GENETIC DIVERSITY OF MOOSE FROM THE KENAI PENINSULA, ALASKA

Kris J. Hundertmark[1], Paul E. Johns[2], and Michael H. Smith[2,3]

[1]Alaska Department of Fish and Game, Kenai Moose Research Center, 34828 Kalifornsky Beach Road, Soldotna, AK 99669; [2]Savannah River Ecology Laboratory, Aiken, SC 29802; [3]Institute of Ecology, Departments of Genetics and Zoology, Faculty of Ecology, and School of Forest Resources, University of Georgia, Athens, GA 30602

ABSTRACT: Six of 20 loci expressed in liver and muscle tissue from Kenai Peninsula moose (*Alces alces gigas*) were polymorphic. Average heterozygosity was 7.7%, which represents an unprecedented level of genetic diversity for moose. This level of diversity was not expected because empirical evidence from other moose populations, as well as theoretical considerations, indicated that moose exhibited low levels of heterozygosity. We propose that moose populations with low diversity reside in areas that were glaciated during the last Ice Age and that the recolonization process reduced heterozygosity, while high-diversity populations reside in areas in the proximity of glacial refugia.

ALCES VOL. 28 (1992) pp. 15-20

Analysis of genetic heterogeneity within and among wildlife populations can yield a better understanding of population processes (Smith *et al.* 1984) which can be relevant in a management context (Smith *et al.* 1976). For instance, spatial differences in allele frequencies can be used to detect breeding structure and delineate functional populations (Manlove *et al.* 1976). These delineations can then be used to define management area boundaries. Furthermore, incidence of phenotypic characteristics related to increased fitness have been associated with the degree of heterozygosity (*H*) in individuals (Johns *et al.* 1977, Smith *et al.* 1982, Cothran *et al.* 1983, 1987, Chesser and Smith 1987, Scribner *et al.* 1989). However, population heterozygosity can be diminished to varying degrees by different hunting regimes (Ryman *et al.* 1981); thus, maintenance of genetic variability of game species is an important management consideration.

Electrophoretic studies of proteins isolated from moose indicated a paucity of detectable genetic variability. Nadler *et al.* (1967) and Wilhelmson *et al.* (1978) found no polymorphisms in serum proteins from populations of moose in Scandinavia (*A. a. alces*), Canada (*A. a. andersoni*), and Alaska (*A. a. gigas*). Ryman *et al.* (1977) examined 23 loci and detected only one polymorphism in Swedish moose. Subsequent studies have revealed multilocus variability in *A. a. alces* (Ryman *et al.* 1980, Baccus *et al.* 1983) and *A. a. americana* (Reuterwall and Ryman 1979), but not to the extent reported for other cervids (Breshears *et al.* 1988, Smith *et al.* 1990). Our objective was to determine the level of genetic variability in a population of moose from the Kenai Peninsula, Alaska.

METHODS

Samples of liver and skeletal muscle were obtained from moose killed by highway vehicles. Thirty-one samples were collected during November 1989-March 1990 and represented 29 adults and 2 fetuses. Seven samples were obtained from fetuses collected during March-June 1988 and kept frozen until analyzed. Fetuses were sampled only when samples from the mother were not available. No samples were collected from individuals known to be a sibling, parent, or offspring of another collected individual.

The following thirteen enzyme systems representing 20 presumptive loci were examined: malate dehydrogenase (MDH-1, MDH-2), phosphoglucomutase (PGM-1, PGM-2), mannose phosphate isomerase (MPI), peptidase (PEP-1, PEP-2, PEP-3,

leucylglycylglycine substrate), malic enzyme (MOD-1, MOD-2), glucosephosphate isomerase (GPI-2), esterase (EST, ß-napthylproprionate substrate), aconitase (ACON), sorbitol dehydrogenase (SORDH), lactate dehydrogenase (LDH-1, LDH-2), amino aspartatetransaminase (AAT-1, AAT-2), α-glycerophosphate dehydrogenase (α-GPD), and adenosine deaminase (ADA). Preparation of extracts, electrophoretic procedures and staining followed Selander *et al.* (1971) and Manlove *et al.* (1975). Allele frequencies, estimates of *H*, alleles per locus (*A*), proportion of polymorphic loci (*P*), and Chi-square testing of conformance to Hardy-Weinberg expectations were performed using BIOSYS (Swofford and Selander 1981). Loci were considered polymorphic if the frequency of occurrence of the most common allele did not exceed 0.99.

RESULTS

Six of 20 (*P*=30%) loci (MDH-1, PGM-1, PGM-2, MPI, PEP-2, and MOD-2) were polymorphic (Table 1). These loci, with the exception of MOD-2, have been reported to be polymorphic in other moose populations (Ryman *et al.* 1980, Baccus *et al.* 1983); however, no single population heretofore exhibited more than 3 polymorphic loci. Ryman *et al.* (1980), in a study of 18 Scandinavian moose populations, reported estimates of *P* ranging from 4.3-13%. Baccus *et al.* (1983) reported *P*=15.8% for Scandinavian moose. Smith *et al.* (1990) estimated average *P* for cervids as 17.4%.

One locus (PEP-2) in the present study exhibited 3 alleles, whereas the remaining polymorphic loci exhibited 2 alleles each (Table 1), yielding an estimate of *A* of 1.35 (SE 0.13), which is within the range exhibited by other cervids (Baccus *et al.* 1983, Smith *et al.* 1990). Direct-count estimates of heterozygosity for polymorphic loci (*h*) ranged from 2.6-47.2% (Table 1). Mean heterozygosity (*H*), including 14 monomorphic loci, was 7.7% (SE 3.4%), which was considerably greater than the inter-population mean of 2% (range 0.6-4.7%) reported by Ryman *et al.* (1980) and the value reported by Baccus *et al.* (1983) (*H* = 1.7%) for Scandinavian moose. Smith *et al.* (1990) estimated *H* = 3.5% for cervids in general. Frequency of occurrence of heterozygotes did not deviate significantly from Hardy-Weinberg expectations (X^2=0.16, d.f.=1, *p*=0.69).

DISCUSSION

Our data represent an unprecedented level of genetic diversity for moose and the only reported variability for *A. a. gigas*. Previous reports characterizing moose as a species exhibiting low to moderate levels of variability

Table 1. Allele (A, B, and C) frequencies and a measure of heterozygosity (*h*) for 6 polymorphic loci from a Kenai Peninsula, Alaska moose population.

	Locus[1]					
Allele	MDH-1	PGM-1	MPI	PEP-2	PGM-2	MOD-2
N	38	38	38	38	32	38
A	0.000	0.000	0.368	0.250	0.031	0.263
B	0.987	0.895	0.632	0.737	0.969	0.737
C	0.013	0.105	0.000	0.013	0.000	0.000
h	0.026	0.211	0.368	0.395	0.063	0.472

[1]Abbreviations defined in text.

were based primarily on data from Scandinavian populations or from North American populations from which only a few loci were examined. It is now apparent that indices of variability in moose can vary dramatically on a large geographic scale. We believe that moose populations potentially could express even greater variability as loci not examined in this study were polymorphic in other populations (Gyllensten *et al.* 1980, Ryman *et al.* 1977).

The dramatic differences in genetic diversity between the Kenai population and others may stem from the origin of populations following the retreat of the Wisconsin ice sheet. *Alces alces gigas* originated in refugia in Beringia (interior Alaska and what is now the Bering Sea). The origin of all other North American subspecies is debatable, but whether they were derived from stocks located south of the Wisconson glacial maximum (Klein 1965, Peterson 1955:14) or from Beringia (Cronin 1992, Geist 1985) it remains that their present range was once entirely glaciated. Recent populations residing in or near refugia likely would retain more genetic diversity than populations established at great distances from refugia through a series of founding events. Prior reports of low genetic diversity in moose dealt with populations residing in previously glaciated areas. Thus, these populations possibly experienced a loss of diversity due to genetic drift during the process of recolonizing new habitat following glacial retreat (Sage and Wolff 1986).

Moose inhabiting the Kenai Peninsula are isolated both spatially and temporally from neighboring populations. A 16-km wide mountainous isthmus connects the peninsula with the remainder of Alaska, and most moose habitat on the peninsula is not contiguous with the isthmus. Thus, any interchange of individuals likely is minimal. Had the peninsula been colonized by moose in the late 19th century, as was the commonly held belief of early explorers of the area (Lutz 1960), we would predict low genetic diversity in the population due to founder effect and inbreeding. Our data support the contention of Lutz (1960) that moose populations increased as a result of 3 documented forest fires between 1870 and 1910, prior to which they had existed at low densities in late-successional forest. This claim is supported by archaeological evidence that indicates that moose were present on the peninsula at least 2000 years ago (deLaguna 1934:13). Theoretically, such a period of relative isolation from other moose populations characterized by fluctuations in population size could lead to reduced genetic variability through drift and bottleneck effects. However, it is possible that the effective population size, even at low densities, was adequate to maintain diversity. Furthermore, Nei *et al.* (1975) demonstrated that reductions in heterozygosity after a bottleneck could be small if the population increased rapidly thereafter. Such rapid increases in moose populations are typical in southcentral Alaska because these populations are irruptive in nature, depending upon wildfire for creation of suitable habitat.

These data offer some insight toward prevailing theories concerning patterns of genetic variability among taxa. For instance, Harrington (1985) proposed that, among cervids, *r*-strategists were less variable genetically than *K*-strategists. However, our data lend support to the conclusion reached by Hartl and Reimoser (1988) that *r*-strategists can display substantial amounts of variation. Selander and Kaufman (1973) hypothesized that large, highly mobile mammals would exhibit low heterozygosity while small, sedentary types would exhibit high degrees of variation. Ryman *et al.* (1980) provided data for moose which seemed to discount this theory, at least at the species level, and our data further demonstrate that large, highly-mobile species can exhibit relatively great amounts of heterogeneity. Smith *et al.* (1990), in reviewing heterogeneity of cervids, ob-

served that boreal species exhibited the lowest average *H*. This observation may be true in a general sense, but it should not be interpreted as meaning that boreal species cannot exhibit high heterogeneity at the population level.

Recent studies in wildlife population genetics have examined relationships between expressions of fitness and genetic structure. Pemberton *et al.* (1988, 1991) demonstrated a relationship between juvenile survival, female fecundity and genotypes at specific loci in red deer (*Cervus elaphus*). Relationships between heterozygosity and male body size and antler characteristics (Scribner and Smith 1990), body condition of over-wintering females (Cothran *et al.* 1983), and conception timing (Chesser and Smith 1987) among other characteristics have been reported for white-tailed deer (*Odocoileus virginianus*). Hartl *et al.* (1990) reported an apparent association between genotypes at specific loci and the number of antler points in red deer, and Harmel (1983) provided evidence that antler size in white-tailed deer is genetically controlled. *Alces alces gigas* is characterized by the largest body and antler size of all moose subspecies (*see* Franzmann 1978, Geist 1987), which may be a result of the high genetic diversity we observed. These characteristics contribute to the fitness of indivduals, and can affect an individual's liklihood of being harvested by a hunter; therefore, they should be considered in a management context.

ACKNOWLEDGEMENTS

C. C. Schwartz provided insight and encouragement and, along with M. A. Cronin, reviewed the manuscript. J. M. Novak assisted with the statistical analysis of the data. This study was funded in part by Federal Aid in Wildlife Restoration and by a Department of Energy contract DE-AC09-765ROO-819 with the University of Georgia.

REFERENCES

BACCUS, R., N. RYMAN, M. H. SMITH, C. REUTERWALL, and D. CAMERON. 1983. Genetic variability and differentiation of large grazing mammals. J. Mammal. 64:109-120.

BRESHEARS, D. D., M. H. SMITH, E. G. COTHRAN, and P. E. JOHNS. 1988. Genetic variability in white-tailed deer. Heredity 60:139-146.

CHESSER, R. K., and M. H. SMITH. 1987. Relationship of genetic variation to growth and reproduction in the white-tailed deer. Pages 168-177 *in* C. M. Wemmer, ed. Biology and management of the Cervidae. Smithsonian Inst. Press, Washington, D. C.

COTHRAN, E. G., R. K. CHESSER, M. H. SMITH, and P. E. JOHNS. 1983. Influences of genetic variability and maternal factors on fetal growth in white-tailed deer. Evolution 37:282-291.

———, ———, ———, ———. 1987. Fat levels in female white-tailed deer during the breeding season and pregnancy. J. Mammal. 68:111-118.

CRONIN, M. A. 1992. Intraspecific variation in mitochondrial DNA of North American cervids. J. Mammal. 73:70-82.

deLAGUNA, F. 1934. The archaeology of Cook Inlet. Univ. Pennsylvania Press. Philadelphia. 263pp.

FRANZMANN, A. W. 1978. Moose. Pages 67-81 *in* J. L. Schmidt and D. L. Gilbert, eds. Big Game of North America. Stackpole Books, Harrisburg, PA.

GEIST, V. 1985. On Pleistocene bighorn sheep: some problems of adaptation and relevance to today's American megafauna. Wildl. Soc. Bull. 13:351-359.

———. 1987. On the evolution and adaptations of *Alces*. Swedish Wildl. Res., Suppl. 1:11-23.

GYLLENSTEN, U., C. REUTERWALL, N. RYMAN, and G. STAHL. 1980. Geographical variation of transferrin allele

frequencies in three deer species from Scandinavia. Hereditas 92:237-241.

HARMEL, D. E. 1983. Effects of genetics on antler quality and body size in white-tailed deer. Pages 339-348. *in* R. D. Brown, ed. Antler development in the Cervidae. Caeser Kleberg Wildl. Res. Inst., Kingsville, TX.

HARRINGTON, R. 1985. Evolution and distribution of the Cervidae. Biology of deer production, The Royal Society of New Zealand, Bulletin 22:3-11.

HARTL, G. B., and F. REIMOSER. 1988. Biochemical variation in roe deer (*Capreolus capreolus* L.): are *r*-strategists among deer genetically less variable than *K*-strategists? Heredity 60:221-227.

_____, G. LANG, F. KLEIN, and R. WILLING. 1991. Relationships between allozymes, heterozygosity and morphological characters in red deer (*Cervus elaphus*), and the influence of selective hunting on allele frequency distributions. Heredity 66:343-350.

JOHNS, P. E., R. BACCUS, M. N. MANLOVE, J. E. PINDER III, and M. E. SMITH. 1977. Reproductive patterns, productivity and genetic variability in adjacent white-tailed deer populations. Proc. Ann. Conf. S.E. Assoc. Fish Wildl. Agencies 31:167-172.

KLEIN, D. R. 1965. Postglacial distribution patterns of mammals in the southern coastal regions of Alaska. Arctic 18:7-20.

LUTZ, H. J. 1960. History of the early occurrence of moose on the Kenai Peninsula and in other sections of Alaska. Misc. Publ. No. 1. U. S. D. A. Forest Service, Alaska Forest Res. Center, Juneau.

MANLOVE, M. N., J. C. AVISE, H. O. HILLESTAD, P. R. RAMSEY, M. H. SMITH, and D. O. STRANEY. 1975. Starch gel electrophoresis for the study of population genetics in white-tailed deer. Proc. S. E. Assoc. Game Fish Comm. 29:392-403.

_____, M. H. SMITH, H. O. HILLESTAD, S. E. FULLER, P. E. JOHNS, and D. O. STRANEY. 1976. Genetic subdivision in a herd of white-tailed deer as demonstrated by spatial shifts in gene frequencies. Proc. S. E. Assoc. Game Fish Comm. 30:487-492.

NADLER, C. F., C. E. HUGHES, K. E. HARRIS, and N. W. NADLER. 1967. Electrophoresis of the serum proteins and transferrins of *Alces alces* (elk), *Rangifer tarandus* (reindeer), and *Ovis dalli* (Dall sheep) from North America. Comp. Biochem. Physiol. 23:149-157.

NEI, M., T. MARUVAMA, and R. CHAKRABORTY. 1975. The bottleneck effect and genetic variability in populations. Evolution 29:1-10.

PEMBERTON, J. M., S. D. ALBON, F. E. GUINESS, T. H. CLUTTON-BROCK, and R. J. BERRY. 1988. Genetic variation and juvenile survival in red deer. Evolution 42:921-934.

_____, _____, _____, _____. 1991. Countervailing selection in different fitness components in female red deer. Evolution 45:93-103.

PETERSON, R. L. 1955. North American moose. University of Toronto Press, Toronto.

REUTERWALL, C., and N. RYMAN. 1979. Genetic variability and differentiation in moose in Fennoscandia (*Alces alces alces* L.) and Canada (*A. a. americana* Clinton) [abstract]. Hereditas 91:312-313.

RYMAN, N., G. BECKMAN, G. BRUUN-PETERSEN, and C. REUTERWALL. 1977. Variability of red cell enzymes and genetic implications of management policies in Scandinavian moose (*Alces alces*). Hereditas 85:157-162.

_____, C. REUTERWALL, K. NYGREN, and T. NYGREN. 1980. Genetic variation and differentiation in Scandinavian moose (*Alces alces*): are large mammals

monomorphic? Evolution 34:1037-1049.

_____, R. BACCUS, C. REUTERWALL, and M. H. SMITH. 1981. Effective population size, generation interval, and potential loss of genetic variability in game species under different hunting regimes. Oikos 36:257-266.

SAGE, R.D., and J. O. WOLFF. 1986. Pletistocene glaciations, fluctuating ranges, and low genetic variability in a large mammal (*Ovis dalli*). Evolution 40:1092-1095.

SELANDER, R. K., and D. W. KAUFMAN. 1973. Genetic variability and strategies of adaptation in animals. Proc. Nat. Acad. Sci. 70:1875-1877.

_____, M. H. SMITH, S. Y. YANG, W. E. JOHNSON, and J. B. GENTRY. 1971. Biochemical genetics in the genus *Peromyscus*. I. Variation in the old field mouse (*Peromyscus polionotus*). Studies in Genetics, VI, The Univ. of Texas Publ. 7103:49-90.

SCRIBNER, K. T., and M. H. SMITH. 1990. Genetic variability and antler development. Pages 460-473 *in* G. A. Bubenik and A. B. Bubenik, eds. Horns, pronghorns and antlers: evolution, morphology, physiology and social significance. Springer-Verlag, New York, N.Y.

SMITH, M. H., H. O. HILLESTAD, M. N. MANLOVE, and R. L. MARCHINGTON. 1976. Use of population genetics data for the management of fish and wildlife populations. Trans. N. Amer. Wildl. Nat. Res. Conf. 41:119-133.

_____, R. K. CHESSER, E. G. COTHRAN, and P. E. JOHNS. 1982. Genetic variability and antler growth in a natural population of white-tailed deer. Pages 365-387 *in* R. D. Brown, ed. Antler development in the Cervidae. Caeser Kleberg Wildl. Res. Inst., Kingsville, TX.

_____, R. BACCUS, H. O. HILLESTAD, and M. N. MANLOVE. 1984. Population genetics. Pages 119-128 *in* L. K. Halls, ed. White-tailed deer: ecology and management. Stackpole Books, Harrisburg, PA.

_____, K. T. SCRIBNER, L. H. CARPENTER, and R. A. GARROTT. 1990. Genetic characteristics of Colorado mule deer (*Odocoileus hemionus*) and comparisons with other cervids. Southwestern Nat. 35:1-8.

SWOFFORD, D. L., and R. B. SELANDER. 1981. BIOSYS-1: a computer program for the analysis of allelic variations in genetics. University of Illinois Press, Urbana-Champaign, 65 pp.

WILHELMSON, M., R. K. JUNEJA, and S. BENGTSSON. 1978. Lack of polymorphism in certain blood proteins and enzymes of European and Canadian moose (*Alces alces*). Naturaliste can. 105:445-449.