Effective Sizes and Dynamics of Uniparentally and Diparentally Inherited Genes

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ABSTRACT

Models to determine the temporal dynamics and spatial heterogeneity for maternally and paternally inherited genes were derived for populations that may or may not exhibit spatial subdivision. Results were compared to those for diparentally inherited genes. The models permit definition of parameters for mean and variance of litter sizes, breeding group (subpopulation) sizes, and numbers of female mates per male, dispersal rates, and multiple paternity. Exact solutions for asymptotic effective size and spatial divergence (F_{LS}) for maternal and paternal genes are derived. It is shown that solutions for effective size and F_{LS} are transformations of the same quadratic equation. When compared to values for diparentally inherited genes, it is shown that effective sizes for maternal genes may be considerably higher when female dispersal is low as in many mammalian taxa. Likewise, effective sizes for paternal genes may be higher than for diparentally inherited traits when male dispersal is relatively low, as in many species of birds. The traditional assumption that the effective size for maternal genes is approximately equal to the number of females is seldom realized. Spatial heterogeneity and temporal dynamics of genes are inextricably linked as is shown by the interdependency of effective size and spatial heterogeneity.

E VOLUTION proceeds only by changes in gene frequency and combinations of genes. Describing the dynamics of gene diversity in mathematical models provides methods by which predictions can be made and probability can be assigned not only to the mode and tempo of evolution, but to solve applied issues relative to conservation of genetic variation. This paper expands the mathematical models necessary to explain the dynamics of genetic variance within and among natural populations for maternally, paternally, and diparentally inherited genes.

Methods available for investigators to assess genetic variation have expanded considerably since the advent of starch-gel electrophoresis (AVISE 1994; BAKER 1994). Molecular techniques available today offer greater precision for the assessment of genetic variation than phenotypic comparisons of gene products or isozymes. Concomitant with greater precision have been developments of methods to determine the genetic patterns for organelles and gene segments associated with maternal and paternal inheritance. Studies using mitochondrial DNA (mtDNA) have been vital in detailing evolutionary patterns and geographic structure from the standpoint of maternally inherited genes (AVISE and LANSMAN 1983; AVISE *et al.* 1984; MORITZ *et al.* 1987). While techniques to identify paternally inherited genes have been less well developed (MAYNARD SMITH 1990), the potential for such analyses have been demonstrated and will likely be refined in coming years (CASANOVA *et al.*

1985; ELLIS *et al.* 1990; RASHEED *et al.* 1991; WHISENANT *et al.* 1991).

There have been great strides in development of techniques to document maternal and paternal contributions to genetic variation and theory to assess gene dynamics and spatial patterns from knowledge of dispersal and mating patterns (NEIGEL *et al.* 1991). It is interesting to note that much of the application of data for mtDNA and nuclear markers, when applied within a species, has been to estimate gene flow and differentiation among geographically separated populations (AVISE 1994). The near ubiquity of spatial variation among populations has been demonstrated for all levels of population organization as well as for diparental, maternal, and paternal genes (SMITH *et al.* 1978; BAKER *et al.* 1989; RABENOLD *et al.* 1990, 1991; AVISE *et al.* 1992; TUCKER *et al.* 1992). However, estimates of temporal gene dynamics from measures of effective population size have traditionally not accounted for spatial variation. BIRKY *et al.* (1983, 1989) explored the differentiation of organelle and nuclear genes in considerable detail. In both investigations, however, they maintain that the effective size for organelle genes is approximately equal to the number of breeding females and the population was treated as essentially "ideal" in that emigration, immigration, and mating tactics other than random were not considered in the calculation of effective size. Models for diploid, sexually transmitted nuclear genes have shown that spatial differentiation among population subdivisions may have a profound effect upon the temporal rate of loss of gene diversity

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(CHESSEY 1991a,b; CABALLERO and HILL 1992a,b; CHESSEY *et al.* 1993a; SUGG and CHESSEY 1994). It is likely that similar effects will be observed for uniparentally inherited genes as well. Clearly, because spatial heterogeneity is a prevalent phenomenon in natural and managed populations, estimates of gene dynamics must take such structure into consideration.

A full appreciation of the geographic population structure of a species requires an integration of evolutionary (genetic) and contemporary (behavioral) perspectives (AVISE *et al.* 1992; AVISE 1994). The complex interaction of behavior and genetic diversity has been recently evaluated in detail with regards to social organization, population subdivision and dispersal patterns (CHESSEY 1991a,b; CHESSEY *et al.* 1993a; SUGG and CHESSEY 1994), and nonrandom mating (CABALLERO and HILL 1992). These investigations documented that the temporal dynamics of genetic variance is inextricably linked to the apportionment of gene diversity across geographic space. The nonindependence of spatial and temporal change of genes has profound ramifications for understanding evolutionary biology (CHESSEY *et al.* 1993b) and for predicting the fate of genetic variance in natural and managed populations of organisms (see CHESSEY *et al.* 1996).

Mutation rates for some organelle genes, such as mtDNA, have been estimated to be five to 10 times greater than those for nuclear genes (BROWN *et al.* 1979). Such high mutation rates have, in part, been speculated to be responsible for the high variation of organelle genotypes in natural populations (MELNICK and HOELZER 1992). However, for the relative importance of mutation and random loss of variation to be accurately assessed, we must understand the parameters affecting the dynamics of diparentally and uniparentally inherited genes in biologically meaningful settings. If, in fact, gene diversity for uniparentally inherited traits is always lost at a faster rate than for diparentally inherited genes (*cf.* BIRKY *et al.* 1983, 1989), then mutation rates may play a pivotal role in maintaining variation. On the other hand, if loss of variation for uniparentally inherited genes can be slower than for diparental genes under certain scenarios, then we may gain new insights regarding parameters influencing gene diversity at various levels of organization.

Effective population size can be operationally defined as the hypothetical number of breeders in an ideal population that will lose genetic variation at the same rate as those in a population under consideration (WRIGHT 1969). An ideal population has the following characteristics (*cf.* HARTL 1980): (1) diploid, sexually reproducing organisms, (2) nonoverlapping generations, (3) independent subpopulations, each of constant size, (4) random mating within each subpopulation with equal numbers of males and females (or monogamous) and population of constant size, (5) no migration between subpopulations, (6) no mutation,

and (7) no selection. The objective of models to estimate effective population size is to provide realistic probabilities of genetic change within the population when one or more of these assumptions has been violated. The purpose of this study is to provide general methods to estimate the temporal dynamics of maternally and paternally inherited genes and the genetic differentiation that would result when there is spatial structure. Specifically we will provide methods to relax the assumptions of diploidy, independent subpopulations of constant size, random mating, isolation of subpopulations, and absence of mutation to predict spatial and temporal dynamics of gene frequencies. Methods will be developed in a general fashion to apply equally to populations subdivided by social structure, by geographic structure, or exhibiting no spatial structure whatsoever. Comparisons of patterns and rates of change of genetic variation for uniparentally inherited genes and those inherited from both sexes will be made.

The models employed herein will assume complete uniparental inheritance, except where otherwise noted, and homoplasmy within individuals. The topics of non-equal contributions of organelles from male and female parents, as well as heteroplasmy, present complications that are beyond the scope of this paper and will be addressed in a separate manuscript. The near ubiquity of homoplasmy for organelles in higher vertebrates (ATTARDI 1985; AVISE 1991) ensures that the results presented will be applicable to a broad diversity of taxa. Methods of development of models will be essentially numeric, but implicit analytical solutions will be provided when possible.

DEVELOPMENT OF THE MODELS

Transition equations for uniparentally inherited genes: The methods for determining the transition correlations of genes inherited from a single sex within and among breeding groups within a subpopulation will be similar to those of CHESSEY (1991a,b) and CHESSEY *et al.* (1993a) for diploid, segregating genes. Initially, however, we need only to derive the correlation of genes transmitted by a single parent within breeding groups (coancestry, θ) and the correlation of such genes among breeding groups (α). Obviously, inbreeding (F) is not a meaningful quantity for maternally or paternally transmitted genes when there is homoplasmy because the correlation of genes within individuals for these traits is always unity. The proportion of the original genetic variance remaining within breeding groups and for the entire population is $1 - \theta$ and $1 - \alpha$, respectively (COCKERHAM 1969, 1973; CHESSEY 1991a). Transition equations for genes transmitted solely by females or by males are very similar and will be expressed in general terms applicable to either contributing sex.

In a breeding group, the expected proportion of the progeny that share the same mother (multiple pater-

nity is not important in this case; thus, the progeny of a mother may or may not be full siblings) is (see Equations 3–5 of CHESSER *et al.* 1993a)

$$E(\text{sibs}) = \psi_f = \frac{\frac{1}{2} \sum_{i=1}^n k_i^2 - k_i}{kn(kn-1)/2} = \frac{\sigma_k^2 + k(k-1)}{k(kn-1)}, \quad (1)$$

where k is the average number of progeny per female and n is the average number of breeding females per breeding group. The probability that a random pair of females within the same breeding group were mated by the same male was defined by CHESSER *et al.* (1993a; SUGG and CHESSER 1994) as

$$\phi_m = \frac{m \left(\sigma_b^2 + b(b-1) \right)}{nl(n-1)}, \quad (2a)$$

where m is the number of males per group, b and σ_b^2 are the mean and variance for the number of female mates per male, and l is the mean number of male mates per female (for single paternity, $l = 1$). If we assume that $m = n$ but that not all males may mate (primary sex ratio is 1:1 but breeding sex ratio may differ), then $b = n/m = 1$ and Equation 2a becomes

$$\phi_m = \frac{\sigma_b^2}{l(n-1)}. \quad (2b)$$

The parameter σ_b^2 is a measure of the variance in male mating success and must be calculated over all *potential* male mates (CHESSER *et al.* 1993a). The value of ϕ_m will be one when a single male mates with all n females within a group, and zero when each female mates with a unique male. Multiple paternity, wherein more than one male may contribute to a female's progeny ($l > 1$), requires including an additional parameter, ϕ_w , for the probability that two randomly chosen progeny within a single brood are the product of the same sire

$$\phi_w = \frac{\sigma_p^2 + p(p-1)}{p(k-1)}, \quad (3)$$

where p and σ_p^2 are the mean and variance of the number of progeny per brood sired by a single male (SUGG and CHESSER 1994). The value of ϕ_w will range between 0, indicating that all progeny within a brood are products of different males, and one, for single paternity. As in maternally inherited genes, the probability that random pairs of progeny born within a breeding group were sired by the same father is

$$\begin{aligned} \psi_m &= \frac{m[\sigma_{k(m)}^2 + k_m(k_m-1)]}{kn(kn-1)} \\ &= \phi_w \psi_f + (1 - \phi_w \psi_f) \phi_m, \end{aligned} \quad (4)$$

with k_m and $\sigma_{k(m)}^2$ referencing the mean and variance in progeny produced by males. Equation 4 suffices for multiple or single paternity depending on the value

of ϕ_w . Obviously, when there is single paternity ($\phi_w = 1$), then $\psi_m = \phi_f + (1 - \phi_f) \phi_m$ (e.g., CHESSER *et al.* 1993a). Below we refer to the parameter ψ , where the dot denotes either the female (ψ_f) or male (ψ_m) parent. Similar notation will be applied to other parameters and variables to reference values for the contributing sex.

If there are s breeding groups that may be variable in size, then the probability that two randomly selected individuals are from the same group is

$$\eta = \frac{\frac{1}{2} \sum_{i=1}^s n_i^2 - n_i}{ns(ns-1)/2} = \frac{\sigma_n^2 + n(n-1)}{n(ns-1)} \quad (5)$$

where σ_n^2 denotes the variance in group size in terms of females (or males, since $n = m$). If sex ratios differ markedly from 1:1 and group sizes are very small, then values of η should be derived for sexes separately; such an approach does not alter equations for uniparentally inherited genes (substituting η for η) but complicates subsequent expressions for diparental genes. We have chosen to use a single value of η . Therefore, a migrant from a breeding group is related to η of the population by the coancestry within breeding groups (θ) and to $1 - \eta$ by the correlation of genes among groups (α). Because we are considering that only one parent may propagate the genes to the progeny, all siblings (ψ , proportion of a breeding group) will share the same gene identical by descent with the contributing parent. The remainder of progeny born within a breeding group ($1 - \psi$) may be related by common descent, depending upon dispersal into and out of the natal group. Of these, $1 - (1 - \eta)d$, with d denoting the rate of dispersal by either females (d_f) or males (d_m) from their native group will be progeny born by natives of the same breeding group and the correlation of their genes is θ^* . The frequency of individuals coming together within a group that are native to other groups is $(1 - \eta)d$ and are related by α^* . Thus, the transition of coancestry from generation t to $t + 1$ is (hereafter superscripts on θ and α will be omitted and these variables will pertain only to the contributing sex)

$$\begin{aligned} \theta_{t+1} &= \psi + (1 - \psi)(1 - (1 - \eta)d)\theta_t \\ &\quad + (1 - \psi)(1 - \eta)d\alpha_t. \end{aligned} \quad (6)$$

The correlation of parental genes among breeding groups within the subpopulation is then

$$\alpha_{t+1} = \eta d \theta_t + (1 - \eta d) \alpha_t. \quad (7)$$

The transition matrix **T**, applicable to either contributing parent can be represented as (for θ , α)

$$\mathbf{T} = \begin{bmatrix} (1 - \psi)(1 - (1 - \eta)d) & (1 - \psi)(1 - \eta)d \\ \eta d & 1 - \eta d \end{bmatrix}. \quad (8)$$

A constant (ψ_*) is present in the transition for θ (Equation 6), and thus a constant vector $\mathbf{C}_*$ is required

$$\mathbf{C}_* = \begin{bmatrix} \psi_* \\ 0 \end{bmatrix}, \quad (9)$$

such that $\mathbf{T}_i \{\theta_i, \alpha_i\} + \mathbf{C}_* = \{\theta_{i+1}, \alpha_{i+1}\}$. The matrix multiplication and addition sequence can be iterated over many generations to determine the values of θ and α for successive time periods (using initial values of zero). The results for each generation provide the gene correlations necessary to determine effective sizes and spatial heterogeneity.

To determine the effective size of a population in terms of a uniparentally inherited gene, the rate of change of the variance of the gene must be calculated. The relative change in intergroup correlation, α , over generations can be readily defined using Equation 7

$$\begin{aligned} \Delta\alpha &= \frac{\alpha_{i+1} - \alpha_i}{1 - \alpha_i} = \frac{\eta d_i \theta_i + (1 - \eta d_i) \alpha_i - \alpha_i}{1 - \alpha_i} \\ &= \frac{\eta d_i (\theta_i - \alpha_i)}{1 - \alpha_i}. \end{aligned} \quad (10)$$

Because the proportion of variance among groups is defined as $F_{LS} = (\theta - \alpha) / (1 - \alpha)$, (COCKERHAM 1973; CHESSER 1991a,b) Equation 10 can be summarized as

$$\Delta\alpha = \eta d_i F_{LS}^*, \quad (11)$$

and because the effective size for a uniparentally inherited gene is $1/\Delta\alpha$ [note that this is not $1/(2\Delta\alpha)$ as would be for a diploid, segregating gene], the intergroup effective size (CHESSER *et al.* 1993a), N_{α} for maternal or parental genes is

$$N_{\alpha}^* = \frac{1}{\eta d_i F_{LS}^*}. \quad (12)$$

Obviously, Equation 9 can be rearranged to obtain the effective amount of gene flow among lineages as

$$d_i \eta N_{\alpha}^* = \frac{1}{F_{LS}^*}, \quad (13)$$

which estimates the effective number of dispersers per group. The total number of dispersers is estimated by $d_i N_{\alpha}^* = 1/(\eta F_{LS}^*)$, which is similar to expressions for N_m (SLATKIN and BARTON 1989; AVISE 1994).

The change in the coancestry can be derived in a similar manner, in that $\Delta\theta = (\theta_{i+1} - \theta_i) / (1 - \theta_i)$, or

$$\begin{aligned} \Delta\theta &= \psi_* + \frac{\alpha_i - \theta_i}{1 - \theta_i} (1 - \psi_*) (1 - \eta) d_i \\ &= \psi_* - \frac{F_{LS}^*}{1 - F_{LS}^*} (1 - \psi_*) (1 - \eta) d_i, \end{aligned} \quad (14)$$

which permits the determination of the coancestral effective size, $N_{\theta} = 1/\Delta\theta$,

$$N_{\theta}^* = \frac{1 - F_{LS}^*}{\psi_* (1 - F_{LS}^*) - F_{LS}^* (1 - \psi_*) (1 - \eta) d_i}. \quad (15)$$

Equations 12 and 15 represent instantaneous effective sizes that change as the F_{LS} changes over generations (CHESSER *et al.* 1993a). Asymptotically, $N_{\alpha} = N_{\theta}$ (CHESSER *et al.* 1993a), which permits the solution for dispersal rate of the contributing sex by solving $N_{\alpha} - N_{\theta} = 0$ for d_i ,

$$d_i = \frac{\psi_* (1 - F_{LS}^*)}{F_{LS}^* [1 - \psi_* (1 - \eta) - F_{LS}^* \eta]}. \quad (16)$$

When the above solution for d_i is substituted into the effective size equations (Equations 12 and 15), the asymptotic effective population size for uniparentally inherited genes is determined to be

$$N_e^* = \frac{1 - \psi_* (1 - \eta) - F_{LS}^* \eta}{(1 - F_{LS}^*) \psi_* \eta}. \quad (17)$$

The asymptotic solution for effective size can be solved for the original parameters, rather than using the F_{LS} , if the asymptotic solution for the F_{LS} could be determined from only those parameters. CHESSER (1991a,b) provides a means of determining the asymptotic F_{LS} without iteration of the transition matrix $\mathbf{T}$. CHESSER (*op. cit.*) defined the asymptotic F_{LS} as

$$\hat{F}_{LS} = 1 - \frac{\delta\theta}{\delta\alpha}, \quad (18)$$

where $\delta\theta = \theta_{i+1} - \theta_i$ and $\delta\alpha = \alpha_{i+1} - \alpha_i$. Therefore,

$$F_{LS}^{(i+1)} = 1 - \frac{\theta_{i+1} - \theta_i}{\alpha_{i+1} - \alpha_i}. \quad (19)$$

Substituting Equations 6 and 7 into Equation 19 we determine that

$$\begin{aligned} F_{LS}^{(i+1)} &= \frac{1 - \psi_*}{\eta} + \psi_* - \frac{\psi_* (1 - \theta_i)}{d_i \eta (\theta_i - \alpha_i)} \\ &= \frac{1 - \psi_*}{\eta} + \psi_* - \frac{\psi_* (1 - F_{LS}^{(i)})}{d_i \eta F_{LS}^{(i)}}. \end{aligned} \quad (20)$$

Because when F_{LS} becomes asymptotic $F_{LS}^{(i+1)} = F_{LS}^{(i)}$, the asymptotic value can be solved using Equation 20 as

$$\hat{F}_{LS} = \frac{d_i (1 - \psi_*) + \psi_* (1 + \eta d_i) - \sqrt{[d_i \psi_* (1 - \eta) - d_i - \psi_*]^2 - 4 \eta d_i \psi_*}}{2 \eta d_i}. \quad (21)$$

The solution to Equation 21 is exact when compared to the asymptotic result obtained by matrix iterations (Equations 8 and 9). The result of Equation 21 can be substituted into Equation 17 to derive the asymptotic effective size for maternally inherited genes using only the original parameter definitions for d_i , η , and ψ_* .

Alternatively, the other half of the quadratic equation in Expression 21 is equal to $N_e^* \psi_*$; therefore,

$$\hat{N}_e^* = \frac{d(1 - \psi_*) + \psi_*(1 + \eta d)}{2\eta d \psi_*} + \frac{\sqrt{[d\psi_*(1 - \eta) - d - \psi_*]^2 - 4\eta d \psi_*}}{2\eta d \psi_*}. \quad (22)$$

Again, Equation 22 is an exact solution for the asymptotic rate of change of uniparentally inherited genes when compared to results determined by iterations of the transition matrix.

Although the effective sizes for maternal and paternal genes appear to be similar, the dispersal rates and variances of progeny numbers for males and females may be very different. Thus, the F_{LS} values for the two sexes may be disparate depending on dispersal rates for each sex, male polygyny (ϕ_m) and multiple paternity (ϕ_w). It can be seen that the dynamics of maternally and paternally inherited genes will be identical when $\psi_m = \psi_f$ and $d_m = d_f$. Equality of maternal (M) and paternal (P) effective sizes, therefore, occur under the conditions of

$$N_e^{(M)} = N_e^{(P)}$$

$$\text{for } \begin{cases} \phi_w = 1, & \phi_m = 0, & d_m = d_f \\ \phi_w < 1, & \phi_m = \frac{(1 - \phi_w)\psi_f}{1 - \phi_w\psi_f}, & d_m = d_f \end{cases}, \quad (23)$$

which apply to cases of single and multiple paternity for the upper and lower conditions, respectively.

Comparisons of effective sizes: It is of interest to compare the effective sizes for maternal, paternal, and diploid genes for a variety of male and female dispersal rates and for various breeding tactics. CHESSER *et al.* (1993) detailed the determination of effective sizes for diploid traits in subdivided populations. For diploid models the correlation of genes within individuals (inbreeding, F) must be included to derive the transitions; thus, three variables, F , θ , and α must be accounted for rather than two, θ and α , in the above models with only uniparental inheritance. As in previous applications (CHESSER *et al.* 1993a), we assume the sex ratio to be equal but note that not all males may breed. Dramatic differences in primary (at birth) sex ratios may affect the resultant gene correlations, especially when breeding group sizes are very small. The diploid models for transitions of gene frequency correlations within individuals, within lineages, and within the total population reported by CHESSER *et al.* (1993a) and SUGG and CHESSER (1994) are [with modifications to now include variation in lineage sizes and to correct a mistake in an appendix equation presented by SUGG and CHESSER (1994)] as follows:

$$F_{t+1} = (1 - (1 - \eta)D)\theta_t + (1 - \eta)D\alpha_t, \quad (24)$$

$$\begin{aligned} \theta_{t+1} = & \frac{\psi_f(1 + \phi_w) + \phi_m(1 - \psi_f)}{8} (1 + F_t) \\ & + \frac{2(1 - (1 - \eta)D) + \psi_f(1 - \phi_w)}{4} \times (1 - (1 - \eta)d_m) + (1 - \psi_f) \\ & \times (2 - \phi_m - (1 - \eta)B) \theta_t \\ & + \frac{(1 - \eta)(2D + \psi_f(1 - \phi_w)d_m + (1 - \psi_f)B)}{4} \alpha_t \end{aligned} \quad (25)$$

$$\alpha_{t+1} = \frac{\eta(2D + d_m + d_f)}{4} \theta_t + \frac{4 - \eta(2D + d_m + d_f)}{4} \alpha_t, \quad (26)$$

where $D = d_m + d_f - d_m d_f$ and $B = d_m(1 - \phi_m) + d_f$. Combining these expressions, the nonzero elements of a transition matrix become

$$\begin{aligned} T_{1,2} &= (1 - (1 - \eta)D), \\ T_{1,3} &= (1 - \eta)D, \\ T_{2,1} &= \frac{\psi_f(1 + \phi_w) + \phi_m(1 - \psi_f)}{8}, \\ & \quad \frac{2(1 - (1 - \eta)D) + \psi_f(1 - \phi_w)}{4} \times (1 - (1 - \eta)d_m) + (1 - \psi_f) \\ & \quad \times (2 - \phi_m - (1 - \eta)B), \\ T_{2,2} &= \frac{(1 - \eta)(2D + \psi_f(1 - \phi_w)d_m + (1 - \psi_f)B)}{4}, \\ T_{2,3} &= \frac{\eta(2D + d_m + d_f)}{4}, \\ T_{3,2} &= \frac{4 - \eta(2D + d_m + d_f)}{4}, \end{aligned} \quad (27)$$

with the only nonzero element of the constant vector is $C_2 = T_{2,1}$. The transitions for the state of all variables can now be determined from

$$\{F_{t+1}, \theta_{t+1}, \alpha_{t+1}\} = \begin{bmatrix} 0 & T_{1,2} & T_{1,3} \\ T_{2,1} & T_{2,2} & T_{2,3} \\ 0 & T_{3,2} & T_{3,3} \end{bmatrix} \{F_t, \theta_t, \alpha_t\} + \begin{bmatrix} 0 \\ C_2 \\ 0 \end{bmatrix}. \quad (28)$$

Unfortunately, exact solutions in terms of the original parameters for diploid $\hat{F}_{LS}$ and $\hat{N}_e$ are not tractable as they were for paternally and maternally inherited genes. Therefore, iteration of the matrix is necessary to attain exact estimates of gene correlations and effective sizes. Following the procedures presented by CHESSER *et al.* (1993a, p. 1228) we can show that the inbreeding and intergroup effective sizes are

$$N_e = \frac{1 - F_{IS}}{2(F_{LS}(1 - (1 - \eta)D) - F_{IS})} \quad (29)$$

and

$$N_{\alpha} = \frac{2}{\eta(2D + d_m + d_f)F_{LS}} \approx \frac{2}{3D\eta F_{LS}}. \quad (30)$$

When the asymptotic effective size is attained, $N_{ef} = N_{\alpha}$; therefore,

$$D = d_m + d_f - d_m d_f \approx \frac{4(\hat{F}_{LS} - \hat{F}_{IS})}{\hat{F}_{LS}(4 - \eta(1 + 3\hat{F}_{IS}))}. \quad (31)$$

When the result of Equation 31 is substituted into 30, an accurate estimate of the asymptotic effective size for diparentally inherited genes becomes

$$\hat{N}_e \approx \frac{4 - \eta(1 + 3\hat{F}_{IS})}{6\eta(\hat{F}_{LS} - \hat{F}_{IS})}. \quad (32)$$

If $\eta = 1/s$, Equation 32 is equal to the result given in CHESSEY *et al.* (1993a).

We compared effective sizes for maternal, paternal and diploid genes using $k = \sigma_k^2 = 2$ and 30 breeding groups (s) in the total population. For single paternity ($\phi_w = 1$), we have depicted six general scenarios of breeding and dispersal tactics: (1) a “random” model wherein there is monogamous mating ($\phi_m = 0$), male and female dispersal rates are equal to 0.5, and 15 females, with a variance of 4, per breeding group; (2) a “minimal social structure” (*e.g.*, CHESSEY *et al.* 1993b) that was identical to scenario 1, above, except that there is slight polygyny ($\phi_m = 0.1$); (3) a “bird-like” population structure (*cf.* GREENWOOD 1980) that was identical to 1, above, except that male and female dispersal rates were set to 0.1 and 0.9, respectively; (4) a “typical mammalian population structure” (*cf.* GREENWOOD 1980) wherein there is greater polygyny ($\phi_m = 0.4$), smaller breeding group size ($n = 8$, $\sigma_n^2 = 3$), and predominant male dispersal ($d_m = 0.75$, $d_f = 0.25$); (5) a “highly social mammal population structure” [*e.g.*, *Cynomys*, CHESSEY (1983, 1991a,b)] with near complete polygyny for each breeding group ($\phi_m = 0.9$), small breeding group size ($n = 5$, $\theta_n^2 = 2$) and near complete female philopatry and male-biased dispersal ($d_m = 0.9$, $d_f = 0.1$); and (6) a “lek” model wherein males are highly polygynous ($\phi_m = 0.99$), a son inherits the father’s breeding group ($d_m = 0.05$) and most females disperse and, thus, mate randomly with the available males ($d_f = 0.99$). Other parameters of the model are the same as scenario number 1.

The random model showed that maternal and paternal effective sizes attained identical asymptotic effective sizes that were approximately one-half that for the diploid trait (Figure 1A). This scenario reflects the traditional expectation for haploid *vs.* diploid effective sizes (CROW and KIMURA 1970). Effective sizes for uniparentally inherited genes are slightly greater than one-half that for diploid genes because the latter includes losses via segregation; there are no segregational losses in homoplasmic genes conveyed by a single parent. Introduction of slight polygyny ($\phi_m = 0.1$) in the minimal social

structure model radically changed the relationships among the asymptotic effective sizes for maternal, paternal and diploid traits (Figure 1B). The reduction in the number of male breeders lowers the asymptotic effective size for diploid traits with a concomitant reduction in paternal effective size. The asymptotic effective size for maternal genes is unchanged from that for the random model. Effective size for maternally inherited genes is now ~90% of that for diploid genes, whereas paternal effective size is <50%. The bird-like population structure (Figure 1C) shows that changing the relative dispersal rates to reflect strong female bias ($d_f = 0.9$, $d_m = 0.1$) raises the asymptotic effective size for paternal genes to become greater than those for maternal ones, whereas effective size for diploid traits is only changed slightly from the first model (panmixia, Figure 1A). Typical mammalian parameters where females show considerably greater philopatry than males and where males tend to be polygynous (Figure 1D) have asymptotic effective sizes for maternal genes that are over one and one-half times larger than that for diploid traits and almost six times larger than that for paternal genes. Female philopatry increases the variance of maternal gene frequencies among groups. The variance among groups slows the rate of loss of overall variation of maternally inherited genes. Effective sizes for paternal genes are low because of a relatively low number of male breeders ($\phi_m = 0.4$) and because high male dispersal rate reduces the variation among breeding groups. Thus, variation in paternal genes will be lost at a faster rate than will maternal and diploid genes. These effects are magnified by greater degrees of female philopatry, male-biased dispersal and male polygyny as would be found in highly social mammals (Figure 2A; CHESSEY 1991a). For the parameters chosen, the asymptotic effective size for haploid maternal traits is almost four times greater than that for diploid genes and about 11 times greater than for paternal genes. When there is high male polygyny but high female dispersal, as in the lek model, effective sizes for paternal and maternal genes far exceed those for diparentally inherited genes (Figure 2B) with paternally inherited traits having the highest effective size and over six times that for diparental genes.

It should be noted that the differences between effective sizes for maternally inherited genes and those for diparentally inherited genes occur predominantly because of reductions in effective sizes for nuclear genes. Whereas effective sizes for all genes are negatively influenced by greater rates of dispersal of one or both sexes, the range in values in the examples for d_f and n made no dramatic changes in ultimate effective sizes for maternally inherited traits. Rates of change of genetic variance cannot be directly compared from the figures; the rate of loss of diparental gene diversity is $1/2N_e$, whereas that for maternally and paternally inherited genes is simply $1/N_e$. For example, the $N_e^{(M)}$ would need to be twice the size of N_e for the rate of loss of

FIGURE 1.—Effective population sizes for uniparentally (maternal and paternal) inherited and diparentally inherited genes for estimated parameters relevant to panmixia (A), minimal social structure (B), bird-like population structure (C), and typical mammalian population structure (D). Values for the parameters are provided and parameter definitions are given in the text.

maternally inherited gene diversity to equal to that for diparental genes.

Multiple paternity always increases the effective population size for diploid genes and in extreme cases may lead to a near doubling of N_e (SUGG and CHESSER 1994). For diploid genes coancestry among progeny born within breeding groups continues to accumulate because of genes contributed by mothers. Even if all progeny produced by a mother have different fathers their coancestry will be at least $1/8$. This is not the case for genes that have uniparental inheritance. All progeny of a dam will inherit the maternally transmitted genes of their mother regardless of sire. Thus, multiple paternity will not affect the dynamics of maternally inherited genes. Multiple paternity may have large effects on the coancestry of genes that are inherited from the sire. In some cases full siblings or half siblings that are related by having a common mother will have no paternal genes that are identical by descent. Therefore, for genes inherited from the sire, multiple paternity can yield very low coancestry values among progeny born within breeding groups and produce large effective population sizes. In fact, the $N_e^{(P)}$ is not limited to twice that for single paternity, but can be infinitely larger

when multiple paternity is common (Figure 3). The extreme case is evident when there is a separate father for each progeny born within a breeding group ($\phi_w = \phi_m = 0$); in this instance, the $N_e^{(P)}$ approaches infinity because there is no coancestry for the paternally inherited genes among progeny born within the population, an unrealistic but interesting scenario.

DISCUSSION

The equations derived above provide novel perspectives for considering changes of gene diversity for uniparentally and diparentally inherited genes. Gene dynamics for uniparentally inherited genes are more complicated than has been traditionally considered (e.g., AVISE 1994). Effective population sizes for maternally and paternally inherited genes are not always smaller than their diploid counterparts, but rather can be considerably larger under some circumstances. Effective sizes for all modes of inheritance are functions of parameters for the means and variances of surviving progeny numbers, breeding group sizes, and numbers of mates, as well as dispersal rates for males and/or females. Because values for these parameters may vary

FIGURE 2.—Effective population sizes for uniparentally (maternal and paternal) inherited and diparentally inherited genes for estimated parameters relevant to a highly social mammalian structure (A) and for groups in which there is high male polygyny and high female dispersal as may be found in some birds that form lek groups (B). Values for the parameters are provided and parameter definitions are given in the text.

with the modes of inheritance, however, the resultant values may differ likewise. Also, it is important to assess the rates of change for the different genes appropriately. Variation for diparentally inherited genes is lost at the rate of $1/(2N_e)$ while that for uniparental genes is reduced by $1/N_e$ each generation. Thus, the effective size for maternally or paternally inherited genes would need to be twice as large as that for diploid genes for them to experience the same rate of loss of variation. On the other hand, if N_e for uniparental genes is one-half that for diparentally inherited genes, as is commonly assumed (BIRKY *et al.* 1983; AVISE 1994), then the rate of loss for maternal or paternal genes will be four times faster than for diploid genes.

Effective sizes for organelles are equal to the number of the transmitting sex only within single, isolated populations exhibiting random mate selection and zero variance in progeny numbers. Equation 3 for maternally inherited genes can be rearranged readily to suffice for a single population without subdivisions ($s = 1$; d_f or $d_m = 1$) as

$$\begin{aligned}\theta_{t+1} &= \psi_f^* + (1 - \psi_f^*)\theta_t, \\ \Delta\theta &= \frac{\theta_t + \psi_f^*(1 - \theta_t) - \theta_t}{1 - \theta_t}, \\ N_e^{(M)} &= \frac{1}{\psi_f^*}.\end{aligned}\quad (33)$$

FIGURE 3.—Three-dimensional diagram showing the relationship between male polygyny and multiple paternity in determining the asymptotic effective size for paternally inherited genes.

The value of ψ_f^* , above, is not equal to ψ_f in Equation 1 because now all females are considered to be in a single population and the contribution of a female's family is relative to the total population rather than simply the size of a breeding group. Because there are $N = ns$ breeding females in the total population, the values for ψ_f^* and $N_e^{(M)}$ for an undivided population are

$$\psi_f^* = \frac{\sigma_k^2 + k(k-1)}{k(kN-1)} \quad N_e^{(M)} = \frac{k(kN-1)}{\sigma_k^2 + k(k-1)}. \quad (34)$$

This result is precisely one-half of the effective size determined for undivided, random mating populations for diparentally inherited genes (CHESSEY *et al.* 1993a). It can be seen in Equation 34 that when $\sigma_k^2 \approx 0$, then the effective size for maternal genes will be approximately equal to the number of breeding females. This is the typical $N_e^{(M)}$ used in prior studies (BIRKY *et al.* 1989; AVISE 1994), but is clearly not applicable unless considering a single, isolated population with no female dispersal and zero variance in progeny per female.

One migrant per generation among population subunits serves only to prevent complete differentiation among groups. BIRKY *et al.* (1983, p. 525) report that there are rates of dispersal at which there will be subdivision for organelle genes but yet nuclear genes will be essentially panmictic. Their conclusions were based on the premise that when the number of dispersers per group (group size = N individuals; m = dispersal rate) exceeded one ($Nm > 1$) then the "rate of decay of nuclear gene diversity for the whole subdivided population is the same as in a random mating population" (BIRKY *et al.* 1983, p. 525; MARUYAMA 1971) and when $Nm < 1$ the rate of decay is $m/2s$. These conclusions are perplexing and are not supported by the derivations presented herein. Inspection of Equation 30 for nuclear genes and Equation 8 for uniparentally inherited genes shows estimates of effective gene flow to be $D\eta N_e \approx 2/3F_{IS}$, and $d_f\eta N_e^{(M)} = 1/F_{IS}^{(M)}$, respectively. These equations clearly show for maternally inherited genes that when the effective number of dispersers per group is

FIGURE 4.—Comparisons of effective sizes and spatial differentiation for maternally inherited and diparentally inherited genes. (A) Comparison of asymptotic effective sizes for maternally inherited genes (—) and diparentally inherited genes (---) over the range of possible dispersal rates for females. Horizontal lines denote expected values for a single randomly mating population. (B) Comparison of resultant asymptotic $F_{LS}^{(M)}$ for maternally inherited genes (---) and F_{LS} for diparental genes (solid line) and the ratio of $F_{LS}^{(M)}$ to F_{LS} (---) over the range of dispersal rates for females. All values were determined using parameter values of $n = m = 15$, $\sigma_n^2 = 4$, $s = 30$, $k = \sigma_k^2 = 2$, $\psi_m = 0$, and $d_m = d_f$. Parameter definitions are explained in the text.

greater than or equal to one ($d_f \eta N_e^{(M)} \geq 1$), the differentiation among groups ($F_{LS}^{(M)}$) must be less than or equal to one. Effective population sizes for maternally inherited genes and diparentally inherited genes were calculated for dispersal rates ranging from zero to one and compared to the effective population size for the scenario where all individuals bred randomly within a single population. Results of this exercise (Figure 4A) show that the rate of decay of gene diversity for the

whole subdivided population will not be the same as in a random mating population unless the dispersal rate is unity (cf. CHESSER 1991a,b). The same criteria hold for maternal, paternal, or diparental genes. The resultant differentiation among groups (F_{LS}) and ratios of $F_{LS}^{(M)}$ to F_{LS} are shown in Figure 4B. The differentiation among families is fourfold for maternal organelle genes than for diparental genes when dispersal is complete ($d_f = 1$) but this ratio varies between two and five for lower dispersal rates. Thus, an average of one migrant per generation among groups is necessary to prevent complete differentiation of groups; nothing more is implied and no conclusions regarding equality of effective size with that of a panmictic population can be made.

In some population structures random losses of organelle diversity may be slower than those for nuclear genes. The mutation rate for mtDNA is reportedly about five to 10 times that for nuclear DNA (BROWN *et al.* 1979), and it has been speculated that mutation rate may be responsible in part for the maintenance of a high diversity of mtDNA types within species. However, as shown in this study, the effective sizes for maternal genes in some taxa may be considerably larger than those for nuclear genes. Such results suggest that maintenance of variation for mtDNA may be expected given complex social structures and low female dispersal rates associated with many groups (particularly mammals). A concomitantly high genetic divergence among breeding groups or subpopulations would be expected. Thus, a high mutation rate may not be needed to explain high variation for mtDNA in natural populations under many circumstances.

Variance in breeding group sizes reduces effective population sizes. It is important to note that when population subdivision is present the variance in group size (σ_n^2) serves to reduce the effective population size in a similar manner as does the variance in progeny number (σ_k^2 ; e.g., CROW and KIMURA 1970; WRIGHT 1978). Reduction of the variance in progeny number has been suggested as a tool for increasing effective population sizes for managed populations (DENNISTON 1978). Clearly, similar reductions in the variance in group sizes will play an important role for increasing effective size when there are interacting subunits comprising the population. Incorporation of a variance in group size more accurately reflects realistic scenarios for metapopulation structures where there are variations in the size and productivity of resource patches (HANSKI 1991).

Most taxa extensively studied thus far show at least some measure of spatial heterogeneity with dispersal rates estimated to be less than unity. Clearly, then, the rate of change of gene diversity for maternal, paternal, and diparental genes will act in accordance to the parameters of the models presented. The size of an empirical sample is immaterial to the processes that are actually affecting the population. Therefore, great care

must be exercised when estimating effective size from single populations, when in fact several populations may be interacting. Efforts to discern accurate demographic information regarding dispersal rates and relative group and litter sizes may be necessary to attain greater precision for estimates of temporal dynamics of maternal, paternal, and diparentally inherited genes. One important aspect of the models presented is that the parameters needed to determine dynamics and heterogeneity of diparentally inherited genes are precisely those needed to estimate values for maternal and paternal genes. In fact, values for ψ_f , ψ_m , η , d_m , and d_f will permit estimation of all variables necessary for gene correlations, *F*-statistics, and effective sizes. Therefore, adequate knowledge of some crucial characteristics permits prediction of effective sizes and genetic divergence for uniparental and diparental genes.

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