primary population regulating mechanism in wild sheep, are discussed. Literature is reviewed and theoretical concepts are postulated that implicate evolution of the immune system as an important intrinsic factor in population regulation. Population density, as an environmental stressor additive with other environmental stressors, acting upon animal's immune system may be a major population regulatory factor in wild sheep. The concept of immune system dependence versus independence of pathogens, especially regarding Pasteurella haemolytica, is proposed by the author. Management implications and recommendations are discussed.

Numerous studies have been conducted to understand complex interactions among animals and environmental factors thought involved in regulation of wildlife populations. As a result, basic concepts have been developed to explain fluctuations or stability, in wildlife numbers and density. These include concepts such as carrying capacity, density dependency, environmental stressors, limiting factors and others, which have helped shape our understanding of population regulation. Without an understanding of concepts and mechanisms that regulate wildlife populations, many management strategies will be ineffective. Although controversy exists and neither scientific nor empirical data adequately justify the relative importance of these concepts, they form the basis of current management strategy. Other, more recent, concepts used to explain population regulation in large ungulates, such as stress and disease as primary factors, have, until recently, been mostly ignored. And, because wild sheep appear more susceptible to adverse affects of epizootic disease than other wild ungulates in North America, they may have evolved with different population regulatory mechanisms making disease more important.

Among biologists, there appear a wide array of opinions concerning cause of sudden declines in wild sheep populations that are similar in nature and circumstance. Most investigators support the concept of carrying capacity and suggest nutritional deficiencies cause or act as triggering mechanisms for these declines. Management strategies, based on this concept, are then used in attempts to reverse or prevent further declines.

However, current management of Rocky Mountain bighorn sheep (Ovis canadensis canadensis) and their habitats does not appear effective

despite genuine efforts (Risenhoover et al. 1988, Smith et al. 1991). Without more effective management, bighorn sheep numbers could be expected to further decline over the next 25 years (Jahn and Trefethen 1978). Therefore, if we are to more effectively manage wild sheep populations, we must critically analyze not only management techniques, but concepts on which current management is based. Although the concept of animal population regulation has not been proven, I support the hypothesis and believe that wild sheep are regulated at least partially by intrinsic factors such as the influence of chronic stress upon immune systems.

This report reviews past studies on population dynamics of wild sheep and proposes concepts that may allow better understanding of proposed population regulation, therefore contributing to more effective management.

NUTRITION VERSUS DISEASE

In 1798, Rev. T. R. Malthus pointed out that animal populations always increase up to the limit of their food resources. And, that lack of food through starvation and disease, then acts to prevent further increase (Sinclair 1989). The Malthusian hypothesis has been at the center of all subsequent debate on what regulates populations. However, others have proposed that animal populations may be regulated at a level below that imposed by food supply (Chitty 1960, Stelfox 1976).

Carrying capacity is a concept developed to explain the relationship between habitat and ungulate population size or density. It is defined as, an equilibrium between animals and vegetation (Caughley 1979). This or some similar concepts are used by wildlife managers to justify various strategies and techniques to alter habitat or population size to achieve desired goals. These strategies are premised on the idea that animal numbers and recruitment are based primarily on nutrition and intraspecific competition. Disease, as a population regulator, is often relegated to a proximate mechanism based on nutrition (Davidson 1981, Cook 1990, Cook et al. 1990).

Many investigators of wild sheep in North America have concluded nutrition is the primary factor involved in population regulation. Allen (1962: 59) in discussing the decline of bighorn sheep, stated, "There are numerous factors involved, it seems, in actual control of herd size; but a great deal of it appears traceable to range." Johnson (1983: 149) stated, "diseases have probably been responsible for large scale mortalities in many bighorn populations in North America." He believes that the impact of parasites and disease on population dynamics of bighorn sheep is not well understood, and concludes, "The most important factor in management of bighorn sheep is habitat" (Johnson 1983: 174).

Buechner (1960: 107), stated, "as a population regulation mechanism, the parasite-disease complex,...operates almost independently of the condition of range vegetation." However, in concluding remarks, he considered poor nutrition as the "triggering" mechanism predisposing

bighorn sheep to the parasite-disease complex. Streeter (1970a) believed Buechner's final conclusion was unfounded because nutrition range-analysis studies before, during and after die-offs were not made. This was often the case with studies of wild sheep die-offs or annual variations in natality where poor nutrition was considered an important factor (Marsh 1938, Buechner 1960, Woodward et al. 1972, Stelfox 1974, Whitten 1975, Long 1980, Schuerholz 1984, Whitfield and Keller 1984, Festa-Bianchet 1988, Cook 1990). And, when correlation of range conditions and mortality or low recruitment was made, physiological assessment of nutrition was not (Marsh 1938, Buechner 1960, Streeter 1970b, Horejsi 1972, Stelfox 1974, Whitten 1975, Hoefs and Brink 1978, Long 1980, Hoefs and Bayer 1983, Schuerholz 1984, Festa-Bianchet 1988, Cook 1990), casting suspicion on conclusions that vegetation had a primary effect on the population. Complex forces such as nutritional, behavioral, environmental and genetics that act upon a population, have been traditionally assessed by secondary indicators such as habitat, food habits, and population characteristics. Theoretically, assessment of primary indicators obtained directly from animals representing a population would minimize many sources of variation and provide greater precision for studies. Primary indicators reflect the health of the animal, and by proper sampling, the health of a population (Franzmann 1972, Fowler 1986). Consequently, there has been a gradual shift from assessment of carrying capacity by vegetation utilization studies and change in population characteristics to that of establishment of nutritional status of wild populations (Franzmann 1971a, Franzmann 1971b, Hebert 1978). It should no longer be sufficient to simply monitor animal numbers or habitat conditions. One should view with suspicion previous studies conducted on wild sheep population dynamics that suggested nutrition as a cause for population declines, when assessment of health or physiological status was not conducted. Investigators that document a positive correlation between recruitment and range vegetation or precipitation often fail to acknowledge decreased forage production may serve to decrease area of available habitat, thereby increasing population densities. Increase in population density could account for decreased natality without regard for decreased forage quantity or quality.

Some investigators found that preceding a disease induced die-off, bighorn sheep were in fact in good body condition, suggesting little correlation between nutrition and the die-off. Festa-Bianchet (1988: 70) in his study of a pneumonia epizootic in bighorn sheep in Alberta, found "dead sheep in good body condition, with no obvious evidence of malnutrition." Post (1962:4) investigated an outbreak of pasteurellosis in a captive sheep population in Wyoming and found the dead animals to be in "excellent body condition, as denoted by amount of body fat." Other studies involving captive sheep found similar results (Foreyt 1988, Miller et al. 1991). Ryder et al. (1992) in investigating a bighorn sheep die-off in 1991 near Whiskey Mountain, Wyoming, concluded there were good range conditions prior to the die-off of approximately 40% of the herd.

Bailey (1986:333) studied a bighorn sheep herd in Waterton Canyon, Colorado, and after investigating an all-age die-off, concluded "Waterton sheep were in excellent physical condition, indicating no

shortage of forage." Other investigators (Fisher 1927, Spraker 1974, McQuivey 1978, Wishart et al. 1980, Spraker and Hibler 1982, Onderka and Wishart 1984, Spraker et al. 1984, Andryk and Irby 1986) found similar results. Cook (1990:215) studied two bighorn sheep herds in Wyoming and, even though he concluded poor nutrition was the cause of death, found nearly all mortalities to be lambs dying from disease and stated "Chlamydia infection likely originated from outside sources and caused substantial mortality even in lambs considered to be in good condition." Heimer (1988:42) in discussing Dall sheep (Ovis dalli dalli) management in Alaska, stated "Nutritional stress may occur in unusual circumstances, but normal circumstances do not lead to this problem. Comparisons of nutrient quality of winter range plants selected by sheep, the summer nutrient quality of these food plants, and the body condition of ewes during rut and in late winter revealed no caloric advantage for a low density population when compared with a high density population." He concluded "Still, population performance was strikingly different between the two; the low density population had better performance."

If nutrition is not a predisposing factor in many die-offs or population regulation of bighorn sheep, what is? Some investigators (Forrester 1971, Hudson 1972, Thorne et al. 1982, Hoefs and Bayer 1983, Spraker et al. 1984, Bailey 1986) believed stress was an important factor in disease caused die-offs in bighorn sheep. And, some (Post 1976, Feuerstein et al. 1980, Spraker and Hibler 1982, Stevens 1982) suggest stress without regard for nutrition, may play a role in bighorn sheep die-offs.

Stress is defined as the need for an individual to make abnormal or extreme adjustments in physiology or behavior to cope with adverse aspects of its environment (Fraser et al. 1975). Stress operates by inducing elevated circulating glucocorticoids and possibly other hormones, which may inhibit immune or reproductive systems, predisposing an animal or population to reduced natality or infectious disease (Christian and Davis 1964, Hunninghake and Fauci 1977, Spraker 1977, Kelley 1988, Griffin 1989, Anderson 1991). Although stress has been shown to affect reproductive function by lowering natality in some animal species (Christian and Davis 1964, Christian 1971), evidence is not available to suggest it operates in this manner in wild sheep populations and will not be discussed. Cohen (1987) proposed that acute stress does not exert a significant effect on immunocompetence, and it is only chronic stress which causes an impairment of immunity compatible with the development of disease. Therefore experiments designed to acutely reduce immune system function by administration of glucocorticoids to mimic stress, may lead to erroneous conclusions. Chronic stress factors, or environmental stressors occur in a variety of forms. Nutrition may be a stressor, acting upon an animal's immune system, predisposing to disease (Chandra and Newberne 1977). Other stressors include population density, inclement weather, chronic diseases, parasitism and harassment.

Several investigators have discussed concepts of stressors that reduce immune system functions and the role stress plays in reducing a populations' resistance to disease. Spraker et al. (1984) discussed

stress and the pathogenesis of stress upon bighorn sheep in a die-off in Colorado resulting in the death of 75% to 85% of the population. They concluded stress, mostly from human disturbance caused increased levels of cortisol resulting in inhibition of the inflammatory process and increased susceptibility to bacterial pathogens.

Schwantji (1986:247) studied 3 herds of bighorn sheep in southeastern British Columbia and concluded, "...at a certain population density, additional or increased levels of pre-existing stressors appear to overwhelm the functional immune system and precipitate the occurrence of epidemic disease." Streeter (1970b: 50), in comparing demography of 2 bighorn sheep herds in Colorado stated "Based on these statistics, mean breeding population mortality varied directly with mean population density. Mean survival of lambs and mean breeding population growth varied inversely with mean population density." He concluded, "The mechanism could be social stress induced by intraspecific interaction or a nutritional stress induced by a decrease in available forage." He did not find a definitive correlation between mortality and specific forage limitations.

Wehausen et al. (1987) reviewed data from a desert bighorn sheep herd in southern California during 1953 through 1982. During the periods 1953-61 and 1977-82, the population experienced low recruitment rates and suspected disease epizootics. Concerning the population decline beginning in 1977, they stated (p. 95): "Given that average recruitment ratios suggest that this population essentially had been stable since 1977, it would be tempting to interpret its recent dynamics as nothing more than a usual attainment of an ecological carrying capacity set by the interaction of vegetation and the sheep population density alone. Under this interpretation disease would act only as secondary factors precipitated by nutrition. Two factors do not support this interpretation: (1) the substantial discontinuity in recruitment rate that occurred in 1977 and (2) the increased precipitation in critical months during the 1977-82 period, the effect of which should have been a raised carrying capacity and subsequent population increase rather than stabilization." They further concluded, "Our analysis indicate that 3 independent precipitation factors, 1 disease factor and 1 probable population density factor all exhibited measurable effects on the dynamics of lamb recruitment in the Santa Rosa Mountains between 1962 and 1982."

Hudson (1972:33) investigated the functional activities of lymphocytes in sheep that were subjected to capture, transport, and confinement fed medium quality hay and demonstrated alterations of *in vitro* characteristics of peripheral lymphocytes during stress associated with introduction to captivity. He concluded, "Since bighorn sheep are particularly susceptible to disease when brought into captivity, it is possible that depressed *in vitro* lymphocyte reactivity reflected impaired immunity." Other investigators have also suggested stress is a factor that reduces the ability of the immune system and increases susceptibility to disease.

Belden et al. (1990) found, in their study of captive bighorns, fluctuations in B cell responses occurred around time of lambing and

concluded that this would not be beneficial to the population. This suggests that stress factors are operating upon the immune system at a critical time, during lambing. Time of lambing and especially 6-8 weeks thereafter, when colostral antibodies begin to wane, are certainly a time of high susceptibility in bighorn sheep. At this time densities of sheep can attain highest levels on nursery areas and cause social stress and increased disease transmission, predisposing to disease. Spraker et al. (1984) suggested that stress-related summer lamb mortality was a result of 1 of 2 factors: stressed ewes producing less colostral antibody resulting in lamb deaths due to lack of maternal protection or secondly, surviving ewes carry high levels of Pasteurella spp. which they may transfer to lambs, overwhelming their immune system and causing fatal pneumonia. I suggest both factors could be a result of stress caused by high population density.

Population density historically was viewed as a factor upon which environmental variables depended; increasing their effect as density increased and functioning to reduce density to an equilibrium. These density dependent variables were thought to be an integral part of population regulation. In fact, environmental factors that were not density dependent were not part of the regulation mechanism (Sinclair 1989).

However, I propose population density, or overcrowding, acts as another environmental stressor, intrinsic in nature and may at times act independent of nutrition. It acts upon an animal's immune system via complex interactions of hormones, including mainly glucocorticoids, predisposing to disease. And, other environmental stressors, as previously mentioned, are additive forces serving to suppress population numbers in the same manner as stress resulting from population density.

Marsh (1938) pointed out that crowding was an important factor in pneumonia in bighorn sheep in his studies in Glacier National Park. Hobbs et al. (1990) reported that disease-induced die-offs in bighorn sheep were repeated at approximately 20 year intervals when populations attained peak densities.

Onderka and Wishart (1984) found that the timing of pneumonia outbreaks corresponded to the breeding season of bighorns, when animals are concentrated. Dunbar (Wildl. Dis. Res. Inst., unpubl. data) found high summer lamb mortality in a bighorn sheep herd in central Idaho, when population density was at or near a recorded all time high (Idaho Fish and Game files), and physiological assessment of ewes just prior to lambing revealed they were in good physical condition.

Murphy and Whitten (1976) found ewe:lamb ratios of Dall sheep in McKinley Park, Alaska, was significantly lower in the area of highest density. Investigations into all-age die-offs of bighorn sheep in the East Kootenay region of British Columbia in the 1960's and 1980's determined these die-offs were preceded by high population density (Schwantji 1986). Burles et al. (1984) in their study of Dall sheep in Yukon Territory, Canada, found population density to be a significant

contributor to variations in productivity among populations. These studies found little or no correlation with mortality and any specific forage limiting factor.

Based on these and other investigations, I believe information exists that supports a concept that nutrition does not necessarily serve as a predisposing agent to either catastrophic die-offs or reduced natality due to disease. While much remains to be discovered about proximate causes, predisposing agents, and etiology of disease, circumstantial evidence suggests that bighorn pneumonia is induced by multiple stress factors (Potts 1938, Spraker 1977, Feuerstein et al. 1980, Spraker and Hibler 1982, Spraker et al. 1984, Onderka and Wishart 1984, Bailey 1986).

This interaction of stressors can be visualized if a stress index factor of 1.0 is required to suppress a population to the point of stabilization. The effect of population density could have a stress factor range of 0.3-1.0, inclement weather 0-1.0, harassment 0-0.4, chronic parasitism 0-0.2, nutrition 0.1-0.6 (unless the regulatory system is overridden, then 1.0). One could then understand how these factors become additive. However, population density or weather alone could account for a population decline, or with no inclement weather and a lowered density, a combination of other factors could achieve a population decline. But a lowered population density, hence reduced stress, would allow a population to increase again to an equilibrium once the other stressors returned to lower levels. The effect of all environmental stressors acting upon a population would be in a dynamic equilibrium with the population.

The concepts of carrying capacity, density dependency and intra-specific competition for forage are not discounted here. Stelfox (1976) hypothesized that bighorn populations that increase in density will suffer die-offs before they reach a balance with their environment. Chitty (1960) proposed that intrinsic factors, those characteristics of an individual such as behavior, physiology and genes, could act to regulate the population at a level below that imposed by the food supply. I believe that forage is the ultimate factor that will limit population size, but that food serves in many cases, not as a regulatory but a limiting mechanism. Concepts of population limitation and regulation should not be confused. For example, the amount of space, food, or water (limiting factors) may limit the size of a population but competition (stress) for space, food, or water may serve to regulate a population and is a function of population density. Extrinsic and intrinsic factors may function to regulate populations. For example, an intrinsic factor such as chronic stress resulting from high population density may influence physiologic functions such as immune systems and cause disease (extrinsic factor). Populations that are regulated may attain optimum size or density that would be beneficial for long term survival of the species. Populations that are not regulated but limited may at times reach peak population numbers but may not be beneficial for long term survival.

Bailey (1986:332) states, "...if we become satisfied with, or promote "stress" as an explanation [for die-offs], we may impede

development of understanding of the causes of stress in bighorn sheep." I agree; however, many biologists have accepted the simplistic concept of carrying capacity which has impeded our understanding of population regulation in bighorn sheep. I believe the acceptance of the concept of stress, its effect on the immune system and its role in population regulation would be a major step towards understanding this complex issue.

IMMUNE SYSTEM DEPENDENT VERSUS INDEPENDENT

To combat an array of pathogens, animals, including wild sheep, have developed various sophisticated immunological defense mechanisms. The pathogenicity of potential disease causing organisms is usually dependent upon the functional level of the animal's immune system. I will call those pathogens that rely upon the functional level of the immune system to cause disease as "immune system dependent". Those pathogens that, because of their high virulence, the high susceptibility of sheep, or their ability to overwhelm an uncompromised immune system and cause disease, I will term "immune system independent". These immune system independent pathogens are those that usually are not enzootic in sheep populations but increase in virulence or number due to factors independent of sheep populations. These factors may include numbers of secondary or intermediate hosts, or vector populations. An example of an immune system independent pathogen would be Bluetongue virus. Its affect on bighorn sheep populations is related to the size of the vector population Culicoides spp., and not to the functional level of the bighorn sheep's immune system. Some pathogens may serve as both immune system dependent and independent, depending on their nature or virulence such as P. haemolytica. However, most pathogens are immune system dependent and are enzootic to wild sheep populations. It is my belief that immune system dependent pathogens serve to regulate populations versus independent ones that do not.

It is my belief if Pasteurella haemolytica, regardless of source, operated only in an immune system dependent manner, epizootics would be a major factor in regulating populations. However, in my opinion, P. haemolytica, originally dependent on the functional level of the animal's immune system to cause disease, gains in virulence (Spraker 1977, Feuerstein et al. 1980, Ellis 1984, Wilkie and Shewen 1988, Callan et al. 1991) as it passes through many animals weakened by its effects and thereby begins operating in an immune system independent manner. The effects of P. haemolytica would then not be regulating populations, but serve as a catastrophe.

A more virulent P. haemolytica would then be transferred, usually by young rams (Onderka and Wishart 1984), to adjacent sheep and to other populations, overwhelming the immune system without need for stressors. It would then lose its increased virulence over time due to intrinsic factors operating within the bacterial population and again become immune system dependent. A mechanism similar to this may have been proposed by Festa-Bianchet (1988:73) when he stated, "Population dynamics after the [bighorn sheep] epizootic, particularly lamb survival may reflect lingering effects of the disease and therefore not show the expected density dependent relationships."

In an attempt to model population dynamics of bighorn sheep, Hobbs and Miller (1993) postulated that epizootics due to Pasteurella spp. regulated bighorn numbers and that external forces such as habitat changes, environmental stress, parasitism and contact with domestic animals, do not appear necessary to produce periodic pasteurellosis epizootics in bighorn herds. In my opinion, I believe their model points out that bighorn numbers are reduced by epizootics caused by Pasteurella spp. but these epizootics do not function to regulate bighorn populations because they are apparently operating in an immune system independent fashion.

In this postulated system of population regulation, it seems logical that wild sheep have evolved to "fine tune" their immune system to regulate population density and compensate for other environmental stressors to their benefit in long term survival. However, this system has evolved in close association with the environment. Therefore, man's manipulation of the environment may be disrupting this system.

DISEASE VERSUS DIE-OFF

Disease is defined as a specific state of malfunctioning in an animal (Dorland 1963). It is the culmination of various defects, abnormalities, excesses, deficiencies and injuries as they occur at the cell and tissue level which ultimately result in clinically apparent dysfunction (Slauson and Cooper 1982). Disease may sometimes go undetected at the clinical level even though the lesions underlying the disease have been present in the tissues for a long time. In this discussion, disease will be limited to a dysfunction caused by living organisms (bacteria, viruses and parasites, etc.).

Traditional investigations on causes of mortality where disease was believed involved were aimed at identifying single cause (a pathogen) and effect (morbidity and mortality). These studies, in effect, equate disease with die-off or population crash. Investigators (Stelfox 1976, Hobbs et al. 1990, Hobbs and Miller 1993) therefore, attempted to model population dynamics of sheep based on this concept and suggest disease is a population regulator. However, one should realize that disease causing organisms are numerous in bighorn sheep populations (Adrian 1981, Dieterich 1981, Goodson 1982, Thorne et al. 1982, Fowler 1986), and to ignore their significance to long term regulation is missing the point. Die-offs in sheep populations are mostly a result of a respiratory pathogen which is usually P. haemolytica. To study only the mechanics of a population die-off due to Pasteurella spp. will lead only to understanding pathogenesis of die-offs, not of mechanisms of population regulation due to disease factors that constantly affect and regulate sheep populations. To understand population regulation, more emphasis should be placed on understanding bighorn sheep populations that are apparently stable, or where numbers fluctuate little. To understand the regulatory nature of environmental variables and intrinsic factors that are constantly at work, we should not place emphasis on studying populations that may temporarily have lost control due to a catastrophic event, disease or otherwise.

Stelfox (1974) was probably correct in concluding that bighorn sheep in the Canadian National Park did not exhibit any density dependent self-regulating mechanism. He was considering population die-offs, or disease pathogens that are not dependent upon functional level of the immune system, rather than long term effects of pathogens that are dependent upon the animal's immune system to cause disease.

Hudson and Stelfox (1976:110) pointed out in their attempt to model, "disease interfered with a more finely tuned mechanism of population regulation." They equate disease with die-off in their model and would have been justified in their conclusions if they had stated that die-offs interfered with a fine-tuned mechanism for population regulation. The finely tuned mechanism of population regulation they elude to, may in fact be disease other than Pasteurella spp. at least, it may be Pasteurella spp. that operates in an immune system dependent rather than independent manner to which die-offs may be attributed.

The term disease must include pathogenic or potentially pathogenic organisms operating in a population. One should not single out a pathogen, in most cases Pasteurella spp., and model their effect under the guise of disease. Consequently, one must understand the pathogenesis of combined diseases and realize the difference between disease factors that regulate by their immune system dependence and those that effect but do not regulate, by their immune system independence. Many pathogens, combined with or without Pasteurella spp. may serve to regulate bighorn sheep populations by operating on newborn lambs, where mortality due to disease is often high but often ignored by investigators, especially when populations appear stable.

DENSITY VERSUS MOBILITY

Nicholson (1933) revolutionized thinking about population regulation when he introduced the concept of density dependent factors. Because population density appears to be so important, I wish to give some definitions of density and propose a concept which should be considered when disease factors are involved. Animal density is defined as, "individuals per unit of area" (Dasmann 1964:90). He also points out densities should be related to occupied habitat rather than mere geographic area and must always be measured at a particular point in time. I believe density must also be considered when disease is the object of study as a stressor acting upon the animal's immune system. Population density is also a factor as it relates to disease transmission among individuals within a population. The greater the density, the higher the rate of disease transmission. And, in regards to density and disease transmission, another factor to be considered is mobility of a population. Mobility refers to the distance traveled per day. Optimum mobility would include a large mean distance traveled per day with a low standard deviation value.

As individuals become weakened due to disease, number and virulence of pathogens may increase, making transfer of pathogens to others easier. Larger distances traveled would tend to increase the likelihood of weakened individuals lagging behind and being excluded from

the group, thereby reducing disease transmission.

The greater the mobility of a population, the greater the effect of this natural quarantine, which allows populations to achieve greater densities. This would have a dramatic effect at lambing and nursery areas, where ewes concentrate and reach high densities. Lambs in nursery areas that are on discreet limited habitats would suffer greater losses to disease than those on expanded habitats where density is less and mobility greater. Hoefs and Bayer (1983), in their study of Dall sheep in Canada, found sheep densities more than doubled on nursery areas as compared to year-round ranges. Lenarz (1979) reported most lamb mortalities in his study in New Mexico occurred during movements between ewe ranges (those lagging behind).

However, as population size may diminish, lamb nursery areas may diminish in size due to an attempt by ewes to concentrate onto key habitats as determined by physiographic features. Densities may vary little until population size is reduced to a certain point. This concept of sheep maintaining high densities by concentrating themselves into more highly preferred areas in the face of population declines has been observed by Spraker (1974).

It should also be mentioned that mobility may have negative effects on a population. Onderka and Wishart (1984:360) in their study of a sheep die-off in Canada state, "The highly mobile component of rutting rams was probably responsible for spreading the disease [pasteurellosis] over such a large area."

MANAGEMENT IMPLICATIONS

As stated, despite genuine efforts current management of Rocky Mountain bighorn sheep populations and their habitats does not appear to be effective (Risenhoover et al. 1988). Transplanting of bighorn sheep onto historic ranges may, in some instances, be an effective management tool today. Transplants involving California bighorn sheep (*Ovis canadensis californiana*) into southwestern Idaho ranges is an example of the success of this type of management (Bodie et al. 1990). However, historic ranges suitable for transplanting are limited. And Risenhoover et al. (1988) suggests that many transplanted bighorn sheep populations are not expected to survive. Smith et al. (1991) also believe that transplanting bighorn sheep as a management tool to increase numbers has failed in the western United States.

Protection of existing habitats from degradation and protection of sheep from illegal harvest are also management strategies where success is in question. Habitat manipulation and range improvements on existing key bighorn sheep ranges has shown little success. Bighorn sheep populations continue to decline. Jahn and Trefethen (1978) warned that without more effective management, an additional loss of 8% of bighorn sheep could be expected over the next 25 years.

If disease is a major factor contributing to population declines,

and if it is based on population density rather than nutrition, then management strategies based on reducing densities should take priority. However, does reduction of sheep numbers through increased hunter harvest of ewes or transplanting from a population decrease density to the degree required? Possibly not. Decreasing number of ewes on lamb nursery areas should be of greatest benefit if it results in decreasing ewe densities. But in many cases, even if harvest strategies allow such a reduction, they may have to be substantial because ewes in bordering marginal nursery areas may concentrate on key nursery areas, maintaining high densities. It would therefore seem more beneficial to increase the size of nursery areas through habitat manipulation. These habitat manipulations would probably consist of creating optimum physiographic characteristics, i.e., increasing visibility in steep rocky areas overgrown with small trees and shrubs or creating or improving movement corridors between nursery areas, rather than increasing vegetation quality.

This same concept applies to manipulation of winter range habitat. In other words, one should create more area of sheep habitat, rather than creating better quality habitat in the same key areas, as is the present management strategy. Management strategies based on creating more and larger areas of sheep habitat not only would reduce sheep densities but would increase mobility.

A similar management strategy of increasing area was postulated by Risenhoover et al. (1988). They believe bighorn sheep have abandoned migration corridors to less favorable ranges due to forest succession reducing visibility. And, "The resulting phenomenon of mostly small, isolated, and sedentary sheep herds perpetuates population declines and habitat loss through loss of traditional movements", (Risenhoover et al. 1988:348). They also concluded that burning existing winter range will not expand a herd's area and will not encourage a herd to re-establish abandoned movement patterns. They stated, "For many herds, we believe that population size, distribution, and movement must be increased if they are to remain viable", (Risenhoover et al. 1988:348). Increasing access to escape terrain and improving visibility within habitats are generally believed to be the primary determinants of distribution and habitat use (Cook 1990). These management strategies are similar to those that consider immune system dependent disease factors as primarily regulating sheep populations through population density and mobility.

Other factors found important in sheep management such as reducing harassment should be considered. Chronic stress from harassment on sheep populations at high densities, low mobility, and low planes of nutrition may produce catastrophic die-offs. These additive stress factors may lead to respiratory, or other pathogens, reverting from a regulating immune system dependent effect to an immune system independent associated die-off.

SUMMARY

Current management of bighorn sheep does not appear effective. Bighorn sheep numbers continue to decline. If we are to more

Management strategies based on reducing population density and increasing mobility, rather than increasing forage quantity or quality should produce more effective results. Increasing area of habitat rather than increasing forage values in existing sheep habitats should be considered if increasing and stabilizing numbers of bighorn sheep is the desired goal. However, research on the role disease and parasites play in the population dynamics of wild sheep is also needed. The effect of disease on bighorn sheep populations cannot be determined until its interrelationship with population density and nutrition are measured and understood.

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