THE INFLUENCE OF THE MATING SYSTEM ON THE MAINTENANCE OF GENETIC VARIABILITY IN POLYGENIC CHARACTERS

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ABSTRACT

The traditional models of the effect of assortative mating and inbreeding on the genetic variance of polygenic characters (FISHER 1918; WRIGHT 1921) presume that there is no natural selection or mutation. In a large population, the genetic variance determined by additive genes may then increase by up to a factor of two with local inbreeding, and even more with assortative mating. The classical models are still used to interpret data from natural populations. But contrary to their assumptions, most metrical characters in natural populations are usually thought to be under a type of selection which depletes polygenic variation. Mutation is then necessary to maintain genetic variation. The present models show that with the additional features of mutation and selection, in a large population, the mating system has no influence on the amount of genetic variability maintained by additive genes.

EVOLUTIONARY biologists have long been interested in the role of different mating systems in natural populations, especially their effect on the genetic variation of polygenic characters. The original models of FISHER (1918) on assortative mating and WRIGHT (1921) on inbreeding are still widely used in textbooks and in the design of artificial breeding programs. In both of these models, it is assumed that there is no natural selection and no mutation operating on the character of interest. Thus, the gene frequencies do not change and the breeding system only rearranges the existing genetic variation. These assumptions are appropriate in artificial breeding where the transient effect of the mating system on the genetic variance usually occurs much faster than the action of mutation or natural selection.

In these models the genetic variance maintained under different mating systems is expressed relative to the genetic variance in a randomly mating population *with the same gene frequencies*, V_0. In a large population with local inbreeding, the genetic variance of a character determined by additive genes is $(1 + f)V_0$. The inbreeding coefficient for the entire population, f, is the proportional increase in homozygosity in comparison to an effectively panmictic population with the same gene frequencies. With local inbreeding, the genetic variance of additive genes may increase by up to a factor of two.

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In an assortatively mating population with a phenotypic correlation A between mates, the equilibrium genetic variance is $V_0/[1-A\hat{h}^2(1-\frac{1}{2n_E})]$ where n_E is the effective number of loci and $\hat{h}^2$ is the equilibrium heritability of the character in the assortatively mating population. Thus strong assortative mating for a highly heritable polygenic character can produce a large increase in the genetic variance. FISHER (1918) used this model to analyze the effect of assortative mating on the variation in human height.

The situation in natural populations is fundamentally different from the assumptions of selective neutrality and immutable genes on which the traditional models are based. In natural populations which exist for long periods of time, selection and mutation cannot be ignored. WRIGHT (1952) showed that the traditional formula for the genetic variance in an inbreeding population, $(1 + f)V_0$, also holds when the gene frequencies among partially isolated subpopulations are approaching or in a steady state distribution resulting from mutation, migration, selection and random drift. V_0 is then defined as the genetic variance in a random mating population *with the same distribution of gene frequencies*. It should be noted, however, that V_0 is *not* the *equilibrium* genetic variance in the random mating population, except in the absence of selection. When there is selection and mutation, the gene frequencies will change with the system of mating. Thus even the model of WRIGHT (1952) does not allow a comparison of the genetic variance maintained in natural populations with different levels of inbreeding.

Most phenotypic characters in natural populations are probably under a type of selection which depletes genetic variance (stabilizing selection or a combination of stabilizing and fluctuating directional selection). Mutation is then necessary to maintain genetic variability in polygenic characters. LANDE (1976a) has demonstrated that observed amounts of mutation in polygenic characters of maize, mice and Drosophila flies are sufficient to maintain levels of heritable variation typical for such characters, even when there is strong stabilizing selection. The present models of mating systems allow for mutation, linkage and natural selection.

THE MODEL

We consider a character in a diploid population influenced by n genes which have additive effects within and between loci. Following a model of KIMURA (1965) it is assumed that at each locus there is a wide range of possible allelic effects. This is justified by a consideration of the molecular structure of proteins, which indicates that there are many possible amino acid substitutions which could affect the function of the molecule. Among the major mutations of *Drosophila melanogaster*, most groups of independently derived, allelic mutations show a multiplicity of phenotypic effects (LINDSLEY and GRELL 1968), and it must be supposed that the same is true for the mutations of minor effect which contribute to the variation of polygenic characters. It is further assumed that at the i^{th} locus every allele mutates with probability μ_i each generation and the

mutational changes have a mean of zero and variance m_i^2 for all alleles at the locus. It is readily demonstrated that the change per generation in the covariances of allelic effects between the loci is $\delta_{ij}\mu_i m_i^2$, where δ_{ij} is one if $i = j$ and zero otherwise (see APPENDIX). The total increase in genetic variance per generation by mutation in a diploid population is then

$$\sigma_m^2 = 2\sum_{i=1}^{n} \mu_i m_i^2 .$$

It is also assumed that the character is subject to environmental variance, σ_e^2, and that there is no interaction or correlation between genetic and environmental effects. The recombination rate between loci i and j is denoted as r_{ij} and the covariance between allelic effects at these loci in the gametes is $C_{ij}(t)$ in generation t. The covariances satisfy the relationship

$$C_{ij}(t+1) = (1-r_{ij})[C_{ij}(t)]_w + r_{ij}[C'_{ij}(t)]_w + \delta_{ij}\mu_i m_i^2 \qquad (1)$$

where the subscript w indicates that selection has acted and a prime signifies that the covariance is between alleles from different gametes. From this point the problem is to express $[C_{ij}(t)]_w$ and $[C'_{ij}(t)]_w$ as functions of the $C_{ij}(t)$ and $C'_{ij}(t)$ in different mating systems to solve (1) as recursion equations.

In a previous paper, LANDE (1976a) explored the maintenance of genetic variation in such a system of linked, mutable loci in a random mating population. ALAN ROBERTSON and MICHAEL BULMER (personal communication) have suggested the application of regression analysis to determine the change in the covariances due to selection on the phenotype. This method will be applied here to various nonrandom systems of mating.

Before selection, the covariance $C_{ij}(t)$ can be partitioned into a component which is independent of variation in the phenotype and a component which depends only on the variation in the phenotype (KENDALL and STUART 1973, pp. 337–9).

$$C_{ij}(t) = C_{ij\cdot z}(t) + C_{iz}(t)C_{jz}(t)/\sigma_z^2(t) . \qquad (2)$$

$C_{ij\cdot z}(t)$ is the conditional covariance (for a fixed value of the phenotype, z). $C_{iz}(t)$ is the covariance between the phenotype and the alleles at locus i in the gametes,

$$C_{iz}(t) = \sum_{j=1}^{n} [C_{ij}(t) + C'_{ij}(t)] . \qquad (3)$$

The genetic variance that is expressed in the phenotype is

$$\sigma_g^2(t) = 2\sum_{i=1}^{n} C_{iz}(t) \qquad (4a)$$

and the total phenotypic variance is

$$\sigma_z^2(t) = \sigma_g^2(t) + \sigma_e^2 . \qquad (4b)$$

It is assumed that the regression of the allelic effects at locus i, x_i, on the phenotype is linear,

$$x_i - \bar{x}_i(t) = \frac{C_{iz}(t)}{\sigma_z^2(t)}\,[z - \bar{z}(t)] + \varepsilon_{i\cdot z} \tag{5a}$$

$\varepsilon_{i\cdot z}$ is an independent residual element of variation in allelic values at locus i, which is assumed to be homoscedastic (having equal variance for all z). Taking the covariance between x_i and x_j in this form, equation (2) is obtained. Selection on the phenotype may be viewed in a graphical sense as changing the weighting factors for the distributions of the residual ($\varepsilon_{i\cdot z}$) for different values of z. It is evident that selection acting only on z will not alter the regression coefficient (the slope of the x_i versus z graph), the linearity of the regression, or the distribution of the residual term, though all of the covariances will change. Thus, after selection the regression of $[x_i]_w$ on $[z]_w$ is

$$[x_i]_w - [\bar{x}_i(t)]_w = \frac{C_{iz}(t)}{\sigma_z^2(t)}\,\{[z]_w - [\bar{z}(t)]_w\} + \varepsilon_{i\cdot z} \tag{5b}$$

Taking the covariance of $[x_i]_w$ and $[x_j]_w$ in this form gives

$$[C_{ij}(t)]_w = C_{ij\cdot z}(t) + \frac{[\sigma_z^2(t)]_w}{\sigma_z^2(t)}\,C_{iz}(t)C_{jz}(t)/\sigma_z^2(t)\ .$$

Comparing this with equation (2), it can be reasoned that because the last term in (2) is due to variation in the phenotype, it is changed by selection in proportion to the alteration of the phenotypic variance by selection. Using (2) to substitute for $C_{ij\cdot z}(t)$ produces

$$[C_{ij}(t)]_w = C_{ij}(t) - k(t)C_{iz}(t)C_{jz}(t)/\sigma_z^2(t) \tag{6a}$$

where $k(t) = 1 - [\sigma_z{}^2(t)]_w/\sigma_z{}^2(t)$ is the proportional reduction of phenotypic variance caused by selection. Similarly it can be shown that

$$[C'_{ij}(t)]_w = C'_{ij}(t) - k(t)C_{iz}(t)C_{jz}(t)/\sigma_z^2(t)\ . \tag{6b}$$

Conditions under which the assumptions of linearity and homoscedasticity of the regressions are approximately correct will be discussed later. Substituting equations (6) into (1) yields

$$\Delta C_{ij}(t) = -r_{ij}[C_{ij}(t) - C'_{ij}(t)] - k(t)C_{iz}(t)C_{jz}(t)/\sigma_z^2(t) + \delta_{ij}\mu_i m_i^2\ . \tag{7}$$

These are the general dynamic equations for the genetic structure of a polygenic character with additive, mutable genes under natural selection on the phenotype. In subsequent sections we will examine the details of the equilibrium genetic structure and some dynamics for specific systems of mating. First we will derive general formulae for the expressed genetic variance which is maintained by mutation.

The diagonal equations in (7) have $r_{ii} = 0$ and $\delta_{ii} = 1$. Using a caret to denote equilibrium they simplify to

$$-\hat{k}\hat{C}_{iz}^2/\hat{\sigma}_z^2 + \mu_i m_i^2 = 0 \ .$$

$2\hat{C}_{iz}$ is the amount of expressed genetic variance attributable to locus i, which must be positive. This equation therefore has the unique solution

$$\hat{C}_{iz} = +\sqrt{\mu_i m_i^2 \hat{\sigma}_z^2/\hat{k}} \ . \tag{8}$$

The expressed genetic variance is then given by

$$\hat{\sigma}_g^2 = 2\sum_{i=1}^{n} \hat{C}_{iz} = 2\sqrt{\hat{\sigma}_z^2/\hat{k}} \sum_{i=1}^{n} \sqrt{\mu_i m_i^2} \ .$$

This equation may be solved for $\hat{\sigma}_g{}^2$ using (4b). It is also convenient to use

$$n_E = 2\left(\sum_{i=1}^{n} \sqrt{\mu_i m_i^2}\right)^2 \Big/ \sigma_m^2$$

as the effective number of loci at equilibrium; with n equally mutable loci ($\mu_i m_i{}^2 = \mu_j m_j{}^2$) we have $n_E = n$. This definition produces

$$\hat{\sigma}_g^2 = \sqrt{2(\hat{\sigma}_g^2 + \sigma_e^2) n_E \sigma_m^2/\hat{k}} \ . \tag{9}$$

With the segregation of many additive genes and an independent, normally distributed environmental effect, the phenotype distribution will tend to be normal. The selection parameter $\hat{k}$ then depends only on the fitness function for the character and the phenotypic mean and variance, $\hat{\sigma}_g{}^2 + \sigma_e{}^2$. The recombination rates and the correlations between alleles in uniting gametes do not appear in equation (9). Thus the formula shows that the expressed genetic variance which is maintained by mutation is independent of both the system of mating and the linkage map of the loci. The expressed genetic variance is completely determined by the mutation and selection parameters, and the environmental variance. Using a normal phenotype distribution, expressions for the genetic variance can be obtained for particular forms of selection. If the fitness of phenotype z is given by the Gaussian function $W(z) \propto \exp\{-\frac{1}{2}(z-\theta)^2/w^2\}$ the selection parameter is $k(t) = \sigma_z{}^2(t)/[\sigma_z{}^2(t) + w^2]$ which yields

$$\hat{\sigma}_g^2 = \sqrt{2n_E\sigma_m^2(w^2 + \sigma_e^2 + \tfrac{1}{2}n_E\sigma_m^2)} + n_E\sigma_m^2 \ . \tag{10}$$

The quadratic deviations fitness function, $W(z) \propto 1 - \alpha(z-\theta)^2$, acting on a normally distributed character, has the selection parameter $k(t) \simeq 2\alpha\sigma_z{}^2(t)$, (and is valid only for $\alpha \ll 1/\sigma_z{}^2(t)$ because otherwise a significant fraction of the population is assigned negative fitness), so

$$\hat{\sigma}_g^2 \simeq \sqrt{\frac{n_E\sigma_m{}^2}{\alpha}} \ . \tag{11}$$

This expression is in agreement with that of KIMURA (1965). Despite his assumption that the loci were uncorrelated, his calculation is correct because the expressed genetic variability maintained is independent of the recombination rates. Formula (10) was previously derived by LANDE (1976a).

It is notable that for both the Gaussian and quadratic deviation fitness functions, the genetic variance converges to its equilibrium independent of the population mean. The expressions (10) and (11) are therefore valid when the optimum phenotype, θ, is fluctuating in time.

Thus for any form of selection on the phenotype which diminishes the phenotypic variance, the mating system and the linkage map of the loci have no influence on the amount of genetic variation maintained by mutation. It is worthwhile to emphasize the assumptions which led to this rather startling conclusion. The first is the simplifying assumption that the loci are additive and that there is no dominance. The particular model of mutation used here has as its main feature that the rate of production of genetic variance by mutation is a constant, σ_m^2, independent of the background level of genetic variance. The fact that mutagenic agents generally increase genetic variance in quantitative characters of outbred populations indicates that natural populations are not near a maximum or saturation level of genetic variance. The following experiments on bristle number in *Drosophila melanogaster* directly demonstrate that the genetic variance induced by a given dose of X rays is roughly equal in homozygous and heterozygous strains.

SCOSSIROLI and SCOSSIROLI (1959) exposed "isogenic" and hybrid populations of *D. melanogaster* to 3000r of X rays per generation while selecting for increased sternopleural bristle number. Estimates of the background level of additive genetic variance in the control populations were 0.08 ($h^2 \simeq 0.06$) for the isogenic line and 0.42 ($h^2 \simeq 0.21$) for an average of two hybrid populations. The additive variance produced by one generation of irradiation was 0.40 in the isogenic line and an average of 0.42 in the two hybrid lines. In another experiment in which the same dose of radiation and selection procedure were practiced in alternating generations, the additive variance produced by one generation of irradiation was 0.09 in the isogenic line and an average of 0.14 for the two hybrid lines. From a comparison of regression coefficients of the mean bristle number against time in experimental and control populations, they concluded that the irradiation effects in the isogenic and hybrid lines were statistically indistinguishable.

YAMADA and KITAGAWA (1961) studied the effect of X rays on the short term response to selection for abdominal bristle number in *D. melanogaster*. Mutation rates were reported in units of genetic variance produced per roentgen of X rays. When both sexes were exposed to 1500r each generation, the average mutation rate was 28.2×10^{-5} in isogenic populations and 28.0×10^{-5} in hybrid populations. When only females were irradiated the mutation rate in the isogenic populations was 22.4×10^{-5} and 20.0×10^{-5} in the hybrid populations. When males only were irradiated, the mutation rate was 49.8×10^{-5} in the isogenic populations and 31.4×10^{-5} in the hybrid populations.

CLAYTON and ROBERTSON (1964) used the response to selection for abdominal bristle number to measure the amount of genetic variance accumulated by a population of *D. melanogaster* after 23, 60 and 140 generations of exposure to 1800r per generation. From an estimated effective population size of 60 and assuming that there was no selection on the new mutations in bristle number, they calculated the rate of production of new variance at the three times as 1.8×10^{-5}, 0.6×10^{-5}, and 1×10^{-5} units per roentgen. These results seem to be consistent within the experiment, especially in view of fluctuations in the genetic variance which can occur due to random genetic drift in small populations.

It is also presumed in the present models that the regression of allelic effects on the phenotype is approximately linear and homoscedastic. These conditions will be discussed further for particular breeding systems. However, it can be noted here that if the allelic effects at the different loci are independent, then the joint distribution exactly satisfies the requirement of linearity of the regressions. Approximate linearity is also expected when the correlations between loci are small. The assumption of homoscedasticity, that the variance of the residual term in equations (5) is independent of the phenotypic value z, is exactly satisfied by a multivariate normal distribution (which also meets the linearity condition, KENDALL and STUART 1973). If the linearity assumption is satisfied, approximate homoscedasticity is expected when each locus contributes a small fraction of the total phenotypic variation, regardless of the form of the distribution of allelic effects. We now proceed to the analysis of the equilibrium genetic structure for some specific systems of mating.

Random mating

With random mating, there is no correlation between alleles in uniting gametes so $C'_{ij}(t) = 0$. Using (8), the detailed solution of equations (7) are then of the form

$$\hat{C}_{ij} = \frac{-\sqrt{\mu_i m_i^2 \mu_j m_j^2}}{r_{ij}} \quad \text{for } i \neq j \tag{12a}$$

and from (3)

$$\hat{C}_{ii} = \sqrt{\mu_i m_i^2}\left(\sqrt{\hat{\sigma}_z^2/\hat{k}} + \sum_{\substack{j=1 \\ j\neq i}}^{n} \frac{\sqrt{\mu_j m_j^2}}{r_{ij}}\right). \tag{12b}$$

Since all the correlations are negative, it can be shown that the average magnitude of correlation is less than $1/(n-1)$ (LANDE 1976a). Inspection of these equations shows that the correlation between allelic effects at two loci can be high only if the two loci are very tightly linked and isolated on the linkage map from other loci of comparable mutability. Thus, except for some cases of very tight linkage, the smallness of the correlations guarantees that in the gametes, the regressions of allelic effects on the phenotype are approximately linear. With a Gaussian fitness function, this solution is identical to that obtained by LANDE (1976a) for a multivariate normal distribution of allelic effects.

Inbreeding

In a large population which is inbreeding because of limited dispersal or due to some regular system of inbreeding, such as partial self-fertilization, there is a deficiency of heterozygotes compared to that expected in a panmictic population with the same gene frequencies. The inbreeding coefficient f is defined as the proportional deficiency of heterozygotes (WRIGHT 1921). It should be noted that with mutation the maximum value of f is less than one. When the genes have purely additive effects on the phenotype, inbreeding is reflected in a correlation between effects of alleles at the same locus in uniting gametes, $p'_{ii}(t) = f$. This inbreeding will also produce an indirect covariation between alleles at different loci in uniting gametes. This indirect covariance can be derived from the regression of x'_i on the allelic effects at the same locus of the complementary gamete in the zygotes,

$$x'_i - \bar{x}'_i(t) = f[x_i - \bar{x}_i(t)] + \varepsilon_{i'\cdot i} .$$

Multiplying by $x_j - \bar{x}_j(t)$ and taking expectations,

$$C'_{ij}(t) = fC_{ij}(t) \quad . \tag{13a}$$

$E[(x_j - \bar{x}_j(t))\varepsilon_{i'\cdot i}] = 0$ because x_j and x'_i can only become correlated by the inbreeding effect acting through x_i, which is held constant in the residual. This result can also be derived by the method of path analysis (WRIGHT 1934; LI 1976). In the path diagram for this system, the x'_i are completely determined by the set of correlated x_i, and a distinct set of correlated residuals, $\varepsilon_{i'\cdot i}$ as in the above regression equation. The relation which emerges is $\rho'_{ij}(t) = f\rho_{ij}(t)$ in agreement with (13a).

Substituting (13a) in equation (7) gives the rate of decay of the covariance between loci i and j as $(1-f)r_{ij}$ in a large population. By comparison with the random mating system, all recombination rates are multiplied by $(1-f)$. Inbreeding therefore has an effect similar to tightening the linkage. Referring to equation (3),

$$\hat{C}_{iz} = (1+f) \sum_{j=1}^{n} \hat{C}_{ij} \quad . \tag{13b}$$

Using equation (8) the off-diagonal equations in (7) yield the solutions

$$\hat{C}_{ij} = \frac{-\sqrt{\mu_i m_i^2 \mu_j m_j^2}}{(1-f)r_{ij}} \quad \text{for } i \neq j \quad . \tag{13c}$$

Returning to (13b)

$$\hat{C}_{ii} = \sqrt{\mu_i m_i^2}\left(\frac{\sqrt{\hat{\sigma}_z^2/\hat{k}}}{1+f} + \frac{1}{1-f}\sum_{\substack{j=1 \\ j\neq i}}^{n} \frac{\sqrt{\mu_j m_j^2}}{r_{ij}}\right) \quad . \tag{13d}$$

Since all the correlations between alleles in the gametes are negative, it is again the case that the average magnitude of correlation between loci in the gametes is less than $1/(n-1)$. From this solution, it can be seen that the allelic effects at

two loci can be highly correlated only if the loci are tightly linked and isolated on the linkage map from other loci of comparable mutability, as in the case of random mating.

The small average magnitude of correlation between loci in the gametes partially validates the use of the linear regression equations (5) as an approximation. With inbreeding, the joint distribution of x_i and x'_i is composed of an uncorrelated part of weight $(1-f)$ and a perfectly correlated part of weight f. Since both of these parts have linear, homoscedastic regressions, their sum (the regression preceeding equation 13a) is linear, but not exactly homoscedastic. This causes a deviation from homoscedasticity of the regression of x_i on the phenotype in (5a). Nevertheless, equations (5) should still be approximately correct for any degree of inbreeding if the selective coefficients on the individual loci are small, as would be the case when there is a large effective number of loci (BULMER 1971) or the heritability is low.

Though inbreeding has essentially no direct influence on the amount of genetic variance maintained by mutation, there may be an indirect effect if the inbred individuals are less stable in their development than outbred individuals. This would create a heterozygote advantage in fitness at the single locus level (LERNER 1954), further augmenting the genetic variance above that given here.

Assortative mating

Phenotypic assortative mating is assumed to take place such that the regression of the phenotype on that of the mate is linear, with the phenotypic correlation between mates being A. The phenotypic covariance among mating pairs formed after selection is then $A[\sigma_z^2(t-1)]_w$ in generation $t-1$. To distinguish the two parents, an equation analogous to (5b) is written, but with a subscripted phenotype,

$$[x_i]_w - [\bar{x}_i(t-1)]_w = \frac{C_{iz}(t-1)}{\sigma_z^2(t-1)}\{[z_1]_w - [z_1(t-1)]_w\} + \varepsilon_{i\cdot z_1}$$

and there is a similar regression of $[x_j]_w$ on $[z_2]_w$. The covariance between alleles at locus i in the first parent with alleles at locus j in the mate in generation $t-1$ is then

$$[C^*_{ij}(t-1)]_w = A[\sigma_z^2(t-1)]_w C_{iz}(t-1)C_{jz}(t-1)/\sigma_z^4(t-1).$$

$E[\varepsilon_{i\cdot z_1}\varepsilon_{j\cdot z_2}] = 0$ because the only source of correlation of $[x_i]_w$ with $[x_j]_w$ in the mate is through the phenotypes, which are held constant in the residuals. Since mutation and recombination will not affect the covariance of alleles in mates, it is apparent that $[C^*_{ij}(t-1)]_w$ is equal to the covariance between alleles in uniting gametes, $C'_{ij}(t)$. Using the definition $[\sigma_z^2(t-1)]_w = [1-k(t-1)]\sigma_z^2(t-1)$,

$$C'_{ij}(t) = [1-k(t-1)]AC_{iz}(t-1)C_{jz}(t-1)/\sigma_z^2(t-1) \quad . \qquad (14a)$$

In the extreme case of $k(t-1) = 1$, no phenotypic variance remains after selection, and the covariance between alleles in uniting gametes is zero. With no

selection and perfect assortative mating, this reduces to the covariance due to variation in the phenotype alone, as in formula (2). This expression can also be obtained by the method of path analysis. In the path diagram the correlation between $[z_1]_w$ and $[z_2]_w$ is A, and the allelic values within individuals, $[x_i]_w$, are completely determined by the selected phenotype and a set of correlated residuals, $\varepsilon_{i\cdot z}$ as in the regression equation for this system. From this the following relation is derived,

$$[\rho^*_{ij}(t-1)]_w = [\rho_{iz}(t-1)]_w A [\rho_{jz}(t-1)]_w$$

or converting to covariances

$$[C^*_{ij}(t-1)]_w = A[C_{iz}(t-1)]_w [C_{jz}(t-1)]_w / [\sigma_z^2(t-1)]_w$$

Multiplying equation (5b) by $[z]_w - [\bar{z}(t)]_w$, taking expectations and using the definition of $[\sigma_z^2(t)]_w$, it is found that $[C_{iz}(t)]_w = [1-k(t)]C_{iz}(t)$. Substituting this and the definition of $[\sigma_z^2(t-1)]_w$ in the above expression, we again derive (14a).

Employing the relation (14a) in (3) at equilibrium,

$$\hat{C}_{iz} = \sum_{j=1}^{n} \hat{C}_{ij} + \frac{(1-\hat{k})A\hat{h}^2}{2}\hat{C}_{iz} \tag{14b}$$

where $\hat{h}^2 = \hat{\sigma}_g^2/\hat{\sigma}_z^2$. Using (14a) and (8) the off-diagonal equations in (7) at equilibrium give

$$\hat{C}_{ij} = -\sqrt{\mu_i m_i^2 \mu_j m_j^2}\left[\frac{1}{r_{ij}} - \frac{A(1-\hat{k})}{\hat{k}}\right] \qquad \text{for } i \neq j. \tag{14c}$$

Returning to (14b) and again using (8),

$$\hat{C}_{ii} = \sqrt{\mu_i m_i^2}\left\{\sqrt{\hat{\sigma}_z^2/\hat{k}}\left[1 - \frac{(1-\hat{k})A\hat{h}^2}{2}\right] + \sum_{\substack{j=1 \\ j\neq i}}^{n}\sqrt{\mu_j m_j^2}\left[\frac{1}{r_{ij}} - \frac{A(1-\hat{k})}{\hat{k}}\right]\right\}. \tag{14d}$$

The phenotypic correlation between mates, A, may be either positive or negative. If A is negative all correlations between alleles in gametes are negative and, as above, the average magnitude of correlation is less than $1/(n-1)$, showing that the assumption of linearity of the joint distribution of alleles in the gametes is a good approximation. The system of mating, which entails linear correlation between the phenotypes of mates, then guarantees that this approximation holds good for alleles in uniting gametes. With positive assortment of mates, the correlation $\hat{\rho}_{ij}$ will be negative if $A < \hat{k}/\{(1-\hat{k})r_{ij}\}$, which will tend to occur with tight linkage and strong stabilizing selection. Loose linkage, weak stabilizing selection and strong positive assortment of mates can produce large positive correlations between alleles in the gametes, and the assumption of linearity of the regressions may no longer be valid.

The effective number of loci defined here prior to equation (9) can be compared with that used by FELSENSTEIN and CROW for FISHER's model of assortative mating with additive genes (CROW and KIMURA 1970, p. 153). Their effective number, n_e, is expressed in terms of the covariances of allelic effects in uniting gametes. At equilibrium, in the present notation this is

$$n_e = \sum_{i=1}^{n} \sum_{j=1}^{n} \hat{C}'_{ij} \Big/ \sum_{i=1}^{n} \hat{C}'_{ii} \ .$$

Substituting (14a) and then (8) this becomes equivalent to n_E. Their effective number of loci, defined in terms of the assortative mating system, is identical to that used here, which is expressed solely by the mutation parameters.

DISCUSSION

The traditional models of mating systems (FISHER 1918; WRIGHT 1921) predict substantial increases in the genetic variance of polygenic characters in inbreeding and assortatively mating populations in comparison to a randomly mating population with the same gene frequencies. In these models it is assumed that there is no mutation and no natural selection to change the gene frequencies. In the present models the amount of polygenic variation maintained is determined by a balance between mutation and an arbitrary form of phenotypic selection which depletes genetic variation. This includes stabilizing selection, and many types of fluctuating selection with a directional component. Recombination and the mating system act at an intermediate stage of the process to rearrange the mutations that natural selection acts upon. The model of mutation employed here is believed to be fairly realistic for natural populations (see above). After allowing for mutation, linkage and natural selection on a polygenic character with additive genes, it is found that, in contrast to the traditional models, the system of mating has no influence on the amount of genetic variance maintained.

This result indicates that, at least in large populations, the adaptive significance of different mating systems is not to be found in the amount of genetic variability maintained. Thus in a large population, where the rate of evolution is governed by the additive genetic variance (FISHER, 1930), the mating system would not affect the rate of evolution. However, in a spatially subdivided population with partially isolated local populations, evolution may proceed faster by genetic drift to new adaptive peaks for gene frequencies (WRIGHT 1932) or adaptive zones for phenotypes (LANDE 1976b).

In populations which are subject to frequent local extinction, transient effects of the mating system may be important in regulating the amount of genetic variability in local populations. For example, if the genetic variance has been depleted in a population crash, positive assortative mating will speed up the rate of approach to the equilibrium level (see Figure 1). This occurs because positive assortative mating decreases the variance of allelic effects at each locus which must be accumulated by mutation (equation 14d). Conversely, negative assortative mating slows the rate of approach to equilibrium. The influence of inbreed-

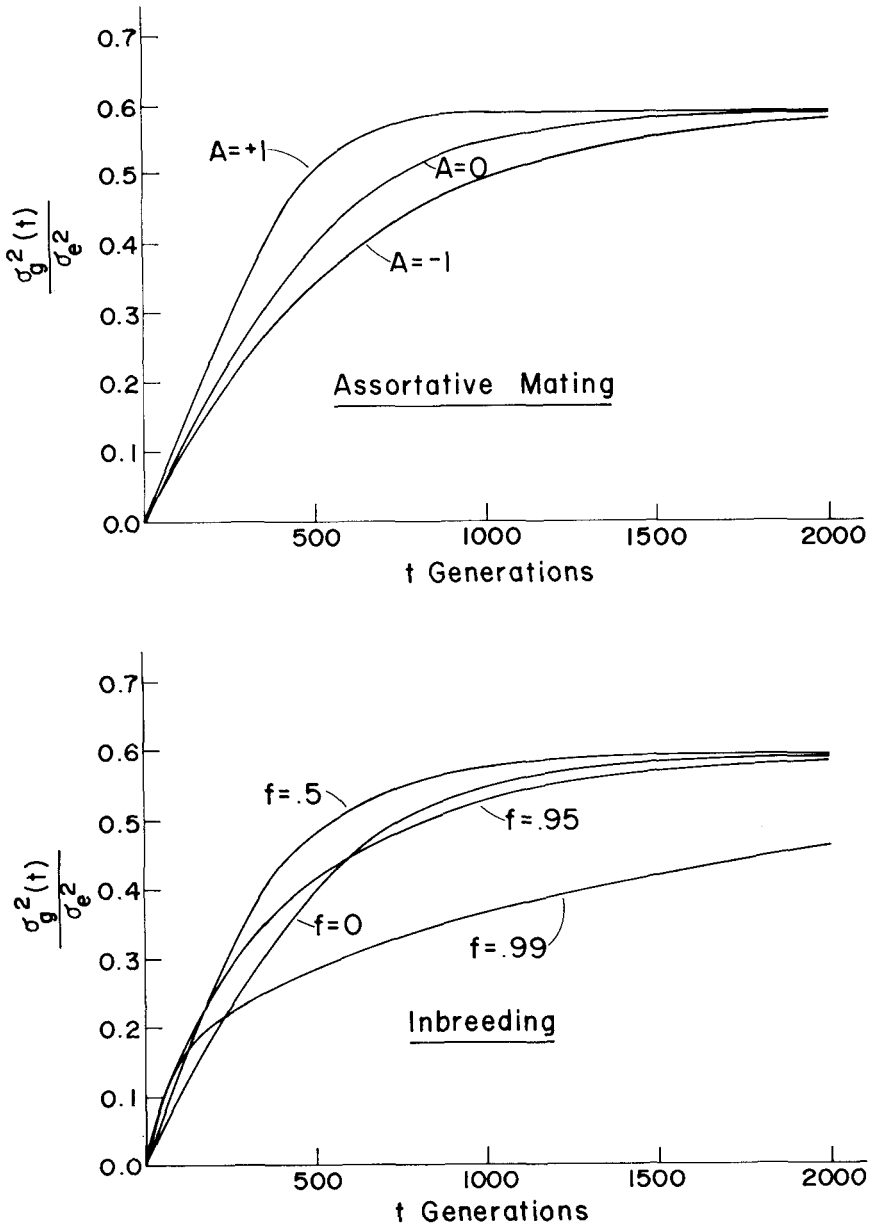

FIGURE 1.—Examples of the approach to equilibrium of the genetic variance, with different degrees of assortative mating and inbreeding. A is the phenotypic correlation between mates and f is the inbreeding coefficient. The genetic variance, $\sigma_g^2(t)$, is plotted relative to the environmental variance, σ_e^2. In both graphs, the character is affected by 10 freely recombining, equally mutable loci with $\sigma_m^2/\sigma_e^2 = 10^{-3}$, which is typical of characters in mice, maize and fruit flies (LANDE 1976a). The phenotype is normally distributed and has a Gaussian fitness function (see equation 10) with $w/\sigma_e = 4$. At equilibrium the heritability is 0.37 and $\hat{k} = 0.09$, which corresponds to a phenotypic load (% selective mortality) of 4.6%.

ing on the rate of approach to equilibrium is more complex, as the variance of allelic effects at each locus is not a monotonic function of the inbreeding coefficient (equation 13d). If linkage is not very tight, low or moderate levels of inbreeding will speed up the rate of approach to equilibrium, while with high levels of inbreeding the rate of approach to the equilibrium is initially faster but ultimately slower than with random mating (Figure 1). In general, the quantitative effect of the mating system on the rate of approach to the equilibrium genetic variance depends on the details of the recombination and mutation rates of the individual loci.

Other aspects of population structure and ecology not treated in these models may exert significant selective pressure on genes controlling the mating system. Positive assortative mating may entail an additional selection against extreme forms, thus reducing the amount of genetic variance maintained in comparison to a random mating population. Inbreeding reduces gene flow from adjacent populations which may be adapted to different environmental conditions, but will increase the genetic load due to deleterious recessive mutations by restricting the local effective population size (CROW and KIMURA 1970). The ability to self-fertilize may be important for colonizing species, as when one seed reaches an island, though STEBBINS (1965) concluded that among the native California flora, no particular type of mating system favors the evolution of weediness.

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APPENDIX

Denoting the allelic affect at locus i in the gametes as x_i with mean $\bar{x}_i$, and letting $\gamma_i = x_i - \bar{x}_i$ be the deviation from the mean, the covariance of allelic effects at loci i and j before mutation is $C_{ij} = E[\gamma_i\gamma_j]$. A mutational change at locus i is written as β_i where $E[\beta_i] = 0$ and $E[\beta_i^2] = m_i^2$. The covariance after mutation for $i \neq j$ is

$$(1-\mu_i)(1-\mu_j)E[\gamma_i\gamma_j] + \mu_i(1-\mu_j)E[(\gamma_i+\beta_i)\gamma_j]$$
$$+ (1-\mu_i)\mu_jE[\gamma_i+\beta_j)] + \mu_i\mu_jE[(\gamma_i+\beta_i)(\gamma_j+\beta_j)] .$$

Because mutation occurs independently at each locus $E[\beta_i\beta_j] = E[\beta_i\gamma_j] = 0$, and this expression then reduces to $E[\gamma_i\gamma_j]$, showing that the covariances, C_{ij} for $i \neq j$, are not altered by mutation. Similarly the variance at locus i before mutation is $C_{ii} = E[\gamma_i^2]$ and after mutation is

$$(1-\mu_i)E[\gamma_i^2] + \mu_i\mathrm{E}[(\gamma_i+\beta_i)^2] .$$

By assumption $E[\gamma_i\beta_i] = 0$ and this expression reduces to $C_{ii} + \mu_i m_i^2$. Thus the change in C_{ij} due to mutation is $\delta_{ij}\mu_i m_i^2$, where $\delta_{ij} = 1$ if $i = j$ and 0 otherwise.