Chapter 4.

NON-INFECTIOUS DISEASES

Introduction

Albert W. Franzmann

Wildlife disease interest, activity, and reporting have primarily concentrated on parasitology and infectious diseases; disease entities with specific and identifiable etiologies. In the relatively new field of wildlife diseases this is understandable, and is based upon modern trends in human and veterinary medicine. With the discovery of pathogenic agents in the 19th century, a narrowing of the definition of disease from the time of Hippocrates was embraced. Hippocrates defined disease as disharmony within the body, between the body and mind, and between man (or animal) and the environment. The 20th century redefinition espoused that disease resulted from a collision between a pathogenic agent and a susceptible individual.

The consequence of concentrating on specific etiology in disease has led to emphasis on cure and not prevention. Thereby, the focus was on individuals rather than populations. The pendulum has made its full swing, and many researchers and clinicians today recognize the falacy of a narrow interpretation of disease (Dixon 1978).

One of our goals in this compendium is to make the wildlife disease student cognizant of the broad definition of disease. Wildlife diseases, in particular, are related to populations of animals and their response to environmental forces. Involvement in wildlife diseases generally concerns disharmony syndromes rather than diseases with specific etiologies. Difficulty in identifying causes of disease without a specific etiology and, in the past, a lack of research effort on disharmony syndromes has resulted in a poor understanding of mechanisms involved. For example, the most common disease of free-ranging mammal populations is malnutrition, yet many times this syndrome is not recognized, and the discovery of specific pathogens secondarily associated with the syndrome are labeled as causative. This is not to infer that diseases with a specific etiology do not regularly occur, in fact some non-infectious diseases as well as infectious diseases do have an identifiable single etiology. However, most non-infectious diseases of free-ranging animals are primarily environmentally related, and can be broadly considered as resulting from habitat alteration by natural, mechanical or chemical means resulting in stress, deficiencies or toxicities culminating in impairment and possibly death.

In this treatise we have classified the non-infectious diseases of land mammals as metabolic diseases, stress and degenerative diseases, diseases of physical and chemical origin, and congenital and miscellaneous non-infectious diseases.

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Malnutrition and Starvation

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Introduction and History

Malnutrition (poor or inadequate nourishment) and it's negative consequence--starvation, are the most prevalent disease syndromes in free-roaming mammal populations. Both can occur at any time of year. Winter starvation is generally associated with ungulates and other herbivores dependent upon vegetation for food. Some carnivores, omnivores and winter hibernators may experience greater food shortages during summer months. For example, wolves (Canis lupus) on the Kenai Peninsula depend primarily on moose (Alces alces) for food, and are aided in their predation by deep crusted snow and peak pack organization. During summer the pack is less organized (whelping and pup raising) and of course there is no snow. Weight, general condition, and blood parameters in summer are lower than in winter from Kenai Peninsula wolves (Paterson and Franzmann, unpub. data).

The question may arise as to why concern ourselves with a natural phenomenon over which we have little control? Persons concerned with wildlife diseases must be able to recognize malnutrition and starvation in animals. Other diseases may be predisposed by malnutrition, and judgement errors in diagnosis may be made. Conversely, other diseases may predispose malnutrition and starvation and must be recognized.

We will not dwell on the basic causes of malnutrition and starvation which stem from lack of quantity and/or quality of available food because this involves a complex of many factors related to animal habitat requirements. An understanding of basic ecological prin-

ciples (Odum 1971, Smith 1966) and wildlife management (Dasman 1966, Leopold 1933) is necessary to background the causes of food shortages in free-roaming populations.

Malnutrition and starvation are as much a part of Alaskan mammal population dynamics as anywhere; no populations are immune. In fact, with many mammal populations at the northern limits of their distribution in Alaska, starvation may have a relatively greater impact than elsewhere.

Signs--Live Animal

The best measure of the success of a population is that population's productivity as reflected by reproduction, survival and growth of young, and recruitment of breeding animals into the population. Measurement of these qualities may be difficult, but in some populations these data are obtained, particularly for game animals by state management agencies. These data may be important in developing a case history, and perhaps critical when extreme situations occur. For example, in the early 1970s nearly 100% moose calf mortality was experienced on the Kenai Peninsula due to persistence of snow depths which limited available food.

Fluctuations of animal populations that are more regular than one would expect by chance are called cycles. The 2 most common in Alaska are the 3 to 4 year brown lemming (Lemmus sibiricus) cycle and the 9 to 10 year snowshoe hare (Lepus americanus) and lynx (Felis lynx) cycles (Smith 1966). Many theories have been advanced concerning the cause of these cycles, and other animal cycles of

less definite regularity. Nevertheless, lack of food supply and subsequent starvation are integral parts of the mechanism, and knowledge of the timing of these events is helpful in wildlife disease assessment.

Early signs of malnutrition may formulate from observing the behavior of a population coupled with a good understanding of the habitat requirements of the population. In ungulates for example, if the animals are concentrating their feeding activity on vegetation not generally considered a select food or known to be of low nutritional quality, we have a clue that malnutrition may occur. Behavioral aberrations such as animals moving and migrating out of traditional use areas may signal food shortages. Increased aggressive behavior as well as timid behavior may add to our case history.

Feeding and behavioral aberrations of a population do not provide a diagnosis, but provide a sound history for the case. It becomes apparent that the observer must have a firm knowledge of the ecology of the species in question to build a diagnosis. Clues from the population history ,may be subtle, but they may be so obvious that overlooking them would be disastrous.

A physical examination of the live animal may reveal signs that reflect malnutrition, such as; lack of fat cover, poor muscle development, general weakness, poor hair or fur coat, dry and flaky skin and relatively decreased antler and/or skeletal size. A condition scale was developed for white-tailed deer (Odocoileus virginianus) (Robinson 1960) and adopted and used for moose which takes into account the physical characteristics and attitudes of the animal and classifies the animal on a scale from 1 (poorest) to 10 (best) (Franzmann et al. 1976).

Impaired growth or loss of hair is a common and early non-specific response to many nutritional deficiencies in animals (Flesh 1954). Any reduction in food consumption will affect hair growth however, some specific deficiencies have been identified. These deficiencies include; vitamins A, B complex, and E, and mineral deficiencies of copper, zinc and iron (Ryder 1958).

Reindeer (Rangifer tarandus) introduced to St. Matthew Island, Alaska increased from 29 to 6000 over 19 years and subsequently suffered a crash die-off. Body weights following the crash decreased by 38% for adult females and 43% for adult males (Klein 1968).

Sampling and analysis of blood components may further assist in identifying malnutrition. Studies of moose blood chemistry and hematology values have provided a tool for comparative evaluation of populations (Franzmann and LeResche 1978). Certain blood values (packed cell volume, hemoglobin, calcium, phosphorus, total protein, albumin, beta globulin and glucose) were used to rank relative condition of moose populations in Alaska. Using these condition influenced blood parameters, the following blood levels in adult moose were concluded to represent moose in average or better condition: packed cell volume--50%, hemoglobin--18.6 g/dl, calcium 10.4 mg/dl, phosphorus 5.2 mg/dl, total protein--7.5 g/dl, albumin--4.5 g/dl, beta globulin--0.7 g/dl and glucose--140 mg/dl (Franzmann and LeResche 1978). Levels lower than these from a moose population should alert the observer that there is a physiological aberration. Blood urea nitrogen levels reflect protein intake and cholesterol levels reflect saturated fatty acid intake (Coles 1967). Blood urea nitrogen levels decrease as protein intake decreases

to a point where catabolism occurs. At this time body protein tissue rather than food intake, is used to supply the energy needs of the animal. A decrease in albumin levels may occur with increased catabolism (Dimopoullos 1970).

Signs--Post Mortem

Fat deposition in most ungulates first disappear over the rump, chronologically followed by the disappearance of subcutaneous fat, kidney and mesenteric fat and pericardial fat (Harris 1945). The last fat deposits to disappear are those of bone marrow (Cheatum 1949), and consequently measuring this fat reserve has been utilized to reveal condition of ungulates (Anderson et al. 1972, Franzmann and Arneson 1976, Greer 1968). Other indices of carcass fat such as bled carcass weight, eviscerated carcass weight, kidney fat index, carcass density, percentage of carcass fat, and depth of back fat have been used to evaluate ungulate body conditions (Anderson et al. 1972). Elk (Cervus elaphus) were considered in excellent condition with femur marrow fat of 80% or more and in poor condition below 20% (Greer 1969). Femur marrow fat values below 10% dry weight may reliably identify moose that have starved (Franzmann and Arneson 1976). Fat deposits can be used as a direct measure of an animal's condition by reflecting its physiological adjustment to the environment (Riney 1955).

Post mortem examination of the quantity and quality of stomach, rumen, or digestive tract contents may assist in assessing potential malnutrition. Skeletal ratios were used to reflect range related growth differences in black-tailed deer (Odocoileus hemionus) (Klein 1964). Histopathological changes in starved Wisconsin white-tailed deer were described as; abundant hemosiderin in the spleen, decreased fat in marrow, a reduction in number and size of follicles in the spleen and fatty degeneration of the heart (Rausch 1950). Signs and causes of neonatal starvation were reported for white-tailed deer (Cook et al. 1971, Verme 1962) and elk (Cowan 1950).

Diagnosis

Some or all of the above signs may be seen with a variety of diseases with or without a specific etiology. Malnutrition accompanies or may predispose many specific disease syndromes, but it also accompanies a lack of quantity or quality of food intake. It is therefore important that infectious and parasitic diseases be eliminated as causative. Malnutrition confirmation may be strengthened by the history of the population as reflected by reproductive success, behavioral and feeding characteristics and seasonal or cyclic period. Physical examination of the animal and subsequent clinical pathology results will support a malnutrition assessment. Post mortem signs as outlined will provide additional evidence when a dead animal is presented, or when collecting a sample from the population is possible.

Specific forms of malnutrition which begin to attain a specific etiology have been identified and may be the next progression in a diagnosis. Making a specific diagnosis as to a component of nutritional intake entails a detailed diagnostic procedure as outlined for the copper deficiency syndrome in moose (this chapter), and will serve as a guide to diagnosis of other potential mineral deficiencies as outlined for domestic animals (Hays and Swenson 1970, Nicholas and Egan 1975, Underwood 1977).

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Significance and Control

The malnutrition/starvation syndrome is the most prevalent disease affecting Alaskan wildlife. Control of malnutrition and starvation in wildlife populations implies minimizing losses, because some factors influencing this syndrome are not controllable (weather, natural disasters). The application of sound principles of wildlife management to wildlife populations as each situation is presented may improve the welfare of that population and thereby minimize losses from malnutrition and starvation. Various procedures to improve wildlife habitat have been successful and described (Wolfe 1978). Winter feeding of free-ranging ungulates has been done regularly for elk in Wyoming and Washington. The validity of this controversial practice has been reviewed (Mautz 1978).

There is no simple solution to the malnutrition/starvation syndrome and this exemplifies the primary messages of this treatise; (1) an understanding of ecology and wildlife management is basic to wildlife disease investigations, and (2) an understanding of the population from which the animal of concern comes is essential.

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Rumen Overload

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Introduction and History

Rumen overload, often called acute overeating, acute indigestion, rumen impaction or ruminal acidosis, results from an abrupt diet change from roughage or browse to large quantities of readily fermentable carbohydrate in ruminant animals (Siegmund 1973). It is characterized by increased acidity in the rumen (Ahrens 1967). Chemical rumenitis may develop in animals that survive the metabolic acidosis of the acute phase (Blood and Henderson 1968).

This syndrome is well recognized in domestic ruminants and was described in white-tailed deer (Odocoileus virginianus) (Wobeser and Runge 1975). The condition has been associated with "rescue" operations of starving wildlife where ruminants are fed readily fermentable carbohydrate crops (corn, hay, wheat, etc.) after they have exhibited signs of malnutrition (Mautz 1978). The result is generally death. In Alaska, the condition was observed by the author in late winter 1972-1973 in a moose (Alces alces) calf in a "rescue" attempt. The syndrome has most likely occurred in other Alaska ruminants and may potentially occur, but winter feeding "rescue" operations and standing crops are not common.

Signs and Diagnosis

An animal with rumen overload will be anorexic (off feed), dull and exhibit a firm, enlarged rumen (Fincher and Fox 1963). If predisposed by malnutrition, various signs associated with malnutrition may be detected. Diagnosis in wildlife is most often made at necropsy, and is characterized by a rumen containing a large amount of fluid plus grains (Wobeser and Runge 1975). The content is acidic (pH 3.0 to 6.0) and has a distinct sour odor (Wobeser and Runge 1975). When rumenitis develops, the rumen is inflamed and may have signs of secondary infections (Wobeser and Runge 1975).

Significance and Control

In Alaska, the occurrence of rumen overload is undoubtedly rare, but as the human population increases we will undoubtedly see an increase in efforts to "rescue" starving free-ranging animals resulting in an increased incidence of rumen overload. Secondary control is inherent in understanding the management of starving animals and the consequences of improper feeding of these animals, but primary control lies with application of sound wildlife management principles to minimize starvation in free-ranging ruminants.

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Metabolic Bone Disease

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Introduction and History

Fowler (1978) described metabolic bone disease as a prolonged deficiency of calcium, vitamine D, or an improper calcium/phosphorus ratio. He has an excellent outline of the disease syndrome and should be referred to for more detailed information.

Conditions associated with the syndrome osteoporosis (porous bone), osteomalacia (soft bone), rickets (disturbed bone formation), fibrous osteodystrophy (defective bone formation) and secondary hyperparathyroidism (enlarged parathyroid gland) are more often seen in caged animals, but may occur in free-roaming species, particularly carnivores. Osteomalacia was reported in a captive coyote (Canis latrans) in Alaska (Dieterich and Van Pelt 1972) and secondary hyperparathyroidism and fibrous osteodystrophy were reported in captive Alaskan red fox (Vulpes vulpes) (Van Pelt and Caley 1974). Rickets and hyperparathyroidism were detected in arctic fox (Alopex lagopus) trapped on St. Paul Island, Alaska (Conlogue et al. 1979).

Signs and Diagnosis

The metabolic bone disease syndrome exhibited by an individual animal depends on the species, age and duration of deficiency, degree of deficiency and type of deficiency (Fowler 1978). Details of the complexity of signs and diagnosis have been described (Fowler 1978).

Significance and Control

The incidence of metabolic bone disease is not common in caged or captive wild mammals, but is rarely seen in free-roaming animals. The likelihood of a bone deforming disease requiring a relatively long development time being observed in a wild population is lessened when predators and scavengers are present.

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Copper Deficiency

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Introduction and History.

Copper deficiency (hoof overgrowth syndrome) in a subpopulation of Alaskan moose (Alces alces gigas) on the Kenai Peninsula was identified in 1974 based upon hair, blood, hoof and browse mineral element analyses (Flynn and Franzmann 1974, Flynn et al. 1977, Franzmann et al. 1975, 1977, 1974). Clinical manifestation of the disease is exemplified by the hoof overgrowth syndrome (Fig. 1) and possibly by a decline in reproductive rates (Flynn et al. 1977). The Alaskan moose is the only Cervidae in which a copper deficiency has been confirmed. Copper metabolism studies in white-tailed deer (Odocoileus virginianus) and red deer (Cervus elaphus) have been reported (Schroeder et al. 1966, Tolgyesi and Bencze 1970), but clinical signs were not noted nor was a deficiency confirmed. George K. Davis (pers. comm.), University of Florida, indicated that the hoof overgrowth syndrome was frequently observed in cattle in the Florida Everglades, and white-tailed deer in the area around Perry in Taylor County, Florida. In both areas the soil is recognized as being copper deficient.

Copper deficiency in laboratory and domestic animals has been extensively studied and is manifested by anemia, bone disorders, neonatal ataxis, impaired pigmentation and keratinization of hair and wool, infertility, cardiovascular disorders, diarrhea and impaired fatty acid metabolism (Kramer and Kozlowski 1960, Kubota 1974, Schroeder et al. 1966, Scotter 1971).

Signs and Pathogenesis

The hoof overgrowth syndrome in moose has been sporadically reported on the Kenai Peninsula (Fig. 1). An overgrown hoof is on display at the Damon Museum in Soldotna. Hunters occasionally report a moose with this condition, and several have been trapped and immobilized at the Kenai Moose Research Center. Identification of this condition may be minimized where natural predation occurs, in that these animals would be more susceptible to predation. Nevertheless, the incidence is low, perhaps less than 1% of the population. Reduced reproductive rates in
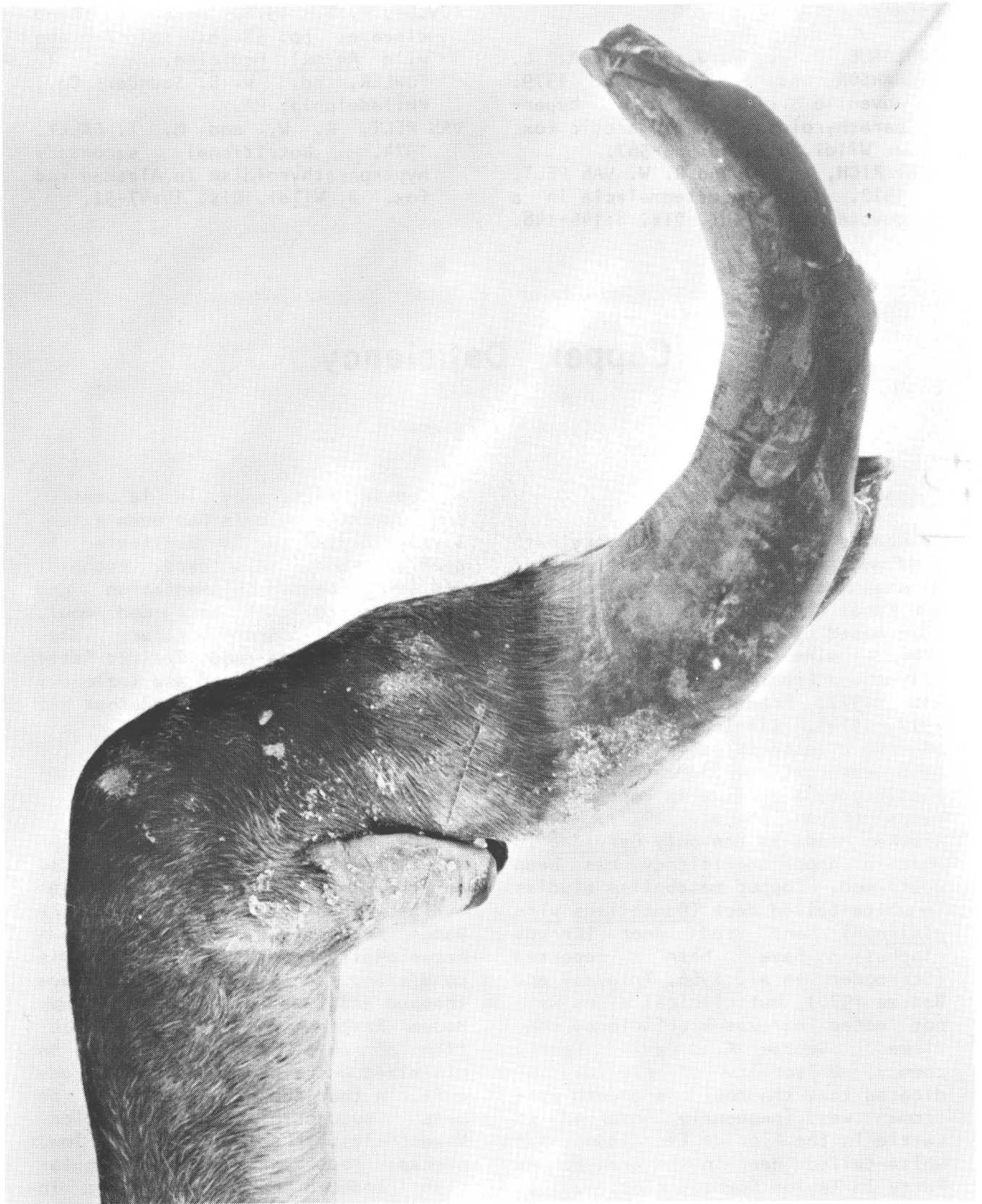
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Figure 1.   Hoof overgrowth from copper deficiency in an Alaskan moose
(A. W. Franzmann).

a population is a difficult facet of this deficiency to evaluate in a wild population.

This deficiency, as with most deficiencies in a free-ranging population, would not generally be identified by clinical signs. More likely, the identification would come as a result of monitoring mineral metabolism via hair element analysis (Franzmann et al. 1977, 1974). This was the mechanism by which the potential for copper deficiency in moose on the Kenai Peninsula was identified. Three years of moose hair analyses indicated a low copper status in this population. Confirmation of the deficiency was based upon further studies of both the animal and plants consumed by moose.

Hoof overgrowth is a result of copper deficiency and subsequent interference with the establishment of a proper matrix in the formation of "hard" keratin (Marston 1952). The mechanism of copper deficiency interfering with fertility is not thoroughly understood, but low fertility in cattle grazing on copper deficient pastures has been reported (Underwood 1977).

Diagnosis

Preliminary information from hair mineral element analysis of a population may indicate the potential for copper deficiency. Hair is an ideal recording filament which reflects intake of various elements (Flynn et al. 1975a, 1974). Hair should be plucked from the shoulder hump of Cervidae and a bundle 2 cm in diameter should be collected, air-dried and identified. Samples are analyzed by atomic absorption spectroscopy, and an array of up to 18 elements may be analyzed (Franzmann et al. 1976). Baseline hair element

values from wild animals are presently available for only moose (Franzmann et al. 1977) and snowshoe hare (Lepus americanus) (Wood 1977). Researchers in Alaska are presently accumulating hair element values from black bear (Ursus americanus), brown bear (Ursus arctos), wolves (Canis lupus) and coyotes (Canis latrans) (Ballard and Spraker 1979, Franzmann and Schwartz 1978a, 1978b). Baseline values from species of concern are needed to help assess relative deficiency states.

Clinical signs of copper deficiency are an essential part of the diagnostic procedure. If a hair element profile for a population appears low in copper, clinical manifestation of hoof overgrowth, impaired fertility, and possibly signs recognized in domestics may be identified (Underwood 1977).

Confirmation of copper deficiency requires eliminating the possibility of copper metabolism interference by cadmium, iron, molybdenum and zinc (Flynn and Franzmann 1974, Flynn et al. 1975a, 1977). In addition to low hair copper levels, an important indicator of copper deficiency is blood ceruploplasmin levels. These 2 copper markers have been used to indicate copper problems even when clinical signs were not evident (Flynn et al. 1977). Additional confirmation can be made by analyses of the affected hoof which would have significantly lower copper and sulphur levels than a normal hoof. Sulphur levels are low due to interference in the disulphide bond from the copper deficiency (Flynn et al. 1977). The tensile strength of moose hair with low copper levels is significantly less than hair with normal copper levels (Flynn et al. 1977).

Analysis of plants browsed by moose, and the respective soils may

also support the diagnosis. Moose browse from the Kenai Peninsula had a mean copper value of 5.6 ppm which was 5.3 to 6.9 times lower in copper than for comparable plant species in Minnesota (Franzmann and Schwartz 1978b).

Significance and Control

Copper deficiency in moose has potential population significance in that basic physiologic processes may be impaired (Hoekstra et al. 1974, Kirchgessner 1977, Mills 1970, Nicholas and Egan 1975, Underwood 1977). Identifying these deficiencies in a population is difficult as the signs may be subtle and effects confused with other conditions. The important consideration is that one must be aware that they can occur, and when a population is showing signs of poor productivity, the possibility of deficiencies existing should not be overlooked. Copper deficiency in moose has been used as an example since it has been identified in Alaska.

Classic symtomatology associated with deficiencies created artificially in a laboratory are not likely experienced in wild populations. It is also unlikely that we will observe "barnyard" type deficiencies in free-ranging animals. What are the effects of subtle deficiencies that do not exemplify themselves with known signs, but may affect the well being of the animal or animals and possibly interfere with reproductive success or calf viability?

Essential mineral elements are classified as macro-elements and micro or trace-elements identified. The classification difference corresponds to concentration in tissues, with the trace elements generally expressed in concentrations of parts per million (ppm) and macro-elements in percent. The classic definition of a trace element is any element that exists in concentration equal to or less than Fe in the body, with iron being the first of the trace-elements. The essential macro-elements are calcium (Ca), chlorine (Cl), potassium (K), magnesium (Mg), sodium (Na), phosphorus (P) and sulphur (S) and they have long been known to be required (Church 1971). The essential micro-elements are iodine (I), iron (Fe), copper (Cu), zinc (Zn), manganese (Mn), cobalt (Co), molybdenum (Mo), selenium, (Se), chromium (Cr), tin (Sn), vanadium (V), fluorine (F), silicon (Si) and nickel (Ni) (Schwarz 1974, Underwood 1977). Twenty additional elements should be considered and investigated as possibly essential (Schwarz 1974).

To elucidate the physiology of these elements and the symptomatology associated with their deficiencies would require a symposium in itself. We will only generalize and state that they all have been established as essential to normal function and their deficiencies are associated with malfunction in basic physiological systems. Some symptoms associated with deficiencies of these elements are listed in Table 1. In addition, all essential elements have recognized toxic levels and associated pathology. The field of mineral metabolism is so complex and interrelated with itself and with other systems, primarily neural and hormonal, that no element can be considered alone. Two classic examples of this are the detrimental effect of high molybdenum intake on copper metabolism (Underwood 1977), and the shift of the Ca: P ratio influenced primarily by phosphorus levels. There are other examples of these types of interractions between minerals which confound our understanding of mineral metabolism.

So what can a wildlife manager do, and why should he or she add the

burden of this potential problem to the many he already has?

First of all he or she need not be concerned with the specifics of mineral element metabolism, but should be aware of the possibility of deficiencies. More specifically, the manager should be concerned with the possible consequences that land use and management practices may have regarding mineral utilization of the animal in question. Our present state of knowledge will not specifically guide the manager, but awareness of the potential alteration of mineral availability in general, may influence management and land use decisions. Following, are some examples that illustrate this.

1. Land-Use Planning and Management. Moose may assimilate and store a majority of their mineral requirements within the short span of summer. The bulk of their mineral requirements are obtained from primary plant species, but they may require additional supplemental sources such as licks. On the Kenai Peninsula and elsewhere (Krefting 1974), a minor source of total food intake comes from submerged vegetation; however, results from other studies (Kubota et al. 1970, Oldemeyer et al. 1975) show these plants to be high in mineral content. On Isle Royale, it was reported that aquatic macrophytes had 500 times more Na than found in terrestrial vegetation (Jordan et al. 1973). Land use planning and management decisions should consider these potentially important mineral sources (licks and submerged vegetation) and not exclude access and use by moose.

2. Fire Supression and Prescribed Burning. Whatever the actual cause, there does seem to be a release of nutrients and a fertilizing effect of fire on the organic soils in Alaska (Viereck 1973). Unfortunately, little is known regarding the uptake

of micro-elements following fire, but the increase in macro-elements has been noted (Lutz 1956, Scotter 1971). Perhaps more important is the demonstrated population response to fire, such as by moose on the Kenai Peninsula, Alaska (Spencer and Hakala 1964).

Changes in soil and plant composition and quality following fire are influenced by many variables such as parent material, moisture, plant composition prior to fire, intensity of fire and climatic events following fire. Nevertheless, the generalization can be made that browse quality and palatability improve and moose populations respond. Mineral content, including trace minerals, must be considered a positive factor in these events.

3. Forestry Practices. The alteration of mineral cycling in forestry practices is influenced by many factors; however, parent material and subsequent weathering basically determine availability in the forest. The mineral content of hardwood litter usually is higher than that of conifer litter, and bark usually contains 3 to 10 times as high a concentration of minerals as wood (Kramer and Kozlowski 1960). Removal of overstory permits revegetation in an area and browsing animals respond to the increase in available vegetative biomass; however with harvest, appreciable amounts of nutrients are removed (Smith 1966). The long term effects that removal of the forest overstory has on mineral cycling and subsequent availability to animals is difficult to establish, but the ecosystem can remain productive only if the nutrients withdrawn are balanced by an inflow of replacements (Smith 1966).

4. Vegetative Rehabilitation and Fertilization. The variation among plants in their ability to assimilate mineral elements may be cause for

consideration in selection of areas for vegetative rehabilitation or chemical fertilization of moose browse. These practices are stimulated by new information on moose mineral requirements and mineral status of the area.

It would not be wise to invest great sums of money on mechanical or chemical rehabilitation in areas where vegetative response may favor plants that poorly assimilate certain mineral elements. We could instead, divert our activity to an area that would stimulate plants that have the ability to assimilate desirable elements.

Much research is required to assess the various effects our management plans have on mineral cycling, and even more is required to assess the often subtle effects mineral deficiencies may have on a population. The advent of atomic absorption spectroscopy permits intensive mineral element investigations. Perhaps the lack of definitive information regarding wildlife mineral metabolism, and the general lack of information on the mineral aspect of forage quality for wildlife, stems from our previous inability to study minerals on a practical level. Persons planning research projects should consider adding to the sparse information available relative to minerals and moose by incorporating this into their studies, when feasible. The need for mineral metabolism information is certainly not intrinsic to moose, and these studies on other species should be encouraged.

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TABLE 1

An Outline of Some Recognized Mineral
Deficiency Symptoms and Signs

1. MACRO-ELEMENTS

Calcium (Church 1971)

a. Skeletal disorders (osteomalacia, osteodystrophy and
   ricketts)
b. Anorexia and weight loss
c. Lowered milk production
d. Tetany and muscular dysfunction

Chlorine (Hays and Swenson 1970)

a. Principal anion in body fluids
b. Alkalosis

Potassium (Church 1971)

a. Anorexia and weight loss
b. Listlessness and weakness
c. Pica (depraved appetite)
d. Impaired response to disturbance
e. General stiffness
f. Kidney degeneration
g. Histologic change in muscles

Magnesium (Church 1971)

a. Opisthotonus--retracted head--muscle tremors--convulsions
b. Ataxia
c. Hypersensitivity
d. Increased heat production and fall in energy retention
   due to tonic muscular activity
e. Reduced appetite
f. Rumen flora changes and reduced digestibility
g. Anemia and jaundice
h. Impaired blood clotting
i. Liver damage and reduced serum albumin and alpha and
   gamma globulins
j. Serum enzyme changes

Sodium (Church 1971)

a. Pica
b. Decrease body weight
c. Anorexia
d. Listlessness
e. Harsh skin
f. Tetany--collapse and death

Phosphorus (Church 1971)

a. Anorexia and weight loss
b. Pica
c. Listless and dull
d. Osteomalacia and ricketts
e. Impaired fertility in females

Sulphur (Church 1971)

a. Anorexia and weight loss
b. Emaciation
c. Pica
d. Hair and wool growth impaired
e. Excessive lacrimation and profuse salivation
f. Dullness and weakness
g. Heart, liver, skeletal muscle and spleenic histologic changes
h. Reduced digestibility

2. MICRO OR TRACE-ELEMENTS

Iodine (Church 1971)

a. Thyroid deficiency (enlarged thyroid--goiter)
b. Lowered metabolic rate
c. Birth of hairless, weak or dead young--cretinism
d. Reduced reproductivity (suppressed estrus in females and reduced libido in males)
e. Relatively non-toxic

Iron (Church 1971)

a. Anemia
b. Anorexia and weight loss
c. Listlessness
d. Gastritis
e. Relatively non-toxic

Copper (Church 1971)

a. Anemia (iron transfer related)
b. Severe diarrhea
c. Depigmentation of hair and defective keratinization of wool and hooves
d. Ataxia and paralysis
e. Fibrosis of cardiac muscles and heart failure
f. Bone deformities (osteoporosis)
g. Reduced fertility and impaired reproductive performance
h. Toxic symptoms recognized

Zinc (Church 1971)

a. Skin irritability, inflammation and parakeratosis
b. Difficult conception
c. Abnormal estrus and cystic degeneration of ovary
d. Retained placenta
e. Excessive salivation
f. Impaired digestibility by reduction of volatile fatty acids
g. Cessation of spermatogenesis
h. Growth retardation
i. Anorexia
j. Impaired wound healing
k. Relatively non-toxic

Manganese (Church 1971)

a. Impaired estrus and conception
b. Enlarged joints and stiffness--skeletal abnormalities
c. Weakness and impaired growth
d. Pica
e. Liver degenerative changes
f. Ataxia of newborn
g. Relatively non-toxic

Chromium (Church 1971)

a. Impaired growth and longevity
b. Disturbances in glucose, lipid and protein metabolism
c. Eye disorder--corneal opacity
d. Toxic symptoms recognized

Cobalt (Church 1971)

a. Listlessness and emaciation
b. Anemia
c. Anorexia
d. Depressed synthesis of B12 in rumen
e. Relatively non-toxic

Molybdenum (Church 1971)

a. Closely related to Cu and S. High levels depress Cu and S
b. Renal calculi
c. Relatively non-toxic, but high levels depress other elements

Selenium (Church 1971)

a. Nutritional muscular dystrophy
b. Impaired fertility
c. Persistent diarrhea
d. Depressed growth rate
e. Associated with diseases responding to vitamin E therapy
f. Toxic symptoms recognized

Vanadium (Hopkins 1974)

a. Reduced body and feather growth
b. Impaired reproduction and survival of young
c. Altered RBC levels and iron metabolism
d. Impaired hard tissue metabolism
e. Altered blood lipid levels
f. Toxic symptoms recognized

Fluorine (Messer et al. 1974)

a. Retarded growth rate
b. Infertility
c. Anemia
d. Toxic symptoms demonstrated

Silicon (Schwarz 1974)

a. Postulated as structural element in metabolism
b. Growth stimulating effect in rats
c. Relatively non-toxic

Nickel (Nielsen 1974)

a. Suboptimal reproductive performance
b. Reduced oxidative ability in liver
c. Toxic symptom recognized

Tin (Schwarz 1974)

a. No definite signs recognized, but it has been demonstrated
    to have a growth stimulant effect
b. Relatively non-toxic

Arterial Disease

Robert A. Dieterich

Arterial disease occurs in wildlife, but it is not a major cause of death as it is in humans. Actual occlusion of the coronary blood vessels that provide the heart with nutrients, oxygen, etc. is very rare in animals. Two types of arterial disease have been documented in Alaskan wildlife. Atherosclerosis has been found in caribou and reindeer (Rangifer tarandus). Atherosclerosis is a disease of large and medium sized arteries with deposits in the intima of the vessel of yellowish plaques. Arterial changes observed in caribou were quite similar to changes found in man and other animals (Wiggers et al. 1971). It is the opinion of this author that reindeer and caribou living on natural diets do not develop lesions of atherosclerosis significant enough to be a serious threat to their life (Dieterich and Luick 1979).

Medial arterial sclerosis has been diagnosed in captive beaver (Castor canadensis) in Alaska fed cuttings of aspen (Populus tremuloides Michx.) and Purina Lab Chow. Unconfirmed reports indicate that this condition may be present in some wild beavers within the state but only in a mild form. In this type of arterial disease, the arteries are firm and very prominent. When transected, the arteries remain distended and their walls appear to be calcified. If vascular occlusion has not occurred, the animal may be completely healthy or, if partial occlusion has occurred, there may be some signs of limited blood flow to affected areas (Dieterich 1969).

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General Adaptive Syndrome

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Introduction and History

In any discussion of diseases in wild mammals, and particularly of non-infectious diseases, stress and the general adaptive syndrome (GAS) must be considered. Stress was defined as the cumulative response of an animal resulting from interaction with its environment via receptors (Selye 1973). The definition alone implies the importance of this syndrome for understanding the mechanism of any disease. A simplified model of the stress mechanism begins with receptors (sensory nerve terminals) which detect change in the animal's environment. The message travels to the central nervous system where a response is transmitted either through the nervous system or through the endocrine system (hormones) to effectors. Effectors are tissues which respond to counteract the challenges to the receptors. The resulting response of the animal depends on the type, duration and intensity of the challenge. Overstimulation· may result in pathologic changes in the body that Selye (1946) termed general adaptive syndrome (GAS). He outlined 3 phases of the syndrome: alarm reaction, stage of resistance and exhaustion phase. This exhaustion phase is of primary concern because it is the period in the GAS when direct effects of overstimulation occur (primarily from excessive corticoid production), and when derangement of the animals adaptive mechanisms play a decisive role in the development of many diseases (Seyle 1946, 1956). The animal during this period is more vulnerable to other challenges. The syndrome occurs in all mammals.

Signs and Diagnosis

Prolonged stimulation and maintenance of the exhaustion phase of the GAS results in adrenocortical hypertrophy (enlarged adrenal gland), lymphoid aplasia (lymph tissue shrinkage), gastrointestinal ulceration and arterial calcification. These lesions are found in man, experimental, domestic and wild animals (Brodie and Hanson 1960, Fowler 1978, Selye 1950). Some examples from wild mammals include: gastric ulcers in snowshoe hare (Lepus americanus) (Iversen et al. 1972); lymphoid aplasia and adrenal hypertrophy (common lesions of wild animals at necropsy) (Fowler 1978); atherosclerosis in captive rodents (T-W-Fiennes 1965), woodchucks (Marmota monax) (Bond 1970) and beaver (Castor canadensis) (Dieterich 1969); atherosclerosis in free-living brown hare (Lepus europaeus) (Sargent 1974) and in Alaskan caribou (Rangifer tarandus) (Wiggers et al. 1971). In Alaska, an investigation of 34 reindeer and 15 caribou for atherosclerosis and other vascular diseases reported no significant lesions (Dieterich and Luick 1979). Stress induced hemopericardium (an accumulation of blood in the heart sac) was reported in white-tailed deer (Odocoileus virginianus) following its capture (Libke and Mosby 1968). Severe hypoglycemia (low blood sugar) was reported in association with acute stress ("shock disease" or "trap sickness") from trapped snowshoe hare (Keith et al. 1968) and gray squirrels (Sciurus carolinensis) (Guthrie et al. 1967).

Significance and Control

Stress exemplified by the general adaptive syndrome plays a part of every disease process as a predisposing factor and/or a responsive factor. The recognition of this syndrome in medicine is responsible for much of the supportive therapy and nursing care provided a patient. On a population level it is no less important. Good husbandry practices evolved around avoidance of the consequences of this syndrome even prior to it being recognized. In wild populations, again many of the wildlife management principles evolved around the avoidance of the consequences of this syndrome. The capture, handling and transportation of wild animals exemplifies this condition in wild animals (Fowler 1978, Jenkins and Kruger 1975) (also see Capture Myopathy, this chapter). As excitability of Alaskan moose (Alces alces gigas) increased with handling, blood corticoid levels increased significantly (Franzmann et al. 1975). This was a measured physiological response to the general adaptive syndrome.

Control of the consequences of the general adaptive syndrome in wild populations reverts to an understanding of the ecology of the species, and application of management principles based upon that understanding. In capture and handling techniques we must employ all means possible to minimize stress (Fowler 1978).

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Capture Myopathy

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Introduction

Capture myopathy (CM) is a syndrome recognized worldwide in many species of mammals, birds and particularly in ungulates (Fowler 1978). Synonyms for this condition include stress myopathy, overstraining disease and white muscle stress syndrome. It is usually associated with the stress of capture, restraint and transportation of wildlife. The usual symptoms are muscular stiffness, weakness, tremors, ataxia, paralysis, myoglobinuria (coffee-colored urine) and death. These symptoms are associated with asymmetrical muscular and myocardial lesions.

History

Capture myopathy is the result of a physiologic response and therefore the potential of this condition is present in all susceptible species.

The capture of wildlife in Alaska for management and research purposes has increased greatly in the past few years and CM has therefore increased accordingly. Workers in Africa were among the first to recognize the syndrome in the mid 1960s (Basson and Hofmeyr 1975). It has been diagnosed in Alaska in a number of species including moose (Alces alces), reindeer (Rangifer tarandus) and muskox (Ovibos moschatus).

Symptoms and Pathogenesis

The exact mechanisms involved which are responsible for the muscle damage seen in CM are not fully understood but it is believed that severe muscular exertion, straining, prolonged muscular trembling, immobilization and stress all contribute to the development of lesions. Apparently, when there is a sudden stressful increase in muscular activity, muscle glycogen is rapidly

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metabolized to lactic acid (Hadlow et al. 1974). The rate at which lactic acid is produced may exceed the rate at which it is removed in the bloodstream and the resultant accumulation leads to acidosis, muscle coagulation and liberation of myoglobin from damaged cells which then appears in the urine.

The common symptoms observed in affected wildlife are mild to severe muscle soreness (lameness), stiffness, weakness, paralysis particularly of the rear legs (Fig. 1) and death (Chalmers and Barrett 1977, Haigh et al. 1977). These symptoms usually appear 1 to 6 weeks after capture. Most commonly, lesions develop within the first week and consist of fairly well demarcated light greyish-brown areas on the muscles. Hemorrhage may be present due to the rupture of muscle groups. In the later stages of the disease, fibrosis and mineralization of necrotic fibers occurs. These changes are often microscopic but significant. The kidneys may be swollen and darker brown than normal from staining due to the excretion of myoglobin. The urinary bladder may contain myoglobin which colors the urine brown. Many times these changes are not evident to the untrained eye and when death occurs suddenly following capture due to myocardial failure, no lesions are apparent.

When muscle cells are damaged, certain cellular enzymes are released into the bloodstream (Lewis et al. 1977). These enzymes can be measured in the laboratory, and those animals having abnormally high levels are at risk. Serum glutamic oxaloacetic transaminase enzymes are found principally in liver, heart and skeletal muscle. Serum creatine phosphokinase is principally found in skeletal muscle, heart and brain. By obtaining serum from animals suspected of having CM and having it analyzed for the presence of these 2 enzymes, one can have confidence in confirming a tentative clinical diagnosis of CM.

Capture myopathy can occur following both physical or chemical methods of restraint and immobilization. Fear and anxiety can contribute to the development of the syndrome as can hyperthermia.

Significance and Prevention

Capture myopathy can be a significant cause of mortality or disability in wildlife which are being handled by biologists. The fact that this condition can occur several weeks after the initial period of stress must be realized and one can not feel he is not imposing CM losses on captured and released animals simply because they get up and run away (Fowler 1978). Besides outright sudden death due to myocardial failure, there is also death resulting from increased susceptibility to predation or disease in these weakened animals. These types of losses would typically occur several days after capture.

Prevention of overexertion, fear and maintenance of proper thermoregulation are the most important factors to consider. Chases prior to capture are best limited to less than 1 minute in many species when animals are under full flight response. Many professional handlers refuse to pursue an animal after an established safe time has been passed. Frightening noises or activities should be avoided. If severe exertion or stress is inflicted, animals can be treated for shock, and intravenous sodium bicarbonate administered to combat acidosis. When animals are captured and transported, periods of quiet rest should be provided whenever possible.

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Degenerative Arthropathy

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Introduction and History

Degenerative diseases generally refer to tissue deterioration associated with age. These conditions are not prevalent in most free-ranging populations due to natural selective forces and most are observed on post mortem examination.

The most commonly observed condition is degenerative arthropathy, also known as degenerative joint or articular disease, osteoarthritis or chronic arthritis. It is generally associated with aging; however, predisposing factors such as metabolic bone disease and copper deficiency may play a part (Siegmund 1973). Degenerative arthropathy of the hip joint from Kenai Peninsula moose (Alces alces) has been seen during examination of moose skeletal remains by the author. It has been reported in white-tailed deer (Odocoileus virginianus) (Wobeser and Runge 1975), moose (Peterson 1977, Timmermann and Lankester 1978, Wobeser and Runge 1975), black-tailed deer (Odocoileus hemionus) (Cowan 1946), coyote (Canis latrans), x dog (Canis familiaris) (Maham and Gipson 1978), wolf (Canis lupus) (Cross 1940) and black bear (Ursus americanus) (Stecher 1963). It has also been described for a variety of zoo artiodactylids (Boever 1978).

Signs

In live animals, lameness may be witnessed. The affected joint, generally hip or stifle, reveals degeneration of the articular cartilage and varying degrees of osteophytes (bony out-growth) are present.

Diagnosis

Diagnosis in wild animals is primarily based upon post mortem examination of joint demonstrating degeneration of cartilage and osteophyte formation.

Significance and Control

With a limited number of animals in a harvested wild population attaining an aged status, the frequency of occurrence is low; nevertheless, in an unharvested ungulate population such as Isle Royale, Michigan, degenerative joint disease probably plays an important role in increasing the vulnerability of moose to wolf predation by restricting the mobility of the prey (Peterson 1977). This may be true in other populations where aging of a prey species occurs.

A form of control is rendered in ungulate populations where harvesting occurs and where the opportunities for aging are limited.

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Gastric Ulcers

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Gastric ulcers have been observed in Alaskan snowshoe hares (Lepus americanus) during periods of peak populations (Dieterich and Feist 1980). These lesions appear as brown to black depressed areas in the glandular region of the stomach. At times, the lesions are multiple. Blood discolored to a brown color by gastric juices is found on and near the erosions in the stomach wall. Most lesions are small, measuring a few millimeters in diameter (Iverson et al. 1972). Ulcers of varying sizes are the rule. Ulcers are 1 manifestation of the general adaptive syndrome (GAS) (see General Adaptive Syndrome section, this chapter) and are present in other species of wildlife which are undergoing stressful situations. For example, stomach ulcers have been found in nearly 80%

of reindeer (Rangifer tarandus) herded by stressful techniques. The presence of ulcers in the stomach of wildlife can be used as an indication of population stress, but one must be careful to consider all possible causative factors that may be involved.

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ALASKAN WILDLIFE DISEASES

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