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GENETIC VARIATION AND NESTING BALD EAGLES[1]

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As habitats become fragmented, once-contiguous populations may be spatially isolated and reduced in size. In addition, new populations may arise through dispersal and colonization. When this occurs, the founding population is often small and there is a reduced chance that the founding individuals represent the full genetic compliment found in the parent population. Resulting populations may be characterized by reduced heterozygosity and allelic diversity. We assessed genetic variation in two nesting populations of Bald Eagles (*Haliaeetus leucocephalus*): a small isolated one in Colorado and a large contiguous one in Ontario, Canada. We predicted that the Colorado population would have reduced heterozygosity and allelic diversity when compared with the Ontario population.

STUDY AREA AND METHODS

The Colorado Bald Eagle population is apparently recent in origin and, at the time of our study, consisted of 14 breeding pairs and was at least 300 km away from any other nesting population (i.e., Arizona and Yellowstone, in Montana and Wyoming). The northwestern Ontario population is part of a large contiguous population of nesting eagles which stretches from the Atlantic seaboard to the Pacific Ocean and numbers close to 10,000 nesting pairs (Stalmaster 1987). The study populations and sites are described in detail elsewhere (Grier 1974, Grier 1982, Kralovec et al. 1993).

During the breeding seasons of 1988–1991, we collected tissue samples from 72 nestlings (Colorado = 20 birds; Ontario = 52 birds) when young were 6–10 weeks old. Tissues included mature pin feathers, blood cells and serum and were analyzed using starch-gel electrophoresis (Stangel et al. 1992). Electrophoretic conditions and general staining procedures were those of Selander et al. (1971) and Harris and Hopkinson (1976). Each sample was scored at 32 presumptive genetic loci (Appendix 1). Buffer codes are identified as follows: A = amino propylmorpholine citrate–pH 6.0 (Clayton and Tretiak 1972); B = lithium hydroxide–pH 8.1 (Selander et al. 1971); C = tris citrate–pH 8.0 (Selander et al. 1971); D = tris maleate–pH 7.4 (Selander et al. 1971). Stain recipes were taken from Selander et al. (1971), Siciliano and Shaw (1976), Harris and Hopkinson (1977), and Lydeard et al. (1989). The following systems were not adequately resolved in our analyses: enolase, glyoxalase 1; guanylate kinase, inosine trophosphate, pyruvate kinase and triosephosphate isomerase.

RESULTS AND DISCUSSION

All loci were monomorphic except peptidase 2. There were eight heterozygotes and no homozygotes for the rare allele observed among the 52 birds from Canada (frequency of 8% [8/104]). Peptidase 2 was monomorphic among the birds from Colorado. The probability of not detecting this rare allele in Colorado from a sample of 20 birds is quite low (0.036 [0.92^{40}]).

Although the two eagle populations we studied differed dramatically in spatial isolation and population size, we found virtually no allozymic genetic variation between and within them. This study is one of very few cases where an apparent lack of variation was reported for a bird species in which a relatively large number of individuals and loci were screened (Bar-

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rowclough et al. 1985, Stangel 1992). Our findings are similar with those recently described by Barrowclough and Gutiérrez (1990) who used starch-gel electrophoresis to investigate genetic variability from seven populations of Spotted Owls (*Strix occidentalis*). No genetic variation was found in six populations from Oregon and California, however one locus in the New Mexico population was polymorphic. Barrowclough and Gutiérrez attributed the absence of genetic variation to either small effective population size or past bottlenecks. Effective population size is not a likely explanation for our findings as the Ontario sample is part of a large and extensive population. Past bottlenecks are a more plausible explanation as the Pleistocene glaciation may have limited effective habitat.

The near-absence of genetic variation between and within the two populations may also be attributed to gene flow of effectively neutral alleles. Bald Eagles are known to range widely, and one individually marked nestling from the Colorado population recently acquired a mate and bred in the Yellowstone population.

Our findings do not clarify the contested subspecific status of *H. l. leucocephalus* and *H. l. alascanus* (AOU 1957, 1983). Presence of one polymorphic loci in the Canadian population is supportive of subspecific status, however, as Morizot et al. (1985) suggest, there may only be a clinal gradation from south to north.

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APPENDIX 1. Thirty-two presumptive genetic loci, including buffer codes given in parentheses, examined from Canadian and Colorado Bald Eagle populations.

Aminoaspartate transaminase-1,2 (B); aconitase-1,2 (A); adenosine deaminase-1 (B); adenylate kinase-1 (C); albumin (B); aldolase-1 (C); acid phosphotase-1 (C); beta hemoglobin (B); carbonic anhydrase-1 (C); creatine kinase-1,2 (C); diaphorase-1 (C); esterase-1,2 (B; beta napthyl phosphate as the substrate); florescent esterase-1 (B; 4 methylumbelliferyl acetate); glyceraldehyde-3-phosphate dehydrogenase-1 (C); glucose-6-phosphate dehydrogenase-1 (D); glucose phosphate isomerase-1 (D); glutathione reductase-1 (C); alpha-glycerophosphate dehydrogenase (B); isocitrate dehydrogenase-1,2 (C); malate dehydrogenase-1,2 (D); malic enzyme-1 (D); menadione reductase-1 (C); mannose phosphate isomerase-1 (C); nucleoside phosphorylase-1 (C); peptidase-1 (B;leucyl glycyl glycine substrate), 2 (B;glycyl leucine), 3 (B;phenyl proline); phosphoglucomutase-1,2 (C); phosphogluconate dehydrogenase-1 (D); phosphoglycerate kinase-1 (C); L-ititol dehydrogenase-1 (A); superoxide dismutase-1,2 (C); and transferrin (B).