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INCREASED OSTEOARTHRITIS IN MOOSE FROM ISLE ROYALE

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ABSTRACT: Over the past 30 yr, moose (*Alces alces*) in Isle Royale National Park (Michigan, USA) exhibited a several-fold increase in the prevalence of osteoarthritis, or degenerative joint disease (DJD). Available evidence points to an environmental explanation for this change. Greater physical activity among afflicted moose is not a likely contributing factor, nor is genetic change in the population. The possible introduction of an unspecified disease agent cannot be dismissed at this time. Moose exhibiting the highest prevalence of DJD were those born during a period of severe undernutrition, and it is hypothesized that nutritional stress early in life was responsible for increased joint disease during senescence. Such an etiology for osteoarthritis has not been suggested previously for any species.

Key words: Moose, *Alces alces*, degenerative joint disease, prevalence data, undernutrition, wolf predation, osteoarthritis, field study.

INTRODUCTION

Osteoarthritis, or degenerative joint disease (DJD), is a common affliction in many species of large mammals (Sokoloff, 1969). Degenerative joint disease may be a consequence of joint trauma or abnormal cartilage, yet its appearance in joints with no history of trauma remains largely a mystery. The physiological and biomechanical explanations for this important disease have focused primarily on short-term etiology (Howell, 1985; Sokoloff and Hough, 1985), especially in old individuals where the disease is common.

In moose (*Alces alces*), DJD afflicts males more than females and becomes increasingly significant after 7 yr of age, coincident with increased vulnerability of moose to wolf predation (Peterson, 1977). Degenerative joint disease in Isle Royale moose primarily affects the weight-bearing coxofemoral joints (femur-pelvic acetabulum) plus the posterior vertebrae. The present study was a longitudinal study of DJD incidence in the Isle Royale moose population, based on data from animals dying as a result of wolf predation and other natural causes.

MATERIALS AND METHODS

Skeletal material from moose at Isle Royale National Park, Michigan (48°N, 89°W), was examined and collected from 1958 to 1987 by the author and previous workers during a long-term study of wolf–moose dynamics (Mech, 1966; Peterson, 1977). Moose carcasses, mostly wolf-kills, were located in winter by aerial search throughout the 544-km² island, and in summer during extensive coverage of the island on foot. Recovery rate was estimated at approximately 20% (Peterson, unpubl. data). In many cases only partial skeletons were available for inspection. When estimating the prevalence of DJD, only moose remains that included a pelvis and/or first sacral vertebra were tabulated, because these were the most common arthritic sites in this population (Peterson, 1977; Peterson et al., 1982). These bones were recovered from 87% of 416 adult moose with detailed data on skeletal remains. Moose younger than 7 yr old rarely exhibited DJD and were excluded from data analysis.

Degenerative joint disease was indicated by bony exostoses surrounding joints, sclerosed articulating surfaces following loss of cartilage, and/or pitted and rough joint surfaces. Vertebral joints were commonly enlarged by bony outgrowth, and coxofemoral joints exhibited initial closure of the acetabular fossa, followed by displaced joint articulation and, finally, luxation.

Sex of moose was determined from skeletal remains by the presence or absence of antler pedicels on the skull. Age was estimated from counts of cementum annuli in molar teeth (Wolfe, 1969). If both year of death and estimated age were available for a moose, it was placed into an annual cohort corresponding to year of birth. The statistical significance of differences in proportions was determined by an arcsin transformation method, useful for examining differences between two percentages (Sokal and Rohlf, 1969).

Pertinent ecological parameters were measured during the study. Moose population den-

TABLE 1. Time of death in relation to prevalence of degenerative joint disease in moose skeletal remains from Isle Royale National Park.

Time of death	Moose 7- to 11-yr-old*	Moose ≥12-yr-old*
1955–1959	7% (42)	53% (17)
1960–1964	14% (85)	13% (30)
1965–1969	23% (52)	22% (46)
1970–1974	21% (90)	31% (35)
1975–1979	39% (111)	70% (79)
1980–1984	53% (30)	62% (71)
1985–1987	33% (9)	80% (55)
Total	26% (419)	53% (333)

* Sample size in parentheses.

TABLE 2. Time of birth in relation to prevalence of degenerative joint disease in moose from Isle Royale National Park.

Time of birth	Moose 7- to 11-yr-old*	Moose ≥12-yr-old*
1946–1949	0% (5)	6% (17)
1950–1954	9% (58)	9% (34)
1955–1959	27% (22)	42% (24)
1960–1964	27% (64)	71% (55)
1965–1969	33% (88)	63% (82)
1970–1974	44% (34)	74% (23)

* Sample size in parentheses.

sity was periodically determined by aerial census (Mech, 1966; Peterson, 1977). Winter severity was indicated by annual snowfall records from Thunder Bay, Ontario, 25 km distant, the closest year-round weather station (Atmospheric Environment Service, Thunder Bay Airport, Thunder Bay, Ontario, Canada P7E 3N9). Nutritional status of the moose population was inferred from fat content of bone marrow (Verme and Holland, 1973) collected from femurs and other leg bones of moose found dead each winter. Wolf predation, responsible for most moose deaths, selectively removed vulnerable prey from the population (Mech, 1966; Peterson, 1977). Thus, the bone marrow fat levels reported in this study are not considered representative of the population, but rather the most vulnerable segment of the moose population.

RESULTS

Prevalence of DJD in moose from Isle Royale was determined from skeletal remains of 752 moose that were at least 7 yr old at the time of death. The overall occurrence of DJD among moose aged 7 to 11 yr (26%) was half that of older moose (53%) (Table 1), so these two groups were analyzed separately as "middle-aged" and "old" moose. Since DJD probably predisposes moose to wolf predation, the prevalence of DJD among living moose is likely much lower than indicated by this study of skeletal remains.

Over a period of more than 30 yr, a steady increase in the prevalence of DJD was evidence (Table 1). Degenerative joint disease increased in parallel fashion in both males and females, although the proportional increase in females exceeded that of males. Among moose (at least 7 yr old) dying in 1974 or before, 15% of the females and 30% of the males were arthritic at death. After 1974, the arthritic proportion was 54% among females and 73% among males. Both sexes were pooled in subsequent analyses, as there were no consistent shifts in sex ratio among skeletal remains.

Excluding time periods with inadequate sample size ($n < 25$), the prevalence of DJD increased from 7 to 53% for middle-aged moose and from 13 to 80% for old moose (Table 1). The change in DJD prevalence in three successive decades (1960's, 1970's and 1980's) was significant ($P < 0.05$) for both age groups in all cases.

When DJD prevalence was analyzed by year of birth, moose born during the early 1970's exhibited the highest occurrence of joint lesions (Table 2). Generally, DJD increased in both age groups from the late 1940's until the early 1970's. When data were grouped by 10-yr periods (1945 to 1954, 1955 to 1964, 1965 to 1974), there were significant differences ($P < 0.05$) in DJD prevalence only between cohorts from 1945 to 1954 and those from 1955 to 1964 for both age groups. Only four middle-aged moose from post-1974 cohorts had been recovered by 1987, so DJD prevalence in recent cohorts is not yet known.

Moose population density and annual snowfall both increased during the 1960's and peaked around 1970, causing considerable nutritional stress in moose (Peter-

TABLE 3. Total annual snowfall in Thunder Bay, Ontario, 1960–1987.

Year*	Snowfall (cm)	Year*	Snowfall (cm)	Year*	Snowfall (cm)
1960	140	1970	296	1980	197
1961	256	1971	273	1981	176
1962	190	1972	252	1982	201
1963	164	1973	127	1983	179
1964	140	1974	192	1984	146
1965	230	1975	154	1985	155
1966	288	1976	243	1986	266
1967	187	1977	117	1987	164
1968	144	1978	223		
1969	249	1979	288	Mean, 1960–1987	201

* 1960 = winter of 1959–1960, etc.

son, 1977). Snowfall records reveal a pattern of variable but increasing winter severity during the 1960's, culminating in a series of four consecutive deep-snow winters beginning in 1969 (Table 3). Simultaneously, the increasing moose population reached a peak density of almost three moose/km², up from one to two moose/km² in the late 1950's (Mech, 1966; Peterson, 1977). For young moose, especially, reduced nutritional status during the early 1970's was clearly indicated by fat-depleted bone marrow, low calf recruitment and delayed epiphyseal closure in leg bones (Peterson, 1977).

During the late 1970's and early 1980's moose density had again been reduced to one to two animals/km² (Peterson et al., 1984), and moose nutritional status improved during the 1970's. Calf marrow fat levels in midwinter increased significantly ($P < 0.05$, t-test) from a mean of 14 ± 8% fat (95% C.I.) in 1968 to 1972 to 31 ± 7% fat in 1973 to 1977, then increased ($P < 0.05$) further to 43 ± 10 in 1978 to 1985. Marrow fat levels in adult moose increased significantly from 50 ± 11% in 1968 to 1972 to 65 ± 6% in 1973 to 1977 and 64 ± 5% in 1970 to 1985.

DISCUSSION

Primary DJD, defined as having no known precursor condition, results from cartilage deterioration associated with aging (Sokoloff, 1969). The etiology of primary DJD remains poorly understood, although secondary DJD may develop as a result of trauma or infectious disease (Howell, 1985). Any retrospective discussion of changing prevalence of DJD in a population over time will be necessarily speculative, although consideration of available data on this population narrows the range of hypotheses.

Possible explanations for the pronounced increase in recorded incidence of DJD include artifactual, genetic, mechanical and environmental causes. Available evidence eliminates the first three possibilities, and supports the contention that an environmental cause, specifically early malnutrition, stands as the most likely explanation for increased DJD in Isle Royale moose. The continuing natural experiment at Isle Royale will provide a test of this hypothesis.

In the early years of the study DJD was unknown in moose, and investigators were not specifically examining moose skeletons for indications of arthritis. Thus, the increase in the recorded incidence of joint disease might simply reflect more careful skeletal examinations resulting from an increased awareness of its occurrence. This could have contributed to the low recorded incidence of DJD in moose born before the late 1950's. However, after 1970 all skeletal remains (64% of the total sample) were examined systematically for arthritis by the author, and it was then that a sub-

stantial increase in DJD occurred (Table 1). Arthritis increased in both middle-aged and very old moose (Table 1), and in both sexes, so changes in the age or sex composition of the population do not appear to be contributing factors. Thus, the recorded increase in DJD after the late 1950's cannot be dismissed as an artifact of change in study methods or population composition.

Data in this study were derived from moose dying of natural causes, primarily wolf predation, during a period when there were major fluctuations in density of both wolf and moose populations. Could a changing pattern of prey selection by wolves explain the increased frequency of DJD over time? Changes in predation pressure may be revealed by wolf kill rates and wolf : moose ratios during the study. Wolf kill rate reached a peak in 1975, then declined to a low, stable level in the 1980's (Peterson and Page, 1988). Wolf : moose ratios declined during the 1960's, increased in the 1970's, then declined again in the 1980's (Peterson et al., 1984). There are no similarities between these trends and DJD prevalence. Furthermore, in both the late 1960's and the late 1980's wolf : moose ratios were similar (approximately one wolf : 50 moose) and wolves preyed most heavily on old adult moose (Peterson et al., 1984; Peterson, unpubl. data), yet the prevalence of DJD among these prey was remarkably different. Thus, fluctuations in predator-prey dynamics do not adequately explain the increased DJD.

There is evidence suggesting differential genetic susceptibility to joint disease in humans (Stanescu et al., 1985) and dogs (Van Sickle, 1984). Different moose populations also exhibit variations in the incidence of DJD and its location in the skeleton (Peterson et al., 1982). However, the increase in DJD among Isle Royale moose occurred in little more than two generations, so genetic change is an unlikely explanation.

Degenerative joint disease may follow progressive deterioration of cartilage resulting from unusual wear or trauma in a normal joint. Likewise, normal activity in an abnormal joint capsule may lead to DJD (Howell, 1985). From 1,000-yr-old osteological remains of a population of North American humans, Goodman et al. (1984) and Goodman and Armelagos (1985) documented a 65% increase in osteoarthritis which was associated with the development of agriculture. They suggested greater physical activity as the likely cause of the increased incidence of arthritis. For Isle Royale moose, this possibility can be eliminated. Actually, moose activity levels probably declined in the late 1970's and early 1980's, when arthritis increased but forage had recovered, because movements of moose are largely associated with finding and ingesting food (Risenhoover, 1986; Renecker, 1987).

We are left with an environmental cause as the most plausible explanation for increased joint disease among Isle Royale moose. It is possible that an unknown disease agent could have become established in the moose population during the study. It cannot be readily explained how such an infectious agent could produce the strikingly consistent pattern of susceptibility in relation to age and sex. Furthermore, joint disease increased when the population was at a historic low level in the late 1970's, contrary to expectations if a density-dependent disease agent was involved.

Likewise, consideration of fluctuations in moose density and marrow fat levels leads to a rejection of undernutrition as a proximal cause of increased DJD, as moose declined and their nutritional status improved while DJD increased. However, cohorts with the highest prevalence of DJD were severely undernourished early in life. In the early 1970's nutritional stress in winter, when females were gravid, led to a series of cohorts of young moose that were small at birth and exhibited retarded growth as young adults (Peterson, 1977; Peterson et al., 1982). Possibly undernutrition early in life led to developmental

abnormalities in cartilage that increased the prevalence of DJD late in life. Accordingly, the gradual increase in DJD incidence among moose born in the 1960's can be explained by a density-dependent decline in nutritional status that accompanied population growth.

This "early nutrition hypothesis" implies the presence of subtle developmental abnormalities in cartilage that ultimately resulted in a higher frequency of degenerative lesions. In young dogs, cartilage anomalies caused by osteochondrosis were associated with arthritis in the same joints in older dogs (Van Sickle, 1984). In moose, normal mechanical use of joints with abnormal cartilage could explain the fact that arthritis increased in both sexes and in all susceptible age categories.

To my knowledge, such an etiology for DJD has not been suggested previously. However, longitudinal studies of experimental populations must span several decades before even rudimentary data are available on the incidence of DJD in large mammals. Natural experiments probably will provide the necessary temporal scale, yet these are often hampered by confounding factors. The improved nutritional status of Isle Royale moose in the 1980's will ultimately provide a potential disproof of the early nutrition hypothesis; it predicts that DJD incidence will decline among moose born after 1980, that the decline will first become evident among middle-aged moose, and then finally in older moose.

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