# JOINT DISEASE IN UNGULATES WITH SPECIAL REFERENCE TO MOOSE

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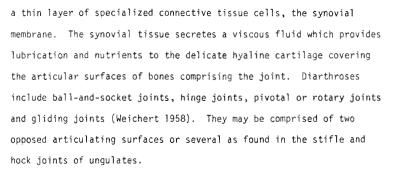
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Abstract: A variety of abnormalities affect the articulating joints of mammals. This paper attempts to review the literature on the major types of joint disease known in ungulates with particular reference to the genus Alces. Data are presented on the prevalence of joint irregularities observed in a sample of lower legs collected from fall, hunter-killed moose. Macroscopic anomalies and wear patterns found on the articulating surfaces of metacarpal joints are described and related to sex and age. The significance of joint disease among free living ungulates is discussed.

#### INTRODUCTION

Joints are a mechanical requirement in a segmented endoskeleton which serve the prime purpose of bearing weight and allowing for motion. In mammals, two distinct types are recognized; fibrocartilaginous or immovable joints (synarthroses) and synovial or freely movable joints (diarthroses) (Sisson and Grossman 1953, Weichert 1958, Boyd 1961). Fibrocartilaginous joints occur between vertebrae where adjacent bones bear on an intervertebral disc. Synovial or movable joints as described by Freyberg (1954) are composed of two bone ends held in apposition by a strong fibrous tissue capsule reinforced by ligaments, and lined with



Joint disease occurs most frequently in the major diarthroses of the skeleton and most, if not all animal species are affected (Fox 1939, Freyberg 1954, Sokoloff 1960, 1969, Boyd 1961, Jubb and Kennedy 1970). Domestic livestock have provided most of the documented cases appearing in the published literature. Unfortunately, relatively little information is available on wild mammals living under natural conditions.

This review is concerned with the occurrence of spontaneous and infectious diseases of the joints in ungulates. They are generally categorized as disease resulting from traumatic injuries, athritis and degenerative changes.

A common type of joint disease results from injury or trauma to the articulation (Freyburg 1954). Sprains subluxation or even complete dislocation may occur when a joint is forced to move beyond its normal limits (Jubb and Kennedy 1970). Stretching of the capsule and synovial tissue causes stiffness, swelling and pain. Sequelae of greater consequence include interarticular haemorrhage, detached calcified synovial membrane (joint-mice) and bone fragments or osteophytes which may penetrate or irritate articular surfaces. Minor sprains



usually heal rapidly leaving no impairment. Severe traumatic injuries seldom repair completely and often initiate degenerative changes which may become debilitating in later life (Neher and Carter 1963; Sokoloff 1969).

Arthritis in animals is restricted by Jubb and Kennedy (1970) to mean an acute or chronic inflammation of the joints caused by a microbial agent. Organisms frequently implicated are Erysipelothrix rhusiopathiae, Streptococcus spp., Haemophilus spp., Staphylococcus spp., Escherichia coli, Mycoplasma spp., Psittacoids, Corynebacterium spp., Salmonella spp., Brucella spp., Shigella equirulis, and Klebsiella genitalium (see Jubb and Kennedy 1970). Invasion of an organism may occur through a wound penetrating directly into the capsule or more commonly, the agent becomes localized in one or several joints (polyarthritis) following a general septicemia. Such haematogenous infections may follow castration or tail cropping or may initiate in the gastro-intestinal tract, the uterus post partum or the umbilicus. Haematogenous infectious arthritis in new-born animals is a common occurrence among domestic species (Sokoloff 1960). Direct spread of infection from adjacent soft tissues into a joint is uncommon (Jubb and Kennedy 1970). The fibrous layer of the joint capsule provides an effective barrier to most organisms with the exception of Sphaerophorus necrophorus causing "foot-rot" in ungulates (Jubb and Kennedy 1970).

The onset of septic arthritis is characterized by effusions of inflammatory exudate, edema, and hyperemia of the synovial tissue. In severe cases, cartilage becomes demineralized with eventual destruction of the subchondrial plates. During the reparative process, the joint capsule frequently becomes filled with fibrous and bony substance,

consequently decreasing the width of the joint space and causing roughened bone surfaces to meet. The organized tissue may remain fibrous or it may ossify producing an enlargement or pannus causing permanent joint stiffness (Sokoloff 1960). A review of the more common infectious arthritic conditions occurring in domestic ungulates is presented by Sokoloff (1960) and Jubb and Kennedy (1970).

Infectious arthritis in wild cervids has been identified and described in the extinct Irish elk (Cervus megaceros) by Spurrell and Day (1958), in moose (Alces alces) by Kozhuklov (1959, 1965) and Dahlberg (1977) and in white-tailed deer (Odocoileus virginianus) by Sikes et al. (1968). Necrobacillosis caused by the bacterium Sphaerophorus necrophorus and affecting the joints and bones of the feet has been reviewed by Rosen (1970). More recent publications on foot-rot include those by Chalmers and Barrett (1974), Runge and Wobeser (1975), and Dingeldein and Wachendorfer (1977). Witter and O'Meara (1970) presented a review of Brucellosis, a disease which frequently localizes in tendon sheaths, joints and other organs where it persists for long periods.

Degenerative joint disease or arthropathy according to Sokoloff (1969) is a "non-inflammatory disorder of movable joints characterized by deterioration and abrasion of articular cartilage and also formation of new bone at the articular surface". Skeletal movement in time subjects the cartilaginous surfaces of articulating bones to varying degrees of wear. This process is accelerated by movements with heavy loads, a decrease in the viscosity of synovial fluid which occurs with age and by mechanical damage to articular surfaces or elements of the joint capsule. Synonomous terms for the disease include degenerative



arthritis, osteoarthritis, arthrosis, degenerative arthrosis and osteoarthrosis. Similarly, degenerative changes in the vertebral column have been termed ankylosing spondylitis, spondylosis deformans, and vertebral osteophytosis (Wobeser and Runge, 1975).

Degenerative changes appear first in the cartilage in the part of the joint which receives the greatest wear and has the poorest nutritive blood supply. Joints of the limbs and spine on which weight is borne, most commonly exhibit earliest and most severe changes. Characteristically, degenerative changes progress slowly enough to allow some functional compensation. Arthropathy may develop in a joint with no history of injury or known etiology. This is referred to as primary degenerative joint disease and lesions are commonly found at more than one site in joints of older individuals. Proliferative lesions may occur in the periarticular tissues, ie. the ligaments, the tendon sheaths, the bursae and in the periosteum. Secondary degenerative joint disease as described by Neher and Carter (1963) and Sokoloff (1969) on the other hand, is initiated by trauma or abnormal skeletal stresses.

Degeneration of joint cartilage follows a definite pattern (Bennett and Bauer, 1931; Callender and Kelser, 1938; Vaughan, 1960). Cartilage loses its sheen and there appear foci of fibrillation, shallow pits and parallel grooves running in the direction of motion of the joint surfaces. Cartilage splits along these grooves and rough, loose hanging threads appear. Eventually cartilage wears thin and in places disappears exposing the underlying bone. With continuing friction the bone develops a thickened, hard, smooth appearance known as eburnation. Osteophytes form at the margins of articular surfaces and later fuse to form large blocks of bone. As the disease progresses with prolonged mechanical



irritation of roughened articular surfaces, the joint capsule undergoes fibrosis with an accompanying thickening of the synovial membrane (Neher and Carter, 1963).

For a comprehensive review of the biology of degenerative joint disease as it affects domestic ungulates, the reader is referred to Sokoloff (1960, 1969), and Jubb and Kennedy (1970). The disease has been described in certain wild ungulates by Rabagliati (1923), Fox (1939), Hamerton (1939), Cowan (1946), Murie (1951), Hansen (1959), Fuller (1961), Jilly (1966), Shelton (1966), Habermehl and Bassewitz (1972), Peterson (1974), Wobeser and Runge (1975) and Runge and Wobeser (1975).

In the present study, only the lower portion of moose limbs (usually one front leg) was available for examination. The literature indicates, however, that the most severe instances of joint damage are likely to occur in the major diarthroses of the skeleton. Nevertheless, few studies of joint disease in moose exist and it might be expected that damage evident in the lower metacarpal joints reflects similar or even more serious impairment in the major articulations of the body.

## MATERIALS AND METHODS

During the period October - December, 1977, successful moose hunters in the North Central Region of Ontario were requested to collect a variety of biological specimens. One front leg severed at the carpal (knee) joint was among the items which also included the head, testes, ovaries, tarsal glands and scrotum. An analysis of hoof wear related to sex and age was made and will be reported by Bubenick <u>et al</u>. in this workshop. The reasonably large sample of lower legs (185) presented the opportunity to initiate a preliminary investigation of joint disease affecting wild moose.

A form including a sketch of the exposed joint surfaces was prepared to facilitate record keeping (Figure 1). Initially each leg was closely examined externally for evidence of cuts or abrasions. Four external measurements including the width and depth of fetlock and pastern joint capsules were made with a set of calipers. A collection reference number, cementum age (Sergeant and Pimlott, 1959), sex and leg position were also recorded. The fetlock and pastern joints were exposed by means of a longitudinal dorsal incision through the hide between the medial and lateral phalangeal bones. The hide was cut back to expose the ligaments surrounding the joints. The bursal sheath and associated tendons were severed to expose the joint cavity and surfaces of the articulating bones. Each of eight cartilaginous surfaces of the fetlock and four of the pastern joints were closely inspected (figure 2). For purposes of describing the location of lesions, the lateral and medial pairs of sesmoid bones were each considered as one surface. Macroscopic anomalies and wear patterns were recorded in a manner similiar to that of Cowan (1946), Freyberg (1954), Jubb and Kennedy (1970) and Wobeser and Runge (1975). Three wear grades were arbitrarily established to categorize each set of fetlock and pastern joints. Briefly, grade A represents either the complete absence of abrasions or those only slightly affected (shallow notches, focal areas of roughness, dark spots). Grade B was applied to moderate abrasions including notches, pits, light grooves and areas of roughness. Grade C identified joints with more severe abrasions (deep grooving, eroded and broken surfaces, fibrous tissue repair, "joint-mice" and grossly torn cartilage).

Legs were available from 143 aged moose (76  $\sigma$ , 67  $\circ$ ). One hundred and thirty-nine of these animals were shot by hunters. A single front-

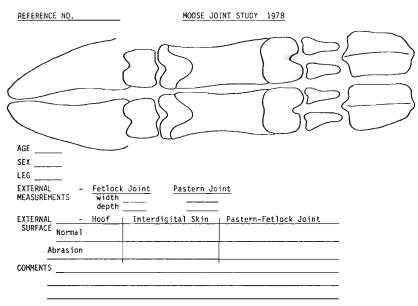


Figure 1. Summary work sheet used to record joint lesions.

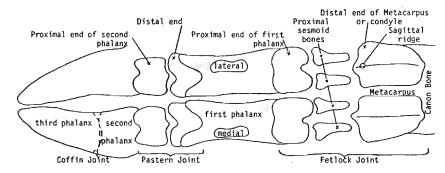


Figure 2. Metacarpel joint surfaces of moose legs examined for cartilaginous lesions.



leg was submitted by most hunters but in some instances pairs of front or rear legs and rarely all four legs were provided. In addition, all four legs were submitted from two vehicle-road killed moose, one found dead of unknown causes and one crippled animal that was dispatched by conservation officers.

Although incidental to the principal study, the clinical histories of two captive moose calves that developed infectious arthritis and a case of foot-rot in one wild moose are included in this paper.

#### **RESULTS**

The frequency of joint damage in relation to sex and age was determined by considering only one front leg from each of 143 moose (Table 1). No (57) or slight (55) lesions (A grade) were observed in 78% (112/143) of the sample. Coincidentally, these figures are identical to the sum of A grade lesions seen in males and females respectively (Table 1). Significant lesions (B & C grade) were found in 22% (31/143) of the legs examined. There was little difference in the frequency of significant lesions in males (25%) and females (18%) of all ages. Although the number of observations was small, the frequency of the most severe grade of damage (C) was more common in females (10%) than in males (4%). Compared by age, significant lesions (B & C) were more common (16%) in young males (<4 years) than in older males (9%). The frequency of such lesions in young and older females was similar (8% and 10% respectively). The external dimensions of the fetlock and pastern joints with articular lesions were similar to those of joints without damage.

The frequency of damaged articular surfaces (A, B or C grades) observed in the fetlock and pastern joints (163 front legs, 22 hind legs) was similar in males and females (Table 2). Surfaces in the



Table 1. Degree of cartilage damage in the metacarpal joints (fetlock and pastern) of 143 moose

		We	Male ar gr			Females Wear grade			
Age	n	A	В	С	n	A	В	. С	
0.5	5	4	-	1	7	7	_	_	
1.5	33	26	7	-	28	25	2	1	
2.5	9	8	1	-	10	10	-	-	
3.5	11	8	2	1	4	2	1	1	
4.5	3	2	1	-	4	3	1	_	
5-10	11	8	2	1.	11	6	1	4	
10+	4	1	3	-	3	2	-	1	
Total	76	57	16	3	67	55	5	7	
	*	75	21	4		82	8	10	

<sup>\*</sup>A = no or slight damage B = moderate C = most severe

Table 2. Number of damaged articular surfaces in the pastern and fetlock joints of wild moose compared by sex

No. legs examined	Total surfaces checked*	No. of surface medial	es with lesions lateral	Total no. surfaces with lesions	Percent
85 front of	340	9/170	8/170	17/340	5
5 rear o <sup>7</sup>	20	0/ 10	0/ 10	0/ 20	0
78 front ₽	312	12/156	12/156	24/312	8
17 rear 🍳	68	3/ 34	2/ 34	5/ 68	7
85 front o	510	66/255	75/255	141/510	28
5 rear of	30	6/ 15	2/ 15	8/ 30	27
78 front ♀	468	57/234	65/234	122/468	26
17 rear ♀	102	20/ 51	18/ 51	38/102	37
	85 front of 5 rear of 78 front \$\frac{9}{17}\$ rear \$\frac{9}{17}\$ s front of 5 rear of 78 front \$\frac{9}{17}\$ front \$\frac{9}{17}\$ front \$\frac{9}{17}\$	examined checked*  85 front of 340 5 rear of 20 78 front \( \frac{9}{4} \) 85 front \( \frac{9}{4} \) 86 front \( \frac{9}{4} \) 87 front \( \frac{9}{4} \) 88 front \( \frac{9}{4} \) 89 front \( \frac{9}{4} \) 89 front \( \frac{9}{4} \) 80 front \( \frac{9}{4} \) 80 front \( \frac{9}{4} \)	examined         checked*         medial           85 front d <sup>1</sup> 340         9/170           5 rear d <sup>7</sup> 20         0/10           78 front P         312         12/156           17 rear P         68         3/34           85 front d <sup>7</sup> 510         66/255           5 rear d <sup>7</sup> 30         6/15           78 front P         468         57/234	examined         checked*         medial         lateral           85 front d <sup>1</sup> 340         9/170         8/170           5 rear d <sup>7</sup> 20         0/10         0/10           78 front t <sup>2</sup> 312         12/156         12/156           17 rear t <sup>2</sup> 68         3/34         2/34           85 front d <sup>8</sup> 510         66/255         75/255           5 rear d <sup>8</sup> 30         6/15         2/15           78 front t <sup>2</sup> 468         57/234         65/234	No. legs examined         Total surfaces checked*         No. of surfaces with lesions medial lateral         Surfaces with lesions           85 frent d <sup>1</sup> 340         9/170         8/170         17/340           5 rear d <sup>7</sup> 20         0/10         0/10         0/20           78 front P         312         12/156         12/156         24/312           17 rear P         68         3/34         2/34         5/68           85 front d <sup>4</sup> 510         66/255         75/255         141/510           5 rear d <sup>7</sup> 30         6/15         2/15         8/30           78 front P         468         57/234         65/234         122/468

<sup>\*</sup>Fetlock joint includes 6 articulating surfaces (2 lateral and 2 medial sesmoids, each considered as 1 surface). Pastern joint includes 4 surfaces (fig. 2).

fetlock joint (especially condyles of the canon bone) were more commonly affected than surfaces in the pasterns. Rear fetlocks showed slightly more wear than fetlocks of front legs. Right and left legs could be distinguished by the greater curvature of the lateral toenail and by a lateral protruberance on the lateral condyle of the canon bone in the fetlock joint. Both medial and lateral articular surfaces showed a similar frequency of damage (Table 2).

Sixteen pairs of front legs and 7 pairs of rear legs were available from 17 different animals. These included all four legs from 6 animals. No damage was seen in the joints of either leg of 5 pairs. In 15 of 18 pairs, some degree of damage was visible in both of the legs. In only 7 of these pairs of legs, was the same degree of wear visible in both legs.

In complete sets of 4 legs from 6 different moose (5  $\sigma$  and 1  $\mathfrak{k}$ ), wear was visible in joints of the hind legs but not in the front legs of 4 animals. Wear was visible in both hind and front legs of 2 animals but was rated more severe in the hind legs of one of these animals.

Skin wounds were visible externally on 5% (10/185 - 9 of and 1 º) of the lower legs examined. The most common wound was a split in the skin at the base of the dew-claws (5 legs). Three legs had purulent ulcerations on the volar surface between the dew-claws and 2 had lacerated hoof pads. In addition, the right canon bone of a 3½ year old male contained a pus-filled abscess (37x16x6 mm) mid-way along its length. The abscess, overlain by a loose piece of bone, did not extend into the marrow cavity. This condition may have been trauma induced. All of the above wounds occurred in front legs.



100

Traumatic injuries to the limbs were seen in 4 animals examined in this study. The articular cartilage was fractured in the front pastern of a male calf and in the rear fetlock of a yearling cow. The latter animal was killed by a vehicle but the damaged joint did not appear to have been caused by the accident. The right rear leg of a 7½ year old cow was dislocated between the tarsus and canon bone and the articular surface of the distal femur was deeply eroded. The animal was shot by conservation officers in February. Lastly, numerous loose pieces of soft tissue (3x7 mm) resembling joint-mice in the right rear fetlock of a yearling cow found dead may have been induced by trauma.

Included in this report are observations on three captive moose calves raised for the purpose of studying the pathogenic effects of the parasitic nematode, Elaphostrongylus cervi Cameron 1931. During the experiment two of these calves developed joint disease. Three wild calves  $(2 \sigma_{\sigma}, 1 \circ)$  weighing 14 to 15 kg were found abandoned between May 16 and May 25. Calf #1  $(\sigma)$  was especially weak in the legs and stood with the toes splayed apart and the pastern joints abnormally extended so that the dew-claws nearly touched the ground. This animal was unusual in having **a single** upper left front tooth (possibly the vestigial canine of some cervids) suspended from the gingival region by a slender piece of tissue which enveloped the base of the tooth. Calves #2  $(\sigma)$  and #3  $(\circ)$  were also weak when captured but otherwise appeared normal. On May 25, calf #1 was given 15 infective third-stage larvae of  $\underline{E}$ . Cervi per os. Calves #2 and #3 were given 30 and 50 larvae respectively.

Thirty-three days after infection calf #3 showed slight signs of incoordination. The right hind leg occasionally turned outward at the hock joint and the left rear fetlock joint remained flexed momentarily when weighted then extended rapidly causing the animal to falter slightly. Similar neurologic signs had been seen in a previously infected moose 30 days after larvae were administered and such signs were anticipated in the present experiment at this time (Lankester 1977). However, the following clinical picture including stiffness and swelling of the joints that developed in Calves #1 and #2 at 34 and 39 days post-infection respectively, appears to have been due primarily to an infectious arthritis which may have been unrelated to the experimental nematode infection.

On June 28, calf #1 refused to eat, had rapid and laboured breathing, congested lungs, watery eyes, rectal temperature of 39.4°C and was reluctant to stand. The animal had been somewhat constipated for the previous week and strained defication had prolapsed the rectum slightly. Stools were now mucousy to watery and liquid could be heard in the rumen. The animal was treated with tetracycline and cortisone for the following 3 days. After 2 weeks, the animal's condition had not improved. It ate irregularly and rose with difficulty only when prodded. Both elbow joints (humerus-radius-ulna) and the left rear fetlock were swollen. The animal walked stiff-legged and as if in pain. Its neck was drawn toward the shoulders, and the shoulder joints flexed in an apparent attempt to shift body weight directly over the front legs. The metacarpal joints were flexed allowing the dew-claws to touch the ground. Three weeks after the onset of signs, the animal was killed and examined.

At necropsy, large lymph nodes among thoracic and neck muscles were hyperemic. Lobes of the lung were less spongy than normal and the dorsal and medial surfaces of the apical lobes exhibited a blotchy, dark red pattern. There was excess, viscous, green-yellow synovial fluid in the capsules of all major limb joints. The synovial villi of the swollen left rear fetlock were particularly conspicuous. Neutrophils were extremely numerous in the synovial exudate.

Calf #2 exhibited a similiar clinical picture 5 days after calf #1. The stools of this animal had been soft to watery since its capture. One week after the onset of signs the left rear and left front fetlock joints were swollen. The animal was also moulting at this time. Twenty-two days after the onset of signs, synovial fluid was drawn from the left rear fetlock and oxytetrayline injected into the joint cavity. Routine aerobic and anaerobic bacterial cultures at the local hospital were negative. Bacterial culture and attempted <a href="Chlamydia">Chlamydia</a> isolation from chilled and frozen synovial fluid at the Ontario Veterinary College, Guelph, Ontario were also negative. A subsequent sample of fluid drawn one week later was negative for <a href="Chlamydia">Chlamydia</a> and did not inhibit a culture of Mycoplasma bovis.

Calf #2 recovered slowly and at 8 months of age walked normally and only occasionally favoured the left hind leg. The left rear fetlock joint was still noticeably enlarged and measured  $70 \times 80 \text{ mm}$  (width x antero-postero depth) as compared to the right rear fetlock which was  $65 \times 73 \text{ mm}$ . The condition observed in these two calves most closely resembled an infectious arthritis although attempts to isolate a biological agent were not successful.

An example of foot-rot, possibly caused by Sphaerophorus necrophorus,



was seen in the lower left hind leg of an older male moose shot in December near Atikokan, Ontario. The leg was grossly enlarged in the region of the fetlock and pastern joints. The skin was denuded of hair, was wet and yellowish-red in colour and irregular on the surface. Extensive subcutaneous fibrosis and large purulent abscesses (4 cm) prevented movement of the enclosed joints.

#### DISCUSSION

It is suspected that traumatic injuries are common in wild species yet there are few reports in the cervid literature devoted to this subject. Bone fractures or dislocations have been described as incidental observations in a few black-tailed deer (Cowan 1946) and moose (Peterson 1974, Dahlberg 1977) but only the long-term study of moose in Poland by Tomeck (1977) appears to approach a meaningful estimate of the significance of accidental injury as a cause of mortality in this species. In an 11 year period, 12 of 55 moose found dead exhibited mechanical injuries, the most common being fractured limbs. The five examples of trauma-induced limb or joint damage seen in the present study undoubtedly underestimate the frequency of mechanical injury in moose of northwestern Ontario since few entire animals were examined.

Arthritis caused by microbial agents occurs frequently in domestic animals and particularly in multiple joints of neonates during the course of a generalized infection (Medlowski and Segre 1960, Duthie and Lancaster 1964, Storz et al. 1966). Reports of infectious arthritis in wild cervids however, are rare. Sikes et al. (1968) isolated Staphylococcus sp. from the enlarged hock and knee joints of a 5½ year old white-tailed deer shot by a hunter in South Carolina. The causative agent of polyarthritis in



104

3 penned moose (at least one calf) described by Kozhuklov (1959, 1965) was not identified. Similarly in the present study, arthritis observed in 2 young calves was presumed to have been due to an infectious agent but none was isolated.

Necrobacillosis caused by the bacterium <u>Sphaerophorus necrophorus</u> affects the gastro-intestinal tract, feet, and occasionally the lower joints of a variety of ungulates (Rosen 1970). Epizootics of the disease have occurred in elk, black-tailed deer and mule deer where these species congregate in winter feed lots or watering sites. Foot-rot, presumably caused by <u>S. necrophorus</u> does not appear to be common in moose. A single report by Murie (1934) and the one here suggest that the more solitary habits of moose may preclude frequent infection. It is generally believed that the bacterium cannot invade unbroken skin. Skin wounds seen in 5% of moose feet examined in this study would provide ideal portals of entry.

Degenerative arthropathy is the type of joint disease most frequently seen in wild cervids. The process may be a natural consequence of aging (Wobeser and Runge 1975). In the conspicuous advanced stages however, it is often impossible to exclude trauma, infectious agents or abnormal wear in the joint as the initial cause. The vertebral column and the larger joints of the limbs, particularly in the hind legs, most frequently exhibit arthropathy (Jubb and Kennedy 1970). These joints are more commonly affected in males than in females. Thomson (1969) and Jubb and Kennedy (1970) suggested that vertebral ankylosis observed in older domestic bulls may be related to the thrust of males during coitus. In white-tailed deer, vertebral osteophytes occurred only in males older than 6 years (Wobeser and Runge 1975) and arthropathy of the stifle joints was almost four times as common in males older than 4 years as in

females of similar age. In moose on Isle Royale, Peterson (1974) found hip arthropathy three times as common in males as in females older than 6 to 7 years.

In the present study, only early degenerative changes were seen in metacarpal and metatarsal joints. Because of their tighter construction, these joints are perhaps less likely to show degenerative changes (Bennett 1931) than the larger diarrthroses of the body. The prevalence of such lesions was similar in both males and females, but rear fetlock joints exhibited wear more frequently than fetlocks in the front legs.

The ecological significance of joint disease in wild ungulates is poorly understood. Under natural conditions however, severely affected animals would undoubtedly be more susceptible to premature death. Two excellent studies substantiate this opinion. A high proportion of moose killed by wolves on Isle Royale (66/205) showed conspicuous bone pathology (Peterson 1974) and degenerative bone lesions were observed in 26 of 128 white-tails that died during an unusually severe winter in Saskatchewan (Wobeser and Runge 1975). In both of these studies bone pathology was confined principally to older animals. These members of a population with joint disease are clearly handicapped in escaping predators and in travelling through deep snow.

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106

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