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Genetic Variability within and among Wintering Populations of Brant

James M. Novak
Eastern Illinois University, jmnovak@eiu.edu

L. M. Smith
Texas Tech University

L. D. Vangilder
Missouri Department of Conservation, Fish and Wildlife Research Center

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through the induction of meiosislike pairing and recombination of chromosomes in somatic tissue, be responsible for some of the somaclonal variation often reported in such studies.[6] Presumably, natural selection would favor cells carrying superior recombinations and would occasionally produce chimeras or entire regenerated plants with an altered genotype. Our failure to observe somaclonal variants among dozens of regenerated sorghum plants can be explained by the low frequency of somatic pairing and separation of chromosomes in our material or by a selective disadvantage of recombinant cells resulting from such processes. Somatic pairing and recombination of chromosomes per se, however, do not explain the novel nature of many somaclonal variants or indicate why such variants frequently cannot be recovered in sexual propagation.

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Genetic Variability within and among Wintering Populations of Brant

J. M. Novak, L. M. Smith, and L. D. Vangilder

Brant (Branta bernicla hrota) were collected from wintering populations in New York, New Jersey, and Virginia. Twenty-eight putative electrophoretic loci were examined to assess genetic variability and to quantify the genetic structure of wintering populations. Multilocus heterozygosity was not significantly different from that expected for an avian species. However, the percentage of polymorphism was lower than expected. One locus, PEP-1, exhibited a null allele, the first report of a null allele at an electrophoretic locus in waterfowl. The wintering populations exhibited some genetic differences, but the magnitude of the differences was small. We concluded that there is some restriction of gene flow between the wintering populations, and thus wintering populations of Brant do not represent a totally panmictic population in the strict sense. This is most likely a result of female philopatry and familial cohesiveness. However, the reduction in gene flow does not indicate strict concordance between breeding and wintering populations.

Brant (Branta bernicla hrota) winter in several discrete areas along the eastern coast of the United States from Massachusetts to North Carolina.[12] Brant populations have undergone dramatic fluctuations not only along the entire winter range but also independently in isolated wintering areas. For example, Kirby and Obrecht[12] stated that Brant populations in New York and Virginia have been increasing while those in New Jersey have been declining. The population fluctuations have been variously attributed to variations in food sup-

ply and nesting success.[3,8,12] The relationship between nesting and wintering populations of Brant becomes critical when attempting to establish whether local wintering population fluctuations are a response to conditions on the winter range, a result of breeding success or failure in different nesting areas, or a combination of both factors. This is especially important in geese such as Brant that arrive on the nesting grounds already paired.[3]

Raveling[18] reported that nesting populations of giant Canada Geese (B. canadensis maxima) are differentially represented on discrete wintering grounds, whereas Cooke et al.[7] found that Lesser Snow Geese (Chen caerulescens caerulescens) mix randomly on the wintering grounds. Traditionally, extensive banding programs have been used to establish the relationship between nesting and wintering populations. This method is expensive in both time and money and gives a poor return in terms of information per unit of effort. Hansen and Jones[10] suggested using feather mineral composition to establish the relationship between wintering and nesting waterfowl populations. Similarly, Smith et al.[21] suggested that these relationships could be established using genetic markers. We use electrophoretic techniques to test the null hypothesis that Brant wintering in different areas represent one genetic population.

Materials and Methods

We collected Brant by shotgun in Cape May County, New Jersey (N = 41), and Accomac County, Virginia (N = 13), and by shotgun and rocket netting in Nassau County, New York (N = 40). We collected all birds in January 1984 to avoid bias from migrational overlap earlier or later in the season. We collected an additional 12 birds in Virginia during January 1985 to establish temporal stability of a rare allele at the PGM locus. We saw no apparent genetic differences between years ($G = 0.28$, $P > .60$), but the small sample sizes and the distribution of data among cells provide little power for this test. Therefore, to prevent any temporal biases in the analysis and to allow for more conservative statistical hypothesis testing, we did not pool samples.

We sexed and aged birds upon collection. Brant have a collar of white feathers—the necklace—around their necks that can be scored for "completeness" according to the method of Boyd and Maltby.[5] We scored the birds "in hand" ac-

cording to Vangilder and Smith.[23] We collected liver samples in the field and packed them in dry ice until they could be stored in an ultracold freezer ($-70°C$). We performed starch gel electrophoresis; a total of 28 electrophoretically detectable loci were reliably scored on the buffer systems listed below. Amine citrate 6.1[6]: β-glucuronidase (*B-GUS*), hexokinase (*HK*), fumaric hydratase (*FH*), aconitate hydratase (*ACO*), adenylate kinase (*AK*). TRIS-maleic acid 7.4[19]: DL-Leucyl-L-alanyl peptidases (*PEP-1,2,3*), catalase (*CAT*), phosphogluconate dehydrogenase (*PGD*), xanthine dehydrogenase (*XDH*), malic enzyme (*MOD-1*), aldolase (*ALD*), glucose-6-phosphate dehydrogenase (*Gd*). TRIS-EDTA-Citrate 7.1[2]: α-naphthyl phosphate esterase (*ES*), phosphokinase (*PK*), glucose dehydrogenase (*GDH*), malic dehydrogenase (*MDH*). TRIS-Citrate 8.0[19]: phosphoglucomutase (*PGM*), isocitrate dehydrogenase (*ICD*), lactate dehydrogenase (*LDH*), NADH diaphorase (*DIA*), creatine kinase (*CK*). TRIS-EDTA-Borate 8.6[11]: purine nucleoside phosphorylase (*NSP*), sorbitol dehydrogenase (*SorDH*), mannose phosphate isomerase (*MPI*), and superoxide dismutase (*SOD-1,2*). Staining methods follow Shaw and Prasad,[20] Selander et al.,[19] Manlove et al.,[14] and Harris and Hopkinson.[11]

We conducted statistical analyses using the BIOSYS-1 program[22] except where corrections for null alleles were necessary. Null alleles cannot be unambiguously recognized in the heterozygous condition. We felt that intensity differences, being too subjective, introduced the possibility of an unacceptable level of scoring error. We therefore scored all single bands as homozygous for the appropriate visible allele. Therefore, expected values for testing deviations from Castle-Hardy-Weinberg (C-H-W) equilibrium had to be corrected to account for this fact. We added expected values for heterozygotes for the null allele to the expected value of the complementary allele in the homozygous condition and calculated allele frequencies using a maximum-likelihood estimator from direct counts of electromorphs. The exact algorithm is a modification of the ABO blood group allele frequency analysis in Li.[13] We interpreted F-statistics as variance proportions standardized to the maximum amount of genetic variation possible with the calculated gene frequencies.[24] F_{is} was the fixation index within a subpopulation. F_{IS} was the mean F_{is} over all subpopulations and therefore represents the proportion of variation due to differences be-

Table 1. Major allele frequencies for three wintering Brant populations in 1984

Locus	Allele	Virginia (N = 13)	New Jersey (N = 41)	New York (N = 40)
PEP-1[a]	100	0.633	0.647	0.720
	92	0.277	0.225	0.181
	null	0.090	0.128	0.099
PGD-1[a]	100	0.769	0.768	0.787
	83	0.231	0.232	0.213
PGM-1[a]	100	0.923	1.000	1.000
	77	0.077[b]	0.000	0.000
ICD-1	100	1.000	0.988	1.000
	127	0.000	0.012	0.000
Gd-1	100	1.000	0.988	0.975
	180	0.000	0.012	0.025
MPI-1	100	1.000	0.988	1.000
	125	0.000	0.012	0.000
GDH-1	100	1.000	1.000	0.987
	85	0.000	0.000	0.013

[a] Major variable loci, $P \le .95$ in at least one population.
[b] Also occurs in the 1985 Virginia sample ($P = .958$, $q = 0.042$, N = 12) and in a breeding population from Baffin Island, Northwest Territories ($P = .958$, $q = 0.031$, $r = 0.010$, N = 48).

tween individuals within a subpopulation. F_{ST} was the proportion of variation due to differences between subpopulations. The significance of F-statistics was calculated as suggested in Nei and Chesser,[15] and they were corrected for the presence of the null allele where necessary. All values were reported as mean $\pm$ 1 SE where variance estimates could be obtained. All tests were considered significant at $P \le .05$.

Results

Seven of 28 electrophoretic systems surveyed showed some variability in at least one of the populations. All systems were diallelic except *PEP-1*, which had three alleles, one of which was a null allele (Table 1). To our knowledge, this is the first time a null allele has been reported for an avian species, and therefore we will elaborate on this system. Using DL-Leucyl-L-alanine as the substrate, we found three zones of activity on the gel; these were referred to as 1, 2, and 3 from most to least anodal. Both *PEP-2* and *PEP-3* were monomorphic and exhibited approximately equal intensity for all samples. *PEP-1* exhibited four banding patterns on the gel, a single-banded pattern for both the 100 and 92 alleles, a double-banded pattern for the 100/92 heterozygote, and a complete absence of banding for the null-allele homozygote. It must be stressed that individuals interpreted as null-allele homozygotes exhibited banding patterns at *PEP-2* and *PEP-3* as dark as or darker than any other individual and were scored blind three different times by three different researchers.

Table 2. F statistics for three wintering Brant populations

Locus	F_{is} Virginia	New Jersey	New York	F_{IS}	F_{ST}
PEP-1	.118	−.310[a]	−.187	−.126	.003
PGD-1	−.300	−.165	.029	−.149	.000
PGM-1	−.083	—[c]	—[c]	−.083	.053[b]
ICD-1	—[c]	−.012	—[c]	−.012	.008
Gd-1	—[c]	−.012	−.026	−.021	.009
MPI-1	—[c]	−.012	—[c]	−.012	.008
GDH-1	—[c]	—[c]	−.013	−.013	.008

[a] $P < .010$, critical value for multiple tests = .0102.
[b] $P < .005$, critical value for multiple tests = .0073.
[c] Locus monomorphic in population.

Thus, it appears unlikely that the null-allele homozygotes were an artifact of sample storage or preparation.

The mean direct-count multilocus-heterozygosity estimate was 0.033 ± 0.020 in Virginia, 0.032 ± 0.020 in New Jersey, and 0.026 ± 0.016 in New York. This was lower than the mean heterozygosity value reported by Nevo et al.[16] of 0.051 ± 0.029 for 46 avian species but still falls within the range of variability exhibited by birds. The percentage of polymorphism at the 5% and 1% levels, respectively, was 10.7% and 10.7% in Virginia, 7.1% and 17.9% in New Jersey, and 7.1% and 14.3% in New York. The percentage of polymorphism at the 1% level for 56 avian species was $30.2 \pm 14.3\%$.[16] The three systems that showed variability at $P \ge .05$ displayed a weak trend of increasing major-allele frequency from south to north (Table 1).

The Virginia population contained a unique allele at the *PGM-1* locus, at low frequency, that occurred in neither the New Jersey or New York populations. The occurrence of this allele in the population from which the fewest birds were taken is contrary to expectations based on sample size considerations. The Virginia population also lacked the homozygotes for the null allele at the *PEP-1* locus that was found in the New York and New Jersey populations. However, this absence is most likely an artifact of the small sample size from Virginia. Allelic composition for both the *PEP-1* and *PGM-1* loci was not significantly different between collection periods for the Virginia population. The allelic difference at the *PGM-1* locus results in a significant F_{ST} value at this locus. The Virginia population appears to differ slightly, on a genic basis, from those in New Jersey and New York (Table 2).

Neither the New Jersey nor the New York population contained unique alleles at a frequency of 0.05 or greater. Thus, the two

populations were not expected to differ based on F_{ST} values because the allelic frequencies did not show sufficient differences between the populations (Tables 1 and 2). These two populations differed in their genotypic deviations from C-H-W equilibrium at the *PEP-1* locus (New Jersey: $G = 7.88$, $P < .01$; New York: $G = 2.79$, $P > .05$). This difference was evidenced by the fact that the only significant inbreeding coefficient (F_{is}) was for *PEP-1* in the New Jersey samples (Table 2). The populations of Brant from New York and New Jersey also differed based on a morphological character, necklace type.[23]

Discussion

Our results indicate that the Brant population in Virginia differs slightly, on a genic basis, from those in New Jersey and New York. The New Jersey population differs genotypically from the New York population, but we concluded that neither of these populations differs as much from each other as they do from the Virginia population. The amount of differentiation, although measurable, is slight, and does not suggest a one-to-one correspondence between nesting and wintering populations but rather some small reduction of gene flow between the wintering populations on the migration route and/or on the wintering grounds or some degree of nonrandom migration between nesting and wintering populations. This pattern is also seen in the distribution of necklace types among these wintering populations.[23]

The degree of local differentiation appears to be more similar to that exhibited by giant Canada Geese[18] than to that exhibited by Lesser Snow Geese.[7] This is reasonable, because the geographic pattern of discrete nesting populations of Brant is similar to that found in giant Canada Geese. Lesser Snow Geese have more continuous nesting populations.[4] In addition, Brant are genetically more similar to Canada Geese than to Snow Geese.[17] Differential sexual expression of breeding-ground philopatry is the accepted explanation for the varied patterns of local differentiation exhibited by giant Canada Geese and Lesser Snow Geese on the wintering grounds. In both species the females exhibit a philopatric response in traveling between nesting and wintering sites, but only the males of giant Canada Geese show a similar philopatric response.[7,18] We predict that Brant should show a pattern of philopatry more similar to that of giant Canada Geese than to that of Lesser Snow Geese. An indirect test of

this hypothesis would examine the genotypic distribution among the wintering populations of female Brant and compare this with the pattern exhibited by male Brant. If the pattern of philopatry is like that of Canada Geese, then we should find no significant sexual differences in the genotypic distributions. This is the case in Brant. There is no difference in the genotypic distributions between sexes for either the New Jersey $(G = 2.41, P = .491)$ or the New York $(G = 2.81, P = .422)$ population.

The situation in New Jersey is complicated by the fact that Brant exhibit positive assortative mating by necklace type.[1] Necklace type and *PEP-1* genotype are positively associated in Brant (J. M. Novak et al., unpublished observations) and thus are functionally or physically linked. Positive assortative mating introduces an "inbreeding" effect and, usually, a confounding selective effect on the character on which assortment occurs (and on any associated characters).[9] A simple population-genetic model based on positive assortative mating by necklace type is sufficient to explain the deviation from C-H-W equilibrium for the *PEP-1* locus in the New Jersey population (J. M. Novak et al., unpublished observations). Thus, the genotypic differences between New Jersey and New York Brant populations seem to be attributable to the proportional distribution of necklace types within each population. This discrete and easily recognized morphological character could act to increase the cohesiveness of small interbreeding groups and cause a functional subdivision within the New Jersey population.

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Two Recessive Rex Coat Mutants in the Guinea Pig

C. E. Whiteway and R. Robinson

Two rex type coat mutants of the guinea pig were found to display monogenic recessive inheritance at independent loci. The mutant alleles were designated rex (*rx*) and waved (*wv*). Both genes modify the normal smooth coat to a more upright, somewhat unkempt pelage. Macroscopically, the two rexes are scarcely distinguishable. Microscopically, however, small differences are apparent in the degree of coat modification. The hairs of rex show a greater curvature than normal and have irregular secondary bends and twists and variable diameter; so do those of waved, but to a lesser degree. The vibrissae of rex are curved or bent and may break off; those of waved are a mixture of straight, curved, and bent hairs.

A distinctive type of coat mutant in mammals is known as rex, so called after the breed of rabbit of that name, which seems to have been the first mutant of the type to be recognized (circa 1919).[7] Rex mutants are characterized by anomalous vibrissae that may be shorter than normal, bent or crinkled and that may break easily and by a wavy juvenile coat that changes into a loose, unkempt coat in adults. Interestingly, many loci engendering rex-mutant alleles are found in several species. These are known by various designations, the more usual, in addition to rex, being waved and curly. The majority of rex-type mutants are recessive, but a number are dominant to normal coat. The mode of inheritance is of doubtful significance, given that dominance in phenotypic terms is a function of the species genome. Laboratory rodents have the greatest array of rex mutants but similar alleles occur in dogs,[14] cats,[11] cattle,[3] and horses.[2]

Materials and Methods

The guinea pig rex-mutant alleles occurred spontaneously on two continents. The first, designated rex, came to notice in England about 1975, although the allele may have occurred a few years earlier (Figures 1 and 2). The second, designated waved, was found in the United States around 1971, and specimens were imported into England in 1983; again, the mutant allele probably occurred a number of years earlier. The English locus will be referred to as rex and the U.S. locus as waved.

The hairs of the guinea pig do not appear to be differentiated into distinctive forms, such as guard hairs, awls, auchenes, and zigzags, as in the mouse or the rat. Hairs that might be comparable to auchenes and zigzags seem to be absent; the guinea pig coat appears to be composed entirely of guard and awl hairs without there being a sharp distinction between them. The length and diameter of the hairs is correlated, with the slightly shorter and thinner hairs outnumbering the stouter. The hairs are gently curved, and each has a well-defined bulb at the base and increases steadily in diameter to a subapical maximum before tapering to a fine point.

There is a corresponding lack of differentiation in the hairs of the rex coat. The hairs are, however, clearly abnormal. They are more curved, with most having several irregular minor bends throughout their length, giving the impression of crookedness. The hair bulb may be less well formed. Diameter generally increases along the length of the hair, but irregularly; it may even decrease slightly for short distances. The point may be either blunted or thinly drawn out. The hairs seem shorter, but this may be due to the greater curvature and irregular secondary bends.

The waved coat resembles the rex coat but does not seem to differ so markedly from normal; for example, the hairs are somewhat less curved and crooked than in rex. Nevertheless, distinguishing macroscopically between the two rexoid coats is difficult to impossible. Both rex and waved display marcel-style waving in the juvenile coat and a rough, unkempt adult coat. The color is slightly but perceptibly darker than normal for both rex and waved mutants of very pale phenotypes.

The mode of inheritance of the rex coat is shown in Table 1. The fact that most of the data were supplied by fanciers explains why backcrosses of the two F_1s to the respective mutant parents were made rather than more orthodox F_2 matings. Nevertheless, it is evident from the progeny of the various matings that both rex and waved coats are inherited as monogenic recessives to normal coat. In particular, rex × waved produced 12 normal-coated offspring, indicating that the genes are at independent loci. Accordingly, the rex gene will be symbolized by *rx* and the waved gene by *wv*.

Table 1. Assortment of the *rx* and *wv* genes and a test for independence of the genes in the guinea pig

Mating	Normal	Rex	Waved
++ × *rxrx*	31	—	—
+*rx* × *rxrx*	4	9	—
+*rx* × +*rx*	2	1	—
rxrx × *rxrx*	—	24	—
+*wv* × *wvwv*	6	—	3
wvwv × *wvwv*	—	—	3
rxrx × *wvwv*	12	—	—

Discussion

The two rex-mutant alleles described in this report have phenotypes very characteristic of this type of hair anomaly. Lovell[5] described another rex (curly) that also has typical characteristics: curly or bent vibrissae, a wavy juvenile coat, and a "fluffed up" adult coat. The curly coat is inherited as a dominant, which distinguishes it from the rex and wavy alleles of this report. The existence of three different mutants for the guinea pig (with the involvement of at least two independent loci) implies that it can be added to the list of species with numerous loci that are capable of producing a rex-type coat.

The cat has eight rex mutants involving at least three loci.[9,11,13] The Norway rat has six rex mutants, probably involving as many loci.[4,8,12] The rabbit has three rex mutants at independent loci.[1] The Syrian hamster has one rex mutant.[10] The house mouse has the greatest number of rex or quasi-rex mutants, befitting its commanding role in mammalian genetics; depending on how strictly the criteria for a rex-type mutant are applied, the house mouse has as many as eight to fifteen loci, a few with more than one mutant allele. The majority are inherited as recessive to normal coat.[6]

Examination of the phenotypes of the rex-type mutants reveals that the degree to which the coat is affected varies from slight to severe modification of hair morphology, with some mutants displaying recurring alopecia. The typical rex coat could be classed as a mild form of hypotrichosis. A number of mutants have partial loss of hair, e.g., Devon rex and Dutch rex in the cat[11,13] and rex in the rat.[12] The comparative microscopic study of rex mutants in the mouse by Trigg[15] revealed subtle but