Journal of Mammalogy, 85(1):58–66, 2004

THE INFLUENCE OF SOCIAL BREEDING GROUPS ON EFFECTIVE POPULATION SIZE IN BLACK-TAILED PRAIRIE DOGS

F. Stephen Dobson,* Ronald K. Chesser, John L. Hoogland, Derrick W. Sugg, and David W. Foltz

Department of Biological Sciences, Auburn University, Auburn, AL 36849, USA (FSD)
Department of Biological Sciences, Texas Tech University, Lubbock, TX 79409, USA (RKC)
Appalachian Laboratory, University of Maryland, Frostburg, MD 21532, USA (JLH)
University of Georgia, Savannah River Ecology Laboratory, Aiken, SC 29802, USA (DWS)
Department of Biological Sciences, Louisiana State University, Baton Rouge, LA 70803, USA (DWF)

Effective population sizes reported in the literature typically range from a small fraction of the adult population to about half the number of breeding adults. Theoretically, however, social structuring of genetic diversity could produce effective sizes as great as or even greater than population size. A colony of the highly social black-tailed prairie dog (*Cynomys ludovicianus*) was studied in the field for 16 years, and data were gathered for estimation of effective population sizes from pedigrees, demography, and allozyme alleles. Social breeding groups ("coteries") within the colony exhibited high correlations of genes among individuals, and different coteries exhibited substantial genetic differentiation. Genetic diversity thus occurred within individuals, within coteries, and among coteries, and shifted among these levels of organization over time. "Instantaneous" estimates of effective size from short-term (annual) changes in genetic correlations were calculated from pedigree information but were not useful because they produced a wide diversity of estimates, due in part to the lack of demographic and genetic equilibrium in the colony. "Asymptotic" measures of effective population size that assumed eventual genetic equilibrium yielded relatively consistent estimates of effective sizes. For 10 years of empirical results from prairie dogs, effective population sizes from pedigrees (harmonic mean = 79.4), demographic model based on breeding groups (asymptote = 88.5), and allozyme data (harmonic mean = 88.9) were similar, and all were somewhat higher than the number of adults in the population (harmonic mean = 74.1). The colony of prairie dogs, therefore, exhibited a lower rate of loss of genetic diversity than expected, due to the genetic substructure created by the presence of social breeding groups.

Key words: allozymes, demography, effective population size, genetics, pedigree, social structure

Rate of loss of genetic diversity in a population is often estimated by a population's effective size (N_e)—the size of an ideal population that loses genetic variation at the same rate as the actual population under study (Wright 1969). Ideal populations exhibit random mating and have no substructure due to philopatry, mating patterns, or aggregation of related individuals, but real populations may have internal genetic substructure due to the presence of social breeding groups (groups in which most matings and social interactions occur between group members). For example, if females in breeding groups are philopatric (i.e., recruit as breeders in the natal group, as occurs in many mammalian species—Dobson 1982; Greenwood 1980; Waser and Jones 1983), then subdivision of the population may occur via kinship of adjacent individuals (Chesser 1991a, 1991b). Also, polygynous breeding groups likely violate the assumption of random mating, since groups of females may be more likely to mate with some males than with others (Nunney 1993). Failure to recognize and account for genetic substructure resulting from the presence of breeding groups could produce bias in estimates of N_e (Chesser et al. 1993; Sugg and Chesser 1994; Sugg et al. 1996).

N_e can be measured from changes in the rate of inbreeding over time ("inbreeding effective size," $N_{eI} = 1/[2\Delta F]$, where F is the coefficient of inbreeding—Wright 1969; Crow and Denniston 1988). If a population has genetic substructure due to breeding groups, however, changes in genetic diversity are not adequately described solely by ΔF. Genetic correlations

* *Correspondent: fsdobson@msn.com*

may occur within a breeding group (defined as coancestry, θ, the mean correlation of genes between individuals within the group) and also among breeding groups (described by α, the mean correlation of genes between individuals from different groups—Chesser 1991a, 1991b; Cockerham 1967, 1969, 1973). Thus, genetic variation in a population occurs within individuals (as heterozygosity), among individuals, and because of population substructure. In addition, genetic variation in a population can shift back and forth between these levels, if demographic variables such as group size and dispersal rates vary. An understanding of gene dynamics of socially structured populations, therefore, requires knowledge about changes in genetic diversity at different levels of population structure (Dobson et al. 1997, 1998; Sugg et al. 1996).

Fixation indices (F-statistics—Wright 1965, 1969) reflect differences in genetic correlations from what would be expected if mating were random. For example, F_{IL} measures the degree to which F differs from that expected if mating was random among offspring in a breeding group (Chesser 1991a, 1991b). $F_{IL} < 0$ indicates less breeding with kin than expected, $F_{IL} > 0$ reflects closer inbreeding than expected, and $F_{IL} = 0$ suggests random mating. Similarly, F_{IS} compares F to an expectation of random mating among offspring from different breeding groups, and F_{LS} compares θ to an expectation based on random mating among offspring from different breeding groups. F-statistics provide a more convenient measure of gene dynamics than short-term changes in genetic correlations. These fixation indices can also be used to estimate the effective sizes of populations (e.g., Caballero and Hill 1992; Chesser et al. 1993). Chesser et al. (1993) and Sugg and Chesser (1994) provided a theoretical framework for calculation of N_es from the fixation indices of socially structured populations. These "breeding-group" F-statistics and N_es reflect changes in genetic correlations caused by patterns of philopatry and dispersal, mating systems, and even details of mating systems such as multiple paternity.

Various methods have been used to estimate the loss of genetic diversity from natural populations. Ecological models incorporate information about the size and number of population subunits, sex ratio of adults, mating patterns, and dispersal rates of males and females (Chesser and Baker 1996; Chesser et al. 1993; Nunney and Elam 1994). Such demographic and mating variables may differ from the assumptions of idealized populations; violations of assumptions must be considered if ecological models are to estimate gene dynamics accurately, including the loss of genetic diversity (Dobson 1998). An alternative to ecological modeling is estimation from biochemical markers (e.g., allozyme alleles or DNA fragments—termed "genetic methods" by Nunney and Elam 1994). Alternatively, pedigrees may be applied to estimate the loss of genetic diversity (this method has seldom been used—Blackwell et al. 1995; Long et al. 1998).

The purpose of our study was to examine changes in genetic diversity of a population of a highly social, group-living mammal, the black-tailed prairie dog (*Cynomys ludovicianus*). The black-tailed prairie dog is an ideal model for examining the loss of genetic variation in a species with breeding groups. Prairie dogs live in polygynous social groups called "coteries," that commonly contain 1–2 males and 2–4 females, plus yearling and juvenile offspring of the females (Hoogland 1995; King 1955). Several contiguous coteries clump spatially to form a local population called a "colony" or "town," and colonies are distributed patchily across the local landscape. Most females are philopatric to their natal coterie, and all males that survive to breed disperse from their natal coteries before sexual maturity. Most dispersal occurs within colonies, but some occurs among colonies (Garret and Franklin 1988; Hoogland 1995).

Our purpose was to study gene dynamics, with particular attention to the coterie level of population structure and the loss of genetic diversity within a single colony. We used 3 techniques to estimate F-statistics and N_e for our prairie dog colony: analyses of pedigrees, predictions of an ecological model, and examination of allozyme variation. Pedigrees reflected the actual breeding structure, and thus should produce accurate estimates of genetic correlations, F-statistics, and N_e. Ecological predictions of F-statistics and N_e were derived from breeding-group models of Chesser (1991a, 1991b), Chesser et al. (1993), and Sugg and Chesser (1994). Finally, we used allozyme data to estimate F-statistics and N_e.

Materials and Methods

Field observations.—Prairie dogs were studied in the field from 1975 through 1989 at Wind Cave National Park, Hot Springs, Custer County, South Dakota (Hoogland 1995). The study colony (1,300 m elevation; about 1 km southwest of Rankin Ridge) occupied about 6.6 ha of meadowland surrounded by coniferous woodland and additional meadow, and measured roughly 500 m (N to S) by 130 m (E to W). The meadow was often grazed by pronghorn (*Antilocapra americana*) and American bison (*Bison bison*). Predators of prairie dogs included coyotes (*Canis latrans*), bobcats (*Lynx rufus*), badgers (*Taxidea taxus*), prairie falcons (*Falco mexicanus*), and golden eagles (*Aquila chrysaetos*). The number of adult and yearling prairie dogs in the colony during May of each year averaged 123 (range 92–143 over 14 years). The number of juveniles weaned in the colony annually averaged 88 (range 41–133). On average, the colony contained 21 coteries (range 15–26).

Adult prairie dogs were captured in live traps baited with whole oats, and juveniles were captured in unbaited traps (Hoogland 1995). Every prairie dog at the study colony was captured at least once each year. Individuals were fitted with numbered metal ear-tags (National Band & Tag Co., Newport, KY) for permanent recognition and dye-marked (Clairol Hydrience Black Pearl, Proctor and Gamble Inc., Stamford, CT) for behavioral observations. Marked prairie dogs were observed during their diurnal activity period from towers at the edges of the study colony. Behavioral evidence of underground matings suggested possible fathers, and observations on all litters that emerged from natal burrows allowed identification of lactating adult females that were the mothers of these litters. Males and females usually began reproducing at 2 years of age, but occasionally yearling females bred. Mating occurred during February and March. Weaning of offspring occurred in May and June, shortly after emergence of juveniles from natal burrows and about 76 days after mating.

Pedigree estimates.—Pedigrees were constructed from field identification of mothers and offspring and assignment of paternity from field observations, combined with maximum-likelihood analyses of

allozyme data (Foltz and Hoogland 1981; Hoogland and Foltz 1982). These pedigrees were used to estimate average correlations of genes for offspring in each year: of individuals to themselves (mean inbreeding coefficient = F), between different individuals in the same coterie (coancestry within coteries = θ), and between different individuals from different coteries (α). In turn, these genetic correlations were used to calculate fixation indices. Each individual captured was assigned a unique identification number, year born, and coterie designation for each year. Sire and dam identification numbers were noted for each offspring. If sire or dam was unknown (as for immigrants or individuals already present in the population at the beginning of the study—1975), then that individual was assigned a genic correlation = 0 relative to other colony residents. Coancestry between any pair i, j of individuals was estimated as

$$\theta_{i,j} = \frac{1}{4}(\theta_{S_iS_j} + \theta_{S_iD_j} + \theta_{S_jD_i} + \theta_{D_iD_j}) \quad (1)$$

where S and D denote sire and dam, respectively, of the ith and jth individuals. This expression can be used to describe the way in which coancestry accumulates over the generations. The coancestry of an individual to itself is $\theta_{i,i} = (1+F_i)/2$, and the inbreeding coefficient of a progeny is equal to the coancestry of its parents:

$$F_i = \theta_{S_iD_i}. \quad (2)$$

The weighted average coancestry within coteries for each year was estimated by the summed pair-wise values from the pedigree for each coterie, divided by the number of pairs (dyads) in the ith coterie $[N_i(N_i - 1)]/2$ (where N is the number of individuals), averaged across the coteries (s) in the population (Chesser 1991a, 1991b; Cockerham 1967, 1969, 1973):

$$\bar{\theta} = \frac{1}{s}\sum_{i=1}^{s}\frac{2}{N_i(N_i-1)}\sum_{j=1}^{N_i-1}\sum_{k=j+1}^{N_i}\theta_{i,j,k} \quad (3)$$

Similarly, the mean correlation of gene frequencies among groups (α) was determined from the mean coancestry of all individuals in different coteries:

$$\bar{\alpha} = \frac{\sum_{i=1}^{s-1}\sum_{j=1}^{N_i}\sum_{k=i+1}^{s}\sum_{m=1}^{N_k}\theta_{j,m}}{\sum_{i=1}^{s-1}\sum_{k=i+1}^{s}N_iN_k} \quad (4)$$

In this calculation, s was the number of coteries (i.e., breeding groups) in the colony. Lastly, the average inbreeding coefficient was determined over all individuals observed in the population (N_T) for a given year:

$$\bar{F} = \frac{1}{N_T}\sum_{i=1}^{N_T}F_i \quad (5)$$

Over a single generation (about 3 years for this species; Hoogland 1995), change in inbreeding at the individual level can be estimated as (Falconer 1989; Sugg and Chesser 1994):

$$\Delta F = \frac{(F_{t+1} - F_t)}{(1 - F_t)} \quad (6)$$

Because changes in inbreeding were more easily measured from year to year, we estimated annual changes rather than generational changes. This allowed for more complete presentation of results and did not change any conclusions; an assumption of non-overlapping generations would be made in either case. Violation of this assumption delays attainment of asymptotic values of N_e, but should not alter the asymptote greatly (Hill 1979). Instantaneous N_es were estimated for annual periods as (Chesser et al. 1993; Sugg and Chesser 1994):

$$N_{eI} = \frac{1}{2\,\Delta F} \quad (7)$$

Instantaneous N_es for θ and α (viz., $N_{e\theta}$ and $N_{e\alpha}$, respectively) were also estimated in the same way (i.e., $\Delta\theta$ and $\Delta\alpha$ were calculated as in equation 6, and substituted for ΔF in equation 7).

For comparison with ecological and genetic estimates, we calculated asymptotic effective population size from the pedigrees for offspring, since such pedigrees produce accurate estimates of gene dynamics (Dobson et al. 1997). To estimate the asymptotic value for a particular time t, we first calculated time-specific F-statistics (fixation indices). Mean values from equations (3), (4), and (5) were substituted into the following equations to determine F-statistics for the rate of inbreeding of individuals with respect to the coterie, of individuals relative to the colony, and of coteries relative to the colony (Chesser et al. 1993; Cockerham 1967, 1969, 1973):

$$F_{IL} = \frac{F - \theta}{1 - \theta} \qquad F_{IS} = \frac{F - \alpha}{1 - \alpha} \qquad F_{LS} = \frac{\theta - \alpha}{1 - \alpha} \quad (8)$$

where L denotes the lineage of a breeding group. Mean annual values of coancestries were calculated for several years of study (1979–1988), and each yearly mean was compared to estimates from previous years to ensure accuracy. For each year of the study from 1979 to 1988, we used these F-statistics to estimate N_e (Chesser et al. 1993; Sugg and Chesser 1994):

$$N_e = \frac{1}{2F_{LS}\left[\frac{d_m + d_f - d_md_f}{2s} + \frac{(kn-1)(d_m+d_f)}{4(kns-1)}\right]} \quad (9)$$

For this estimate, we used the average dispersal rates of males (d_m = 1.00) and females (d_f = 0.02) among coteries, the mean number of coteries in the colony (s = 20.83), the mean number of breeding females per coterie (n = 2.65 ± 0.14) during 14 years of study. The average reproductive success of females (k = 1.974 ± 0.15, based on 118 females) was calculated from the long-term data set, but restricted to females that themselves survived to at least 2 years of age, and included only offspring that survived to at least 2 years of age. Equation (9) is only appropriate for populations that are fairly stable in group number and population size, and that do not exhibit extremely high rates of inbreeding (Chesser 1998; Chesser and Baker 1996). The colony of prairie dogs met these conditions (Dobson et al. 1997; Hoogland 1992). Equation (9) estimates the variance effective size under specific conditions of θ and α that prevail at a given time, and thus may vary slightly from year to year.

The breeding-group model.—We used the breeding-group model of Sugg and Chesser (1994) for making our "ecological" estimates of N_e because it incorporates the breeding structure of a colony and allows for multiple paternity within litters and over the lifetime of females. To estimate genetic correlations and F-statistics for the breeding-group model, it was necessary to obtain means and variances for several model parameters. The number of coteries (s) and numbers of adult males (m) and females (n) in a coterie were averaged over all years of the study. The remaining parameters served to define reproductive success and were based only on the lifetime of individuals that produced surviving progeny of reproductive age. Because prairie-dog lifespan was approximately 5 years and most prairie dogs did not mate until they were 2 years old, data from the last 2 years of the study (1987–1988) were not used for estimating these parameters. The remaining parameters were the mean (k) and variance (σ_k^2) of the number of progeny that survived to maturity for adult females,

the mean (b) and variance (σ_b^2) of the number of female mates of each male that produced surviving progeny, the mean (p) and variance (σ_p^2) of the number of surviving progeny of a female sired by a single male, and the average number of male mates of each female (l). Finally, dispersal of males (d_m) and females (d_f) was calculated as the proportion of individuals that moved from their natal coteries and successfully reproduced in other coteries. These last 2 parameters were estimated using all years of data for individuals where sites of birth were known.

The above parameters were used to calculate the breeding parameters for the model developed by Chesser et al. (1993) and Sugg and Chesser (1994). The first parameter (ϕ_m) defines the probability that 2 randomly chosen progeny in the same breeding group (coterie) are the progeny of the same male. This parameter estimates the genetic polygyny of the average breeding group and is calculated with the following formula:

$$\phi_m = \frac{m[\sigma_b^2 + b(b-1)]}{ln(n-1)} \tag{10}$$

The second parameter (ϕ_f) defines the probability that 2 randomly chosen progeny in a coterie had the same mother, thus termed the probability of shared maternity:

$$\phi_f = \frac{\sigma_k^2 + k(k-1)}{k(kn-1)} \tag{11}$$

The final breeding parameter (ϕ_w) estimated the probability that 2 randomly chosen progeny produced during the lifetime of a female were sired by the same male. This parameter indicated the probability of single paternity over a female's lifetime reproductive success (in the case of prairie dogs, multiple paternity primarily resulted from mating with different males in different years):

$$\phi_w = \frac{l[\sigma_p^2 + p(p-1)]}{k(k-1)} \tag{12}$$

The breeding parameters and ecological data can be used in a series of transition equations to determine the expected change in genetic correlations between generations (Chesser 1991a, 1991b; Chesser et al. 1993; Sugg and Chesser 1994; corrections suggested by Wang 1997 were applied). We made the conservative assumption that the population started with unrelated individuals (i.e., that F, θ, and α were initially zero), and estimated genetic correlations were obtained for generations subsequent to the initial generation until genetic equilibrium was approximated. Annual changes in genetic correlations were substituted into equation (7) to produce instantaneous estimates of N_e. Also, genetic correlations were used to calculate the asymptotic fixation indices F_{IL}, F_{IS}, and F_{LS} from equation (8). F_{LS} was then substituted into equation (9) to produce the breeding-group prediction of asymptotic N_e.

Allozyme estimates.—Blood samples were collected from most of the prairie dogs in the colony studied. Some litters were eliminated by infanticide (usually below ground—Hoogland 1985, 1995) so these young could not be sampled. Horizontal starch-gel electrophoresis was performed (Harris and Hopkinson 1976; Selander et al. 1971), with staining for 4 polymorphic loci: transferrin (3 alleles), nucleoside phosphorylase (3 alleles), 6-phosphogluconate dehydrogenase (2 alleles), and phosphoglucomutase-2 (4 alleles). Data from all 4 loci were available for analyses for the years 1980 to 1988, but in 1979 only transferrin and nucleoside phosphorylase had sufficient sample sizes for analyses. Additional variable loci (esterase and mannose phosphoisomerase) were available for only a small sample of years so were excluded from analyses.

F-statistics were calculated for each electrophoretic locus using standard methods (Nei 1977; Wright 1978). Corrections for small sample sizes (Nei and Chesser 1983; Weir and Cockerham 1984) were not applied because blood was collected and analyzed from virtually all individuals in the colony so coteries and colony were not subject to population sampling error (loci, however, were sampled). Coteries were used as population subdivisions, so that the F-statistics were calculated relative to breeding groups. This procedure yields results from standard F-statistics procedures (that produce estimates of F_{IS}, F_{IT}, and F_{ST}) corresponding to breeding-group F-statistics (F_{IL}, F_{IS}, and F_{LS}, respectively). For each year from 1979 to 1988, breeding-group F-statistics were substituted into equation (9) to produce estimates of N_e from allozyme data.

Results

Pedigrees of offspring yield fairly accurate estimates of gene dynamics within populations (Spielman et al. 1977; Chesser 1991b) and we examined genetic correlations of offspring to estimate loss of genetic variation in the population. Over the study, genetic correlations within individuals (F) increased from 0.000 to 0.050, genetic correlations among individuals within coteries (θ) increased from 0.206 to 0.233 (minimum = 0.181), and genetic correlations among coteries within the colony (α) increased from 0.003 to 0.031 (maximum = 0.034). The relatively high value of coancestry (θ) of offspring resulted mainly from male polygyny and female philopatry within coteries (Chesser 1991b).

We subtracted the genetic correlations from 1 to estimate remaining genetic variation relative to total genetic variation present in 1978 (Fig. 1a). Although genetic variation at all levels of population substructure declined during the study, all levels exhibited periods of increased as well as decreased genetic variation. We estimated annual N_es from inter-annual changes in genetic correlations, with N_{eI} estimated from the change in F, $N_{e\theta}$ estimated from the change in θ, and $N_{e\alpha}$ estimated from the change in α (Fig. 1b). For young (offspring) ranges were N_{eI}, −247 to 248; $N_{e\theta}$, −108 to 75; and $N_{e\alpha}$, −259 to 356. It is important to remember that these estimates are time-specific, for year-to-year changes in genetic correlations. Negative values indicate years in which genetic correlations decreased from year to year (reflecting increases in genetic variation), and positive values indicate years in which genetic correlations increased (reflecting decreases in genetic variation). Separate analyses of adults (the parental generation; genetic correlations not presented) exhibited a similar pattern: N_{eI}, −495 to 498; $N_{e\theta}$, −258 to 1,373; and $N_{e\alpha}$, −76 to 385.

Between 1977 and 1988, there was an average of 20.8 coteries (s), with 2.65 adult females (n), and 1.38 adult males (m) in each of the coteries. Mean annual values of n ranged from a maximum of 3.09 (1977) to a minimum of 2.24 (1982). Mean values of m ranged from a maximum of 2.21 (1983) to 0.74 (1988). The mean number of progeny produced by a female (k) was 2.00 (range = 1 to 4), with a variance (σ_k^2) of 1.44. The mean (b) and variance (σ_b^2) in the number of females mated by a male were 1.68 (range = 1 to 3) and 0.66, respectively. The average number of successful mates per female (l) was 1.25 (range = 1 to 2). The average number of progeny produced by a female that shared the same father (p) may be estimated as k/l, and was 1.60,

with a variance (σ_p^2) of 0.64. Finally, the male (d_m) and female (d_f) dispersal rates were 1.00 and 0.02, respectively. Using these estimates, the degree of genetic polygyny (ϕ_m) was 0.46, the probability of shared maternity (ϕ_f) was 0.40, and the probability of single paternity (ϕ_w) was 1.00.

From 1979 to 1988, genetic correlations within individuals (F) increased from 0.006 to 0.053, genetic correlations among individuals within coteries (θ) from 0.157 to 0.201, and genetic correlations among coteries within the colony (α) from 0.005 to 0.051. We again subtracted the genetic correlations from 1, to estimate the remaining genetic variation relative to the total genetic variation present in 1978 (Fig. 2a). Because the breeding-group model was derived from the ideal-population model, the resulting equilibrium changes in genetic correlations reveal a constant decline in the remaining genetic variation. We again estimated annual N_es from interannual changes in genetic correlations (Fig. 2b). Estimates of all 3 genetic correlations produced the same asymptotic estimates of N_e of 92.9 (Chesser et al. 1993; Sugg and Chesser 1994). Estimates were made using equation (7), using F, θ, and α as inbreeding coefficients (i.e., in place of F in the equation).

Because natural-history variables were averaged across the entire period of study, only a single asymptotic value of each F-statistic was calculated by the breeding-group model: F_{IL} was estimated at about -0.18, F_{IS} at 0.00, and F_{LS} at 0.16 (Table 1). F_{LS} was substituted into equation (9) to yield the asymptotic estimate of N_e of 88.5, a value close to the instantaneous model asymptote of 92.9. Model predictions of N_e began in 1978, so that asymptote was reached by 1981 (Fig. 3). Genetic correlations from offspring pedigrees in each year were also substituted into equation (8) to calculate asymptotic fixation indices. The F_{LS} estimate was then substituted into equation (9) to produce annual estimates of N_e for 1979 through 1988. Estimates of N_e averaged about 79.4 individuals (harmonic mean), and varied from 70.6 to 92.4 among the years from 1979 to 1988.

Fixation indices were also calculated from allozyme data for all individuals that were captured in the populations within each year, and were averaged across genetic loci (Table 1). N_es were calculated from the allozyme data for different years of the study by substituting annual estimates of F_{LS} into equation (9). Estimated N_es from the allozyme data averaged 88.9 individuals (harmonic mean), and ranged from 70.5 to 115.1 in different years (Fig. 3). For comparisons with the above asymptotic estimates of N_es, we estimated the size of the potential breeding population. All males and females older than yearlings and any yearlings that showed signs of active breeding (scrotal testes for males; females that produced litters) were included. The mean size of the breeding colony of prairie dogs from 1979 to 1988 was 74.1 (harmonic mean).

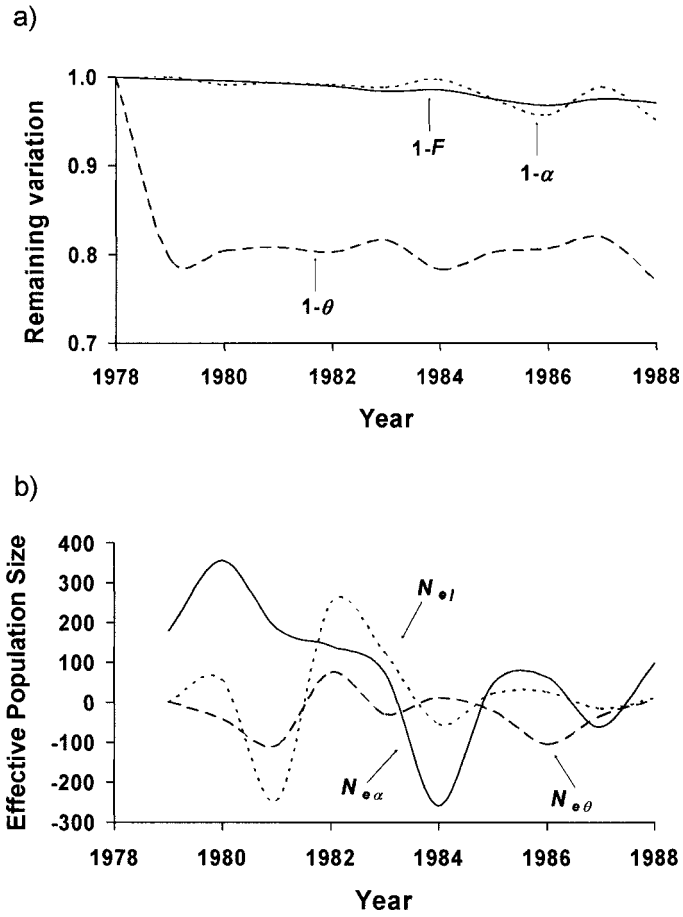

FIG. 1.—a) Pedigree estimates of the proportion of genetic variation that remained in the colony of prairie dogs in each year of the study (1979–1988, assumed to be 1.0 in 1978), at different levels of population structure: within individuals ($1 - F$), among individuals within coteries ($1 - \theta$), and among coteries within the colony ($1 - \alpha$). b) Pedigree estimates of effective population size are from equation (7), using the gene correlations F, θ, and α.

DISCUSSION

Estimating N_e.—The genetic structure of populations can have significant and substantial effects on the rate of loss of genetic diversity and thus on N_e (Crow and Kimura 1970; Wright 1969). Social groupings create genetic structure within populations of many social species, for example social mammals (Pope 1992, 1998; Sugg et al. 1996; Wood 1987). Population-genetics theory has been developed for social breeding groups (Chesser 1991a, 1991b; Chesser et al. 1993; Sugg and Chesser 1994), but this theory needs to be extended to real populations in nature (Dobson 1998). Thus, we asked whether our colony of highly social black-tailed prairie dogs actually exhibited internal genetic structure via the coterie breeding groups. The F-statistics indicated highly significant genetic structuring within the colony: the highly positive F_{LS} values indicated that coteries exhibited substantial genetic differentiation among coteries (at about 17%) even though the near-zero values of F_{IS} indicated that mating within the colony could not be distinguished from random (Table 1). Significant genetic structure was also evident in analyses that were restricted to adult females (Dobson et al. 1998).

Temporal changes in correlations of genes may indicate shifts in genetic variation from one level of genetic structure to another. For example, changes in F from one year to the next reflected temporal differences in the frequency of matings between different classes of kin. Temporal changes in inbreeding might influence the loss of genetic diversity through

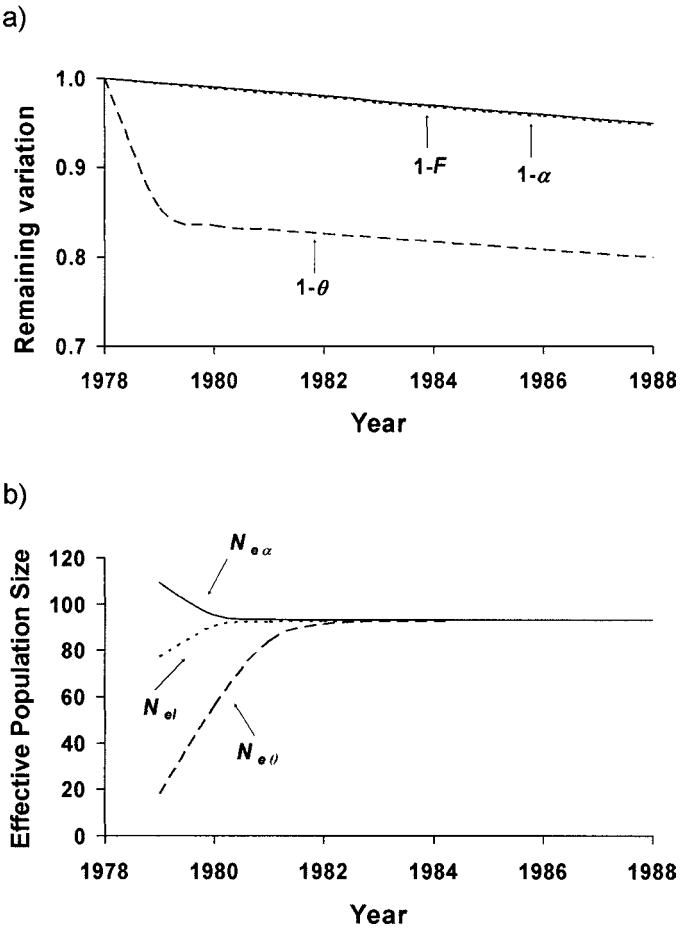

FIG. 2.—a) Breeding-group model estimates of the proportion of genetic variation remaining in the colony of prairie dogs in each year of the study (1979–1988, assumed to be 1.0 in 1978), at different levels of population structure: within individuals $(1 - F)$, among individuals within coteries $(1 - \theta)$, and among coteries within the colony $(1 - \alpha)$. b) Model estimates of effective population sizes are from equation (7), using changes in the gene correlations F, θ, and α.

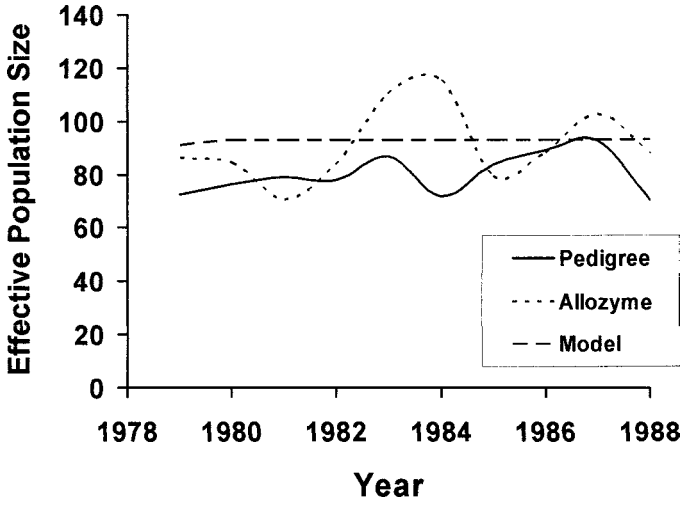

FIG. 3.—Predicted asymptotic effective population size of the colony of prairie dogs from the breeding-group model, and estimates of effective population sizes from pedigrees and allozyme data for 1979 to 1988.

drift, but such diversity could be conserved at other levels of population structure (Chesser et al. 1996). Coteries varied temporally in their genetic diversity, as reflected by annual changes in θ (Fig. 1a). Also, α reflected changes in genetic diversity that occurred among the coteries. Only losses of genetic diversity at the highest (α) level reflect genetic variation that is truly lost within the colony, since losses within individuals and coteries only influence parts of the total colony.

Changes in genetic correlations over time can be used to estimate instantaneous effective population size N_e using equation (7), but the results need to be evaluated carefully because a wide range of values may occur (Fig. 1b). In fact, N_e can become negative because genetic correlations can decrease over time at a given level of structure (i.e., individual, coterie, or colony). For example, from 1983 to 1984, a very small amount of genetic variation was gained among coteries within the colony of prairie dogs (Fig. 1a), resulting in a slightly negative change in the genetic correlation α (coteries were slightly less similar and thus less correlated, reflected by a decrease in α from 0.017 to 0.015). As a result, $N_{e\alpha}$ was strongly negative (Fig. 1b). Inspection of equation (7) reveals that the smallest changes in genetic correlations yield the largest positive or negative estimates of N_e. Such estimates are obviously not useful for predicting future loss of genetic variation (though this method has been used; Blackwell et al. 1995). Instead, instantaneous N_e reflects the shifting of genetic diversity back and forth between different levels of population substructure that occurs when the population is not in demographic or genetic equilibrium. Large changes in instantaneous N_es might be produced by immigration, but this was minor in our study (see below).

Estimates of fixation indices and N_es from our ecological "breeding-group" model were based on an assumption of genetic equilibrium and some of the assumptions of an ideal population. Thus, the loss of genetic variation was constant once genetic equilibrium was reached in the model (Fig. 2a). Estimates of N_e from different genetic correlations converge on the same asymptotic value under equilibrial conditions (Fig. 2b; Chesser et al. 1993; Sugg and Chesser 1994). Once equilibrium is achieved, shifts of genetic variation among levels of population substructure occur at a constant rate, allowing instantaneous calculations of N_e from the genetic correlations. Pedigree data indicated that the population was not in genetic equilibrium (Fig. 1), but only approximated it (compare Fig. 1a and 2a). Thus, the model produced more consistent instantaneous estimates of N_e than did the pedigree data (compare Fig. 1b and Fig. 2b).

Shifts in genetic variation among levels of genetic substructure influence the rate at which genetic diversity is lost from the population. If subunits (breeding groups or subpopulations) of a finite population are isolated, then they might become fixed for alternative alleles. But because several different subunits likely become fixed for alternative alleles, the total genetic variation in the population may be conserved at the α level of structure (Chesser et al. 1996). If migration was promoted among subunits, variation could shift to within

TABLE 1.—Values of *F*-statistics for 1979–1988 from the breeding–group model, pedigrees, and allozyme data for black-tailed prairie dogs. Ranges for values of *F*-statistics calculated separately for each of the 10 years are presented to indicate temporal variation.

	Pedigree		Model	Allozyme	
F-statistic	Mean	Range	Value	Mean	Range
F_{IL}	−0.223	−0.267 to −0.179	−0.184	−0.213	−0.249 to −0.167
F_{IS}	0.000	−0.013 to −0.020	0.002	−0.012	−0.089 to −0.032
F_{LS}	0.186	0.160 to −0.209	0.157	0.166[a]	0.128 to −0.209

[a] Pooled χ^2 tests from allozyme loci indicated highly significant F_{LS} ($P < 0.001$) in all years.

breeding groups (θ level) and within individuals (*F* level). The risk of losing genetic variation from the total population through genetic drift, however, would increase. Conservation biologists would like to minimize the rate of loss of genetic diversity in natural populations (e.g., declines apparent in Figs. 1a and 2a) and to maximize genetic variation within individuals and subpopulations. The interplay between conflicting goals of conserving genetic diversity in both individuals and populations creates a paradox for genetic conservationists (Chesser et al. 1996).

The dynamics of social breeding groups may provide partial resolution of the paradox. Estimates of N_e for prairie dogs were consistent (Fig. 3, and see below) and just above the mean number of adults in the colony, indicating a lower rate of loss of genetic diversity than we would expect in an ideal population of the same size as the real population. Thus, social breeding groups may have conserved genetic diversity (Chesser et al. 1993, 1996). The polygynous mating system and female philopatry of prairie dogs results in strong genetic differentiation of coteries (high θ and low α), which has a conserving influence on genetic diversity because different alleles may come to predominate in different coteries. Complete male dispersal, however, results in breeding conditions that differ little from random mating within the colony, so that fairly low rates of inbreeding occur (Dobson et al. 1997, 1998; Hoogland 1992, 1995).

Evaluation of methods.—Methods for estimating N_e that do not include social substructuring of populations cannot lead to the conclusion that social behavior and reproduction in breeding groups result in conservation of genetic diversity. Previous models have provided estimates of N_e under conditions of unequal contributions of the sexes during mating, non-random mating within an unstructured population, and various forms of polygyny (Caballero and Hill 1992; Crow and Denniston 1988; Nunney 1993). When population substructure is lacking or unrecognized, however, estimates of N_e for polygynous species may vary from a small fraction of the breeding population (Frankham 1995) to about half the number of adult individuals (Nunney 1993). Thus, it is important to recognize and account for social breeding groups when estimating N_e in highly social species (Chesser et al. 1993; Sugg and Chesser 1994; Sugg et al. 1996), or gross overestimates of the rate of loss of genetic diversity may result.

Three problems may have biased our estimates of N_e. First, the influence of overlapping generations might cause N_e to be underestimated (Nunney 1999; Vucetich et al. 1997). Not incorporating overlap of generations did not appear to greatly bias N_e estimated from the breeding-group model. Our estimates of gene correlations from pedigree and allozyme data incorporated the real overlapping of generations that occurred in the prairie dog colony, and of estimates of N_e from these data were similar to model estimates of N_e using alternative methods (equations 7 and 9). The specific influences of overlapping generations on estimates of N_e, however, are unknown. Second, the breeding-group model assumed that immigration did not occur, and such immigration could inflate estimates of N_e. Again, estimates of N_e from both the pedigree and allozyme data incorporated the real immigrants that moved into the prairie dog colony (and thus could have been biased upwards; but see below), but the estimates from the breeding-group model fell within the range of values of empirical estimates. The number of immigrants that bred in the colony (12 males and 4 females during the 10 years from 1979 to 1988; Hoogland 1995) was much lower than the number of colony residents and thus had little effect on the mean genetic correlations that were estimated from pedigrees or heterozygosity estimates from allozyme data. Third, variation in population size over long periods of time can result in substantial lowering of estimates of N_e (Vucetich et al. 1997; Vucetich and Waite 1988). The prairie dogs exhibited variation in population size over the study, and we used the harmonic mean for estimating population size of potentially breeding adults. Estimates of mean population size over longer periods of time might be lower, but our conclusion of N_e close to the harmonic mean number of potentially breeding adults would still hold, since the latter estimate would also decline.

Our study used more detailed data than Sugg et al. (1996; data from Hoogland 1995) and our estimates of N_e and mean census population size were lower. The relationship of our estimates of N_e to adult population size, however, remained about the same (at 1.07–1.24 times census size, compared to 1.12 in the earlier study). Nunney (1999) suggested biases that may occur in the breeding-group model due to the mode of population regulation (local as opposed to population-wide). The model of Chesser et al. (1993) and Sugg and Chesser (1994) assumes that population regulation is local (viz., within breeding groups, or coteries for prairie dogs). In fact, the lack of dispersal of females (estimated at 2%) among coteries suggests local regulation of population size (contra Nunney 1999). However, measurement of the mode of population regulation ("x" factor—Nunney 1999) is currently undefined, making it difficult to quantify the mode of population regulation and test for its importance.

Further cautions in application of these different methods are needed. For example, temporal changes in genetic correlations of offspring from pedigrees, which should yield extremely accurate estimates of N_es, yielded values that fluctuated from year to year and even became negative (Fig. 1b). This resulted from the use of instantaneous estimates that reflected small non-equilibrial changes in genetic diversity among levels of population structure. In addition to the tendency of genetic correlations to increase over time (Figs. 1a and 2a), additional

small positive or negative changes in genetic correlations may have reflected sampling effects associated with mating or with survival of offspring that produced shifts of genetic diversity among levels of population structure. Asymptotic gene dynamics, when applied to empirical information from pedigrees or biochemical data (Fig. 3), yielded predictions of N_e that should prove useful for conservation practice. Both instantaneous and asymptotic estimates of gene dynamics from the breeding-group model seemed reasonably close to empirical estimates (compare Fig. 2b and Fig. 3).

When pedigree or allozyme data were used to predict asymptotic changes in genetic diversity of prairie dogs, estimates of N_e varied among years (Fig. 3). Both techniques predicted the rate of loss of genetic diversity for specific sampling patterns of genes during breeding so were subject to interannual changes in demography. Thus, in some years, either method might yield an estimate of predicted asymptotic N_e that was slightly lower or much higher than the number of adults in the colony. Accurate estimation of the long-term N_e might therefore require averaging many years of data (using the harmonic mean—Wright 1969). The breeding-group model incorporated salient aspects of dispersal and mating patterns, averaged over several years, to produce an asymptotic estimate of N_e (Fig. 3). If data were limited, however, parameters for the breeding-group model might be taken from studies conducted at different times or localities. Thus, the breeding-group model has the advantage of being extremely flexible for making preliminary predictions of N_e.

We estimated N_e only within our study colony. Other colonies of prairie dogs exist in the local region and, although restricted, gene flow occurs among colonies (Daley 1992; Foltz and Hoogland 1983; Garrett and Franklin 1988; Hoogland 1995). Therefore levels of population structure (e.g., for the region) exist that were not taken into account in our study. Although immigrants from outside the colony were included in the pedigree and allozyme estimates of N_e, the influence of immigrants was minor compared with changes in genetic diversity among levels of colony substructure. However, breeding-group model estimates of N_e that took into account both local breeding groups and the local regional population structure, including dispersal among colonies, might yield higher asymptotic values. Nevertheless, the total genetic diversity partitioned within and among multiple levels of the regional population would be expected to increase, complicating the estimation of N_e.

Our consideration of population substructure and its influence on the loss of genetic diversity was applied to a species that exhibited considerable genetic structuring of populations from social breeding groups. Sugg et al. (1996) showed that translocations of females (effectively increasing the female dispersal rate) within a colony would result in more rapid loss of genetic diversity, because the influence of the genetic structure of the coterie breeding groups would be disrupted. Translocation of females could actually increase the rate of inbreeding and loss of genetic variation by bringing closely related males and females into spatial proximity, followed by breeding between them. Translocation of females might also increase the loss of genetic variation in populations of plateau pikas (*Ochotona curzoniae*), which also exhibit high N_e due to social family groups (Dobson et al. 2000).

The breeding-group model appears to be appropriate for estimating F-statistics and N_e in polygynous species that exhibit sex-biased dispersal, but the models should not be applied in their current form to other mating and dispersal systems (Basset et al. 2001). Nonetheless, many species of mammals, notably primates and rodents, have social groups that exhibit female philopatry and polygynous mating (Dobson 1982; Greenwood 1980). The breeding-group model may therefore prove useful for estimating N_e in species that exhibit genetic structure that is associated with social groups.

Acknowledgments

Our greatest debts are to the 112 field assistants who worked with JLH at the study colony and the staff at Wind Cave National Park. We thank J. F. Crow, L. Nunney, T. S. Risch, M. J. Wade, and M. Winterrowd for helpful comments on the manuscript. Financial support was provided by an Oak Ridge Institute for Science and Education Fellowship and Visiting Faculty Fellowships at the Savannah River Ecology Laboratory (FSD); contract DE-FC09-96SR18546 between the United States Department of Energy and the University of Georgia (RKC and DWS); the National Science Foundation, the National Geographic Society, the American Philosophical Society, the Center for Field Research, the Eppley Foundation for Research, the Universities of Maryland, Michigan, and Minnesota, Princeton University, and the Harry Frank Guggenheim Foundation (JLH); and the National Institutes of Health and the National Science Foundation (DWF). Field work was completed in 1989, but all procedures conformed to the ASM guidelines for the capture, handling, and care of mammals (Animal Care and Use Committee 1998).

Literature Cited

Animal Care and Use Committee. 1998. Guidelines for the capture, handling, and care of mammals as approved by the American Society of Mammalogists. Journal of Mammalogy 79:1416–1431.

Blackwell, B. F., P. D. Doerr, and J. M. Reed, J. R. Walters. 1995. Inbreeding rate and effective population size: a comparison of estimates from pedigree analysis and a demographic model. Biological Conservation 71:299–304.

Basset, P., F. Balloux, N. Perrin. 2001. Testing demographic models of effective population size. Proceedings of the Royal Society of London B268:311–317.

Caballero, A., W. G. Hill. 1992. Effective size of nonrandom mating populations. Genetics 130:909–916.

Chesser, R. K. 1991a. Influence of gene flow and breeding tactics on gene diversity within populations. Genetics 129:573–583.

Chesser, R. K. 1991b. Gene diversity and female philopatry. Genetics 127:437–447.

Chesser, R. K. 1998. Heteroplasmy and organelle gene dynamics. Genetics 150:1309–1327.

Chesser, R. K., R. J. Baker. 1996. Effective sizes and dynamics of uniparentally and diparentally inherited genes. Genetics 144:1225–1235.

Chesser, R. K., and O. E. Rhodes, Jr., M. H. Smith. 1996. Conservation. Pp. 237–252 in Population processes in ecological space

and time (O. E. Rhodes, Jr., R. K. Chesser, and M. H. Smith, eds.). University of Chicago Press, Chicago, Illinois.

Chesser, R. K., O. E. Rhodes, Jr., D. W. Sugg, and A. Schnabel. 1993. Effective sizes for subdivided populations. Genetics 135:1221–1232.

Cockerham, C. C. 1967. Group inbreeding and coancestry. Genetics 56:89–104.

Cockerham, C. C. 1969. Variance of gene frequencies. Evolution 23:72–84.

Cockerham, C. C. 1973. Analysis of gene frequencies. Genetics 74:679–700.

Crow, J. F., and C. Denniston. 1988. Inbreeding and variance effective population numbers. Evolution 42:482–495.

Crow, J. F., and M. Kimura. 1970. An introduction to population genetics theory. Harper and Row, New York.

Daley, J. G. 1992. Population reductions and genetic variability in black-tailed prairie dogs. Journal of Wildlife Management 56:212–220.

Dobson, F. S. 1982. Competition for mates and predominant juvenile male dispersal in mammals. Animal Behaviour 30:1183–1192.

Dobson, F. S. 1998. Social structure and gene dynamics in mammals. Journal of Mammalogy 79:667–670.

Dobson, F. S., R. K. Chesser, J. L. Hoogland, D. W. Sugg, and D. W. Foltz. 1997. Do black-tailed prairie dogs minimize inbreeding? Evolution 51:970–978.

Dobson, F. S., R. K. Chesser, J. L. Hoogland, D. W. Sugg, and D. W. Foltz. 1998. Breeding groups and gene dynamics in a socially-structured population of prairie dogs. Journal of Mammalogy 79:671–680.

Dobson, F.S., A.T. Smith, and Wang X.G. 2000. The mating system and gene dynamics of plateau pikas. Behavioural Processes 51:101–110.

Falconer, D. S. 1989. Introduction to quantitative genetics, 3rd ed. Longman Group Limited, Essex, United Kingdom.

Foltz, D. W., and J. L. Hoogland. 1981. Analysis of the mating system in the black-tailed prairie dog (*Cynomys ludovicianus*) by likelihood of paternity. Journal of Mammalogy 62:706–712.

Foltz, D. W., and J. L. Hoogland. 1983. Genetic evidence of outbreeding in the black-tailed prairie dog (*Cynomys ludovicianus*). Evolution 37:273–281.

Frankham, R. 1995. Effective population size/adult population size ratios in wildlife: a review. Genetical Research 66:95–107.

Garrett, M. G., and W. L. Franklin. 1988. Behavioral ecology of dispersal in the black-tailed prairie dog. Journal of Mammalogy 69:236–250.

Greenwood, P. J. 1980. Mating systems, philopatry, and dispersal in birds and mammals. Animal Behaviour 28:1140–1162.

Harris, H., and D. A. Hopkinson. 1976. Handbook of enzyme electrophoresis in human genetics. American Elsevier, New York.

Hill, W. G. 1979. A note on effective population size with overlapping generations. Genetics 92:317–322.

Hoogland, J. L. 1985. Infanticide in prairie dogs: lactating females kill offspring of close kin. Science 230:1037–1040.

Hoogland, J. L. 1992. Levels of inbreeding among prairie dogs. American Naturalist 139:591–602.

Hoogland, J. L. 1995. The black-tailed prairie dog. University of Chicago Press, Chicago, Illinois.

Hoogland, J. L., and D. W. Foltz. 1982. Variance in male and female reproductive success in a harem-polygynous mammal, the black-tailed prairie dog (Sciuridae: *Cynomys ludovicianus*). Behavioural Ecology and Sociobiology 11:155–163.

King, J. A. 1955. Social behavior, social organization, and population dynamics in a black-tailed prairie dog town in the Black Hills of South Dakota. Contributions from the Laboratory of Vertebrate Biology, University of Michigan 67:1–123.

Long, J. C., F. C. Romero, M. Urbanek, and D. Goldman. 1998. Mating patterns and gene dynamics of an American Indian population isolate. Journal of Mammalogy 79:681–691.

Nei, M. 1977. *F*-statistics and analysis of gene diversity in subdivided populations. Annals of Human Genetics 41:225–233.

Nei, M., and R. K. Chesser. 1983. Estimation of fixation indices and gene diversities. Annals of Human Genetics 47:253–259.

Nunney, L. 1993. The influence of mating system and overlapping generations on effective population size. Evolution 47:1329–1341.

Nunney, L. 1999. The effective size of a hierarchically structured population. Evolution 53:1–10.

Nunney, L., and D. R. Elam. 1994. Estimating the effective population size of conserved populations. Conservation Biology 8:175–184.

Pope, T. R. 1992. The influence of dispersal patterns and mating system on genetic differentiation within and between populations of the red howler monkey (*Alouatta seniculus*). Evolution 46:1112–1128.

Pope, T. R. 1998. Effects of demographic change on group kin structure and gene dynamics of red howling monkey populations. Journal of Mammalogy 79:692–712.

Selander, R. K., M. H. Smith, S. Y. Yang, W. E. Johnson, and J. B. Gentry. 1971. Biochemical polymorphism and systematics in the genus *Peromyscus*. I. Variation in the old-field mouse (*Peromyscus polionotus*). University of Texas Publications 7103:49–90.

Spielman, R. S., J. V. Neel, and F. H. F. Li. 1977. Inbreeding estimation from population data: models, procedures and implications. Genetics 85:355–371.

Sugg, D. W., and R. K. Chesser. 1994. Effective population sizes with multiple paternity. Genetics 137:1147–1155.

Sugg, D. W., R. K. Chesser, F. S. Dobson, and J. L. Hoogland. 1996. Population genetics meets behavioral ecology. Trends in Ecology and Evolution 11:338–342.

Vucetich, J. A., and T. A. Waite. 1998. Number of censuses required for demographic estimation of effective population size. Conservation Biology 12:1023–1030.

Vucetich, J. A., T. A. Waite, and L. Nunney. 1997. Fluctuating population size and the ratio of effective to census population size. Evolution 51:2017–2021.

Wang, J. 1997. Effective size and *F*-statistics of subdivided populations. II. Dioecious species. Genetics 146:1465–1474.

Waser, P.M., and W. T. Jones. 1983. Natal philopatry among solitary mammals. Quarterly Review of Biology 58:355–390.

Weir, B. S., and C. C. Cockerham. 1984. Estimating *F*-statistics for the analysis of population structure. Evolution 38:1358–1370.

Wood, J. W. 1987. The genetic demography of the Gainj of Papua New Guinea. 2. Determinants of effective population size. American Naturalist 129:165–187.

Wright, S. 1965. The interpretation of population structure by *F*-statistics with special regard to systems of mating. Evolution 19:395–420.

Wright, S. 1969. Evolution and the genetics of populations, Vol. 2: The theory of gene frequencies. University of Chicago Press, Chicago, Illinois.

Wright, S. 1978. Evolution and the genetics of populations, Vol. 4: Variability within and among natural populations. University of Chicago Press, Chicago, Illinois.

Submitted 15 April 2002. Accepted 21 February 2003.

Associate Editor was Edward H. Miller.