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Author(s): Michael J. Wade
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SOFT SELECTION, HARD SELECTION, KIN SELECTION, AND GROUP SELECTION

MICHAEL J. WADE

Department of Biology, University of Chicago, Chicago, Illinois 60637

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In this paper I illustrate the theoretical relationship among several different models of selection in structured populations, soft selection, hard selection, kin selection, and group selection, by using the covariance formulations of Li (1967) and Price (1970, 1972) to partition the operation of selection into within- and between-group components. This partitioning of covariance can be directly extended from the single-locus population genetic models derived in this paper to the description of selection within and between groups for continuously distributed, quantitative traits of the type presently under investigation in several research programs of kin and group selection. In addition, because of the algebraic relationship between the covariance and the coefficient of linear regression, regression coefficients can be used to evaluate the relative importance of selection within and between groups in these models of subdivided populations.

Price (1970, 1972) was the first to apply the covariance approach to the mathematics of group selection but only for ''the limiting case of reproductively isolated groups with no intergroup migration'' (Price 1972, p. 487). Because of this assumption concerning the complete isolation of groups, his major result (eq. [A17] of Price [1972]) did ''not depend upon any assumptions about mechanisms of heredity or anything else of that sort'' (Price 1972, p. 487). I will show that Price's equations for gene-frequency change represent a special case of a general partitioning of covariance into within- and between-group components and, in addition, that it can be applied to cases with intergroup migration. Furthermore, the algebraic relationship between the covariance and the coefficient of linear regression will be used to illustrate how assumptions concerning hereditary mechanisms are implicit in the covariance formulations. In particular, the equations of Price (1970, 1972) completely describe selection in terms of the change in the mean value of a trait within a generation, but they require a hereditary mechanism in order to predict how much of this within-generation change is transmitted across generations (see below and also Arnold and Wade 1984*a*). The covariance formulation completely describes selection but it does not necessarily describe the response to selection. In the derivation presented below individuals will be assigned character values in such a way that the average value of the character is the

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gene frequency. Because the processes of reproduction and transmission in this model do not change gene frequency (as would be the case with, e.g., meiotic drive), the covariance formulation will not be affected by the hereditary mechanism. When applied to other traits where the average value of the trait is not a gene frequency or where it is affected by transmission and reproduction, the covariance formulation will be adequate only for describing selection and not the response to selection. In recent theoretical discussions of the evolution of altruism by kin selection, Uyenoyama and coworkers (Uyenoyama and Feldman 1981; Uyenoyama et al. 1981) have demonstrated that the regression coefficient of the recipient's additive genotypic value on that of the altruist can provide an exact "Hamilton rule" for describing both the initial conditions for increase in the frequency of altruistic alleles as well as the interior equilibria. By defining M_{ijk} as the frequency of allele k within individuals of genotype i in group j as was done by Cockerham (1973), I will show that a different and somewhat simpler covariance formulation is possible that more clearly elucidates the evolutionary roles of within- and between-group variations in fitness and gene frequency. This approach may not be as useful as that of Uyenoyama, however, for describing interior equilibria.

Lastly, this approach in no way depends upon the specific assumptions made regarding the distribution of fitnesses or the genotypic composition of groups. It can be extended to investigations of more complicated hierarchies of group structure and, as an example, I will apply it to the three-level model of individual, family, and group selection of Wade (1982*a*).

THE GENERAL SINGLE-LOCUS MODEL

Let M_{ijk} be the frequency of allele k within individuals of genotype i in group j. If there are m possible genotypes at the locus in question numbered $i = 1, 2, \ldots, m$, then the genotypic composition of a group is specified by a vector of m genotypic frequencies. If the groups are finite, there are a countable number of possible genotypic configurations which will be numbered $j = 1, 2, \ldots m$. Let G_{ij} be the frequency of genotype i in groups with genotypic composition j and let F_j be the frequency of groups with this composition. The local frequency of allele k in group j, q_{jk}, is then given by $q_{jk} = \Sigma_i G_{ij} M_{ijk} = M_{.jk}$. The dot subscript signifies the average has been taken. It also illustrates that the trait M_{ijk} has been defined in such a way that the average value of the trait in a locality is equal to the local gene frequency. This formulation permits the results of population genetic theory to be compared with the results of the more recent application of quantitative genetic theory to evolution in natural populations. The frequency of allele k in the entire population is given by $q_{.k} = \Sigma_i \Sigma_j F_j G_{ij} M_{ijk} = M_{..k}$. Clearly, in a diploid model, M_{ijk} can only take values of 0, .5, or 1, while in a haploid model it must be either 0 or 1.

Defining W_{ij} as the absolute fitness of genotype i in group j, it follows that the average fitness over all genotypes in group j is $W_{.j} = \Sigma_i G_{ij} W_{ij}$ and the average fitness over the entire population is $W_{..} = \Sigma_j F_j \Sigma_i G_{ij} W_{ij}$. With these definitions, the rate of gene frequency change for allele k is found to be

$$\Delta q_{.k} = \left(\sum_j F_j \sum_i G_{ij} W_{ij} M_{ijk} - M_{..k} W_{..}\right) \Big/ W_{..}. \tag{1}$$

On inspection, equation (1) can be seen to be the covariance between relative fitness, $W^*_{ij} = W_{ij}/W_{..}$, and M_{ijk},

$$\begin{aligned}\Delta q_{.k} &= \sum_j F_j \sum_i G_{ij} W^*_{ij} M_{ijk} - M_{..k}\\ &= \sum_j F_j \sum_i G_{ij}(W^*_{ij} - 1)(M_{ijk} - M_{..k}) \qquad (2)\\ &= \mathrm{Cov}(W^*_{ij}, M_{ijk}).\end{aligned}$$

In a subdivided population, this covariance can always be partitioned into two components. The first is the covariance between the individual's relative fitness within its group j and the individual's frequency of allele k,

$$\begin{aligned}\mathrm{Cov}_j(\tilde{W}_{ij}, M_{ijk}) &= \sum_i G_{ij}(W_{ij} - W_{.j})(M_{ijk} - M_{.jk})/W_{.j}\\ &= \sum_i G_{ij} \tilde{W}_{ij} M_{ijk} - M_{.jk}.\end{aligned} \tag{3}$$

When averaged over all groups after selection we have, $\mathrm{Cov}_.(\tilde{W}_{ij}, M_{ijk}) = \Sigma_j F_j(W_{.j}/W_{..})\mathrm{Cov}_j(\tilde{W}_{ij}, M_{ijk})$, where $\tilde{W}_{ij} = W_{ij}/W_{.j}$ is the local relative fitness.

The second component is the covariance between group mean relative fitness, $\tilde{W}_{.j} = (W_{.j}/W_{..})$, and the average frequency of allele k within groups,

$$\begin{aligned}\mathrm{Cov}(\tilde{W}_{.j}, M_{.jk}) &= \sum_j F_j(W_{.j} - W_{..})(M_{.jk} - M_{..k})/W_{..}\\ &= \sum_j F_j \tilde{W}_{.j} M_{.jk} - M_{..k}.\end{aligned} \tag{4}$$

This partitioning is accomplished by simply adding and subtracting the quantity $(\Sigma_j F_j \tilde{W}_{.j} M_{.jk})$ from equation (1).

Thus,

$$\Delta q_{.k} = \mathrm{Cov}(W^*_{ij}, M_{ijk}) = \mathrm{Cov}\,(\tilde{W}_{ij}, M_{ijk}) + \mathrm{Cov}(\tilde{W}_{.j}, M_{.jk}). \tag{5}$$

These components of covariance are equivalent to the components of gene frequency change within and between families, respectively, derived in the kin selection models of Wade (1980) and Wade and Breden (1981). They can be of the same or of opposite sign. For example, when the average covariance within groups is negative but the covariance between groups is positive, then individual selection within groups operates against the increase in frequency of allele k but between-group selection favors it. Genes that bear this sort of relationship to fitness within and between groups are commonly referred to as "altruistic." If the signs of the covariances were reversed, the allele k would be favored within groups but selected against between groups as Wright (1969) suggested would be the case for genes for interference behaviors or "social parasitism." The signs of these covariances need not be opposing for the partitioning of selection into within- and between-group components to be relevant. Indeed, when selection within and between groups is practiced in the breeding of domesticated animals and plants, the covariances are generally of the same sign (Falconer 1981). Furthermore, the rates of change observed with each type of selection are different and a selection regime that combines the two levels of selection can be significantly more efficient than either level practiced alone (Falconer 1981; Griffing 1977). Although the arguments for and against group selection in evolutionary ecology have focused almost exclusively on those cases in which the components of covariance are of opposite sign, theoretical and empirical studies in quantitative genetics provide no such justification for restricting discussion of the levels of selection to this case.

Because the ratio, $\mathrm{Cov}(X,Y)/V_X$, is by definition the regression coefficient, $b_{Y \cdot X}$, of Y on X, equations (2) and (5) can be further rewritten as

$$\begin{aligned}\Delta q_{jk} &= V_{W^*_{ij}} b_{M_{ijk} \cdot W_{ij}} = V_{\tilde{W}_{ij}} b_{M_{ijk} \cdot \tilde{W}_{ij}} + V_{\tilde{W}_{.j}} b_{M_{.jk} \cdot \tilde{W}_{.j}} \\ &= V_{M_{ijk}} b_{\tilde{W}_{ij} \cdot M_{ijk}} + V_{M_{..k}} b_{\tilde{W}_{.j} \cdot M_{.jk}}\end{aligned} \qquad (6)$$

where $V_{\tilde{W}_{ij}}$ and $V_{M_{ijk}}$ are the variances in relative fitness and gene frequency between individuals within groups and $V_{\tilde{W}_{.j}}$ and $V_{M_{..k}}$ are the variances in group mean relative fitness and group mean gene frequency, respectively.

Note that this general formulation makes no assumptions concerning the process of group formation that determines the distribution, F_j, or the selection regime that determines the selective values, W_{ij}. I will now apply this model and the partitioning of covariance to the models of soft selection, hard selection, kin selection, and group selection.

SOFT SELECTION

In a subdivided population with locally varying viability selection preceding migration, two very different models of evolution, called soft and hard selection (Wallace 1968), have been extensively studied (Levene 1953; Maynard Smith 1962; Deakin 1966; Levins and MacArthur 1966; Prout 1968; Bulmer 1972; Christiansen 1974, 1975; Gillespie and Langley 1974; Gillespie 1974, 1975). The two models differ in the mode of population regulation with regulation occurring locally within subdivisions with soft selection but globally with hard selection. These two different methods of population regulation influence the second covariance term in equation (5) but do so in different ways. (In the discussion to follow, the index j will have a somewhat different meaning than that given above. Here it will refer to a particular group and, because each group is assumed to be large, each will have a unique distribution of genotypes.)

Following Christiansen (1975), assume a population subdivided into N local groups and let $c_j = (c_1, c_2, \ldots, c_j, \ldots, c_N)$ represent the relative sizes of the subpopulations before selection ($\Sigma_j c_j = 1$; $c_. = [1/N]$) and $c_j^* = c_j W_{.j}/W_{..}$ the relative sizes after selection at the time of migration ($\Sigma_j c_j^* = 1$; $c_.^* = [1/N]$).

In the soft selection model, $c_j^* = c_j$ for all j so that local selection does not influence local population size. The c_j^* are the group mean relative fitnesses of the general model and the frequency of each group is $(1/N)$. Equation (5) for soft selection becomes

$$\Delta q_{.k} = \mathrm{Cov}_.(\tilde{W}_{ij}, M_{ijk}) + \mathrm{Cov}(c_j^*, M_{.jk}).$$

However, $\mathrm{Cov}(c_j^*, M_{.jk})$ equals zero in the soft-selection models by assumption. There is no association between the relative size of a local population, c_j, and its gene frequency, $M_{.jk}$. It is clear that although both variation in mean gene frequency and productivity exist among groups, group selection does not occur because there is no covariance between group mean gene frequency ($M_{.jk}$) and group productivity (c_j). For soft selection this covariance is zero by assumption. Even if the within-group genotypic fitnesses, W_{ij}, were frequency dependent, the assumption that $c_j = c_j^*$ would prohibit the operation of group selection.

Maynard Smith (1962) introduced habitat selection into the soft-selection model and proved that it could ". . . appreciably extend the range of values of the fitnesses for which a stable [interior] equilibrium is possible" (p. 61). This modification of the soft-selection model does lead to the association of alleles with the local subpopulation in which they are most fit, but it does not create the nonzero covariance between $M_{.jk}$ and c_j^* necessary for group selection. Indeed, for tractability, Maynard Smith (1962) set all c_j equal to a constant, c, obviating the possibility of differential group reproduction.

HARD SELECTION

In the hard selection model the local selection regime changes the size of the local subpopulation by changing c_j before selection to $c_j W_{.j}/W_{..}$ after selection. Because $W_{.j}$ can be a function of $M_{.jk}$, the between-group covariance term in equation (5) is generally nonzero:

$$\mathrm{Cov}(\tilde{W}_{.j}, M_{.jk}) = \sum_j (W_{.j} - 1)(M_{.jk} - M_{..k})/N \neq 0.$$

It is not possible without further specification of the exact nature of the genotypic fitnesses to determine whether or not the within- and between-group covariance components are of the same or opposite sign. As Wright (1959) emphasized in his discussion of interdemic selection in relation to his shifting balance theory, even when relative selective values within groups are not genotype frequency-dependent, absolute selective value, $W_{.j}$, may still be a function of genotype frequency. Interdemic selection could occur owing to the local differences in mean absolute fitness by means of differential dispersion (Wright 1931, 1978). For this reason, the global fitness of a genotype is not constant but frequency dependent. In this view, it is local individual or mass selection that causes the local differences in mean absolute fitness. Individual selection in Wright's shifting balance theory in this way can provide the between-group variation on which interdemic selection might operate (see also Via and Lande, MS).

This type of interdemic selection requires that three conditions be met: (1) local selection must result in local variation in average absolute fitness, $V_{\tilde{W}_{.j}} > 0$; (2) local subpopulations must vary in average gene frequency, $V_{M_{.jk}} > 0$; and (3) there must exist a covariance between group mean fitness and group mean gene frequency, $\mathrm{Cov}(W_{.j},M_{.jk}) \neq 0$. It is this third condition that is absent under the assumptions of soft selection but which can be present under hard selection.

This examination of the two extreme models of soft and hard selection using the covariance formulation permits the conclusion that local frequency-dependent selection alone does not necessarily imply that group selection will occur (soft selection models) and, conversely, the absence of local frequency-dependent selection does not prevent group selection (hard selection models; Wright 1931, 1959, 1969, 1978, 1980; see also Boyd 1982; Michod 1982).

KIN SELECTION

There are two primary differences between the models of kin selection and soft and hard selection. First, the genetic variance between local groups under models of kin selection generally exceeds that of the soft and hard selection models. This happens as a result of the different ways in which the groups are constituted. The kin selection models postulate that individuals are nonrandomly distributed into groups on the basis of genetic relatedness while the soft and hard selection models usually assume a random distribution of individuals into groups (but see the habitat selection models of Maynard Smith [1962]). The genetic correlation among relatives within groups guarantees a greater genetic variance among kin groups than among randomly constituted groups (see Wilson [1979] and Charlesworth [1979] for recent discussion). Within-group mating can further enhance the between-group genetic variance in both models (Breden and Wade 1981; Michod 1982; Wade and Breden 1981) as can the preferential positive assortment of genotypes resulting from habitat selection in the soft and hard selection models, or from a genic correlation between mates or foundresses in the kin selection models (Wade 1982*b*; Wade and Breden 1981).

The second difference arises as a result of the different methods by which the local absolute genotypic fitnesses are generated. In all of the soft and hard selection models cited above, the genotypic fitnesses are considered a fixed characteristic of the local subpopulation independent of the genotypic composition of the subpopulation's members. In the kin selection models, however, the genotypic fitnesses are explicitly frequency dependent and the fitness of a genotype varies from group to group depending upon the group's genotypic composition (Templeton 1979; Wade 1979; Michod 1980; Wilson 1980; Uyenoyama and Feldman 1981). Most models of kin selection implicitly assume hard selection. Thus, the between-group variance in fitness in the first case can be made large or small depending on whether the postulated ecological variation giving rise to the local selection regimes is large or small. In the kin selection models, however, the members of a group create their own selection regime by their interactions with one another. Here, the between-group variance in mean fitness is large or small depending upon the intensity of the social behaviors that generate selection within the kin groups. Because the variance in group mean fitness is a function of the genotypic composition of kin groups, the covariance between group mean gene frequency and group mean fitness is generally implicit in the initial assumptions of the kin selection models. As was seen above, this covariance may or may not exist under the assumptions of the hard and soft selection models.

A covariance approach to kin selection models has been elaborated by Hamilton (1975) and Uyenoyama and Feldman (1981) but was restricted to the between-group component or the total covariance, respectively. To illustrate the application of the partitioning of covariance to both components of selection and to minimize rederivation of results, I will use the explicitly family-structured models of kin selection (reviewed in Michod [1982]) that have been shown to be the appropriate population genetic framework for deriving the seminal results of Hamilton (1964*a*, 1964*b*) using the classical theoretical approach of population genetics with additive effects.

In Wade (1980), I showed that Δq, the total change in gene frequency of an allele for altruistic behavior under kin selection, could be partitioned into two components,

$$\Delta q = \Delta q_I + \Delta q_G$$

where $\overline{\Delta q_I}$ was the average change in the gene frequency resulting from selection within kin groups and Δq_G was the change in gene frequency resulting from selection between kin groups. These components of gene frequency change are equivalent to the components of covariance given in equation (5). The component of gene frequency change owing to individual selection within kin groups (eq. [3] of Wade [1980]) rewritten in the notation of the present paper is

$$\begin{aligned}\overline{\Delta q_I} &= \sum_j F_j(W_{.j}/W_{..}) \sum_i \left(\tilde{G}_{ij}\tilde{W}_{ij}M_{ijk} - \sum_i G_{ij}M_{ijk}\right)\\ &= \sum_j F_j\tilde{W}_{.j}\left(\sum_i G_{ij}[\tilde{W}_{ij} - 1][M_{ijk} - M_{.jk}]\right)\\ &= \sum_j F_j\tilde{W}_{.j}\mathrm{Cov}_j(\tilde{W}_{ij},M_{ijk}) = \mathrm{Cov}_{.}(\tilde{W}_{ij},M_{ijk}).\end{aligned}$$

Similarly, the component of gene frequency change caused by selection between kin groups, Δq_G, given in equation (5) of Wade (1980) can be seen to equal the between-group component of covariance

$$\begin{aligned}\Delta q_G &= \sum_j F_j\tilde{W}_{.j}M_{.jk} - M_{..k}\\ &= \sum_j F_j(\tilde{W}_{.j} - 1)(M_{.jk} - M_{..k}) = \mathrm{Cov}(\tilde{W}_{.j},M_{.jk}).\end{aligned}$$

By setting the $\mathrm{Cov}(\tilde{W}_{.j},M_{.jk}) > \mathrm{Cov}_{.}(\tilde{W}_{ij},M_{ijk})$ and specifying the distribution of kin group types, F_j, and the fitness effects of the genotypic interactions, W_{ij}, all of the results of Wade (1978, 1979, 1980, 1982*a*, 1982*b*), Wade and Breden (1981), and Breden and Wade (1981) can be rederived.

GROUP SELECTION

The application of the covariance approach to the problem of group selection was first carried out by Price (1970, 1972). He showed that, for completely isolated groups, the rate of change of gene frequency, p, is given by (Price 1972, eq. [A 17])

$$\Delta P = \mathrm{ave}_{n'}[\mathrm{cov}(\tilde{z},q)] + \mathrm{cov}_n(\tilde{s},p)$$

which, in the notation of this paper, is

$$\Delta p = \mathrm{cov}_{.}(\tilde{W}_{ij},M_{ijk}) + \mathrm{cov}(\tilde{W}_{.j},M_{.jk}).$$

In order to demonstrate the more general application of this formula to the problem of group selection with migration between groups, I will apply the results to two recent models of group selection: (1) D. S. Wilson's haploid trait-group model for the evolution of altruism (Wilson 1980); and (2) a model for the evolution of interference competition involving the three levels of individual, family, and group selection (Wade 1982*b*).

In his model of trait-group selection, Wilson (1979, 1980, p. 23; see also Charlesworth 1979) defines the fitness of a haploid altruistic type, A, in a group of size N with a frequency, p_j, of the altruistic type to be

$$W_{Aj} = d + (Np_j - 1)r$$

where d is the cost to the individual of performing the altruistic behavior and r is the benefit received by every other member of the group from each altruist. The fitness of a nonaltruistic B-type in this group is given as

$$W_{Bj} = Np_jr.$$

After selection, the frequency of A types in this group is

$$P_j' = p_jW_{Aj}/W_{.j}$$

where $W_{.j} = [p_jW_{Aj} + (1 - p_j)W_{Bj}] = W_{Bj} + p_j\,(W_{Aj} - W_{Bj})$ is the average fitness within the group. Letting F_j represent the frequency of groups with Np_j altruists and $W_{..} = \Sigma_jF_jW_{.j}$, the overall gene frequency after selection is given by

$$p' = \sum_j F_j(W_{.j}/W_{..})p_j'.$$

The conditions for the average gene frequency of the entire population after selection, p', to exceed that before selection, $p = \Sigma_jF_jp_j$, were shown by Wilson to depend critically upon the between-group variance in gene frequency with respect to the distribution before selection, s_p^2.

The partitioning of covariance can be applied to Wilson's model as follows. The change in gene frequency from one generation to the next is, by definition

$$\begin{aligned}\Delta p = p' - p = \sum_j F_j(W_{.j}/W_{..})p_j' - \sum_j F_j(W_{.j}/W_{..})p_j \\ + \sum_j F_j(W_{.j}/W_{..})p_j - \sum_j F_j p_j,\end{aligned} \tag{7}$$

using the definitions of p' and p and adding and subtracting the term $\Sigma_j F_j(W_{.j}/W_{..})p_j$. The covariance between relative individual fitness and individual gene frequency within a group with Np_j altruists is given by

$$\begin{aligned}\mathrm{Cov}_j(W_{Aj}/W_{.j}, M_{ij}) = p_j[(W_{Aj}/W_{.j}) - 1](M_{Aj} - p_j) \\ + (1 - p_j)[(W_{Bj}/W_{.j}) - 1](M_{Bj} - p_j).\end{aligned}$$

Noting that $M_{Aj} = 1$ and $M_{Bj} = 0$ for this haploid model, this equation reduces to

$$\mathrm{Cov}_j(W_{Aj}/W_{.j},M_{ij}) = [p_j(W_{Aj} - W_{Bj}) - p_j^2(W_{Aj} - W_{Bj})]/W_{.j}.$$

Substituting $(W_{.j} - W_{Bj})$ for $p_j(Wa_{Aj} - W_{Bj})$ in the second term in the numerator, we find

$$\mathrm{Cov}_j(W_{Aj}/W_{.j},M_{ij}) = p_j W_{Aj}/W_{.j} - p_j.$$

The average covariance between individual relative fitness and individual gene frequency is obtained by summing over all groups (see eq. [3])

$$\begin{aligned}\mathrm{Cov}_.(W_{Aj}/W_{.j},M_{ij}) &= \sum_j F_j \mathrm{Cov}_j(W_{Aj},M_{ij}) \\ &= \sum_j F_j(W_{.j}/W_{..})p_j(W_{Aj}/W_{.j}) - \sum_j F_j(W_{.j}/W_{..})p_j.\end{aligned}$$

This expression for Cov. is equal to the first two terms on the r.h.s. of equation (7).

The covariance between group mean relative fitness, $W_{.j}/W_{..}$, and group mean gene frequency, p_j, is found using equation (4)

$$\begin{aligned}\mathrm{Cov}(W_{.j}/W_{..},p_j) &= \sum_j F_j[(W_{.j}/W_{..}) - 1](p_j - p) \\ &= \sum_j F_j(W_{.j}/W_{..})p_j - \sum_j F_j p_j.\end{aligned}$$

This between-group covariance term is equal to the last two terms in expression (7). Thus, in the Wilson trait-group model

$$\Delta p = \mathrm{Cov}_.(W_{Aj}/W_{.j},M_{ij}) + \mathrm{Cov}(W_{.j}/W_{..},p_j).$$

In a group-selection model investigating the evolution of social interactions in a population with three levels of biological organization (Wade 1982*b*), I partitioned the total change in gene frequency, Δq, into the sum of three components: (1) $\overline{\Delta q_I}$, selection between individuals within families averaged over all families and over all groups; (2) $\overline{\Delta q_f}$, selection between families within groups averaged over all groups; and, (3) Δq_G, selection between groups of families. In that paper I defined G_{ijk} as the frequency of genotype i in family j in group k and W_{ijk} as the fitness of that genotype. P_{jk} was the frequency of family j in group k and F_k was the frequency of groups of type k. M_{ijkl} is the frequency of allele I in individuals of genotype i in family j in group k. The total gene frequency change (eq. [5] of Wade 1982) is

$$\Delta q_I = \left(\sum_k F_k\left[\sum_j P_{jk} \sum_i G_{ijk} W_{ijk} M_{ijkl}\right]\right) - W_{...}\left(\sum_k F_k M_{..kl}\right)/W_{...}.$$

By adding and subtracting the quantities $[\Sigma_k F_k(\Sigma_j P_{jk} \tilde{W}_{.jk} M_{.jkl})]$ and $(\Sigma_k F_k \tilde{W}_{..k} M_{..kl})$, it follows that

$$\begin{aligned}\Delta q_I &= \mathrm{Cov}(W_{ijk}/W_{...}, M_{ijkl}) = \mathrm{Cov}_{..}(W_{ijk}/W_{.jk}, M_{ijkl}) \\ &\quad + \mathrm{Cov}_{.}(W_{.jk}/W_{..k}, M_{.jkl}) + \mathrm{Cov}(W_{..k}/W_{...}, M_{..kl}).\end{aligned}$$

That is, the total covariance between individual gene frequency, M_{ijkl}, and global individual relative fitness, $W_{ijk}/W_{...}$, can be formally partitioned into the sum of three components: (1) $\mathrm{Cov}_{..}$, the covariance within families between individual relative fitness, $W_{ijk}/W_{.jk}$, and individual gene frequency, M_{ijkl}, averaged over all families and all groups after selection; (2) $\mathrm{Cov}_{.}$, the covariance between family mean relative fitness, $W_{.jk}/W_{..k}$, and family gene frequency, $M_{.jk}$, averaged over all groups after selection; and (3) Cov, the covariance between group mean relative fitness, $W_{..k}/W_{...}$, and group mean gene frequency, $M_{..kl}$. Each covariance term here is equivalent to the respective component of gene frequency change in Wade (1982*b*; see also Wade 1984).

DISCUSSION

The conceptual relationships between the different kinds of selection in structured populations, soft, hard, kin, and group, have been discussed at least in qualitative terms by Hamilton (1975), Maynard Smith (1976), and Wilson (1979, 1980). Some authors choose to emphasize the differences between the various types (e.g. Maynard Smith) while others (Hamilton and Wilson) have stressed that population structure can be considered a continuum with the different selection types representing regions of that continuum. The theoretical development presented here illustrates the nature of selection within and between groups for each of these types of selection. In so doing, the similarities and differences between the selection models in their assumptions concerning the distribution of genotypic fitnesses and the distribution of genotype frequencies within and between local groups have been made more explicit. The unique assumptions of each model exert their influence on evolution by changing the relative signs and magnitudes of the components of covariance. Using the regression formulas (eqs. [6]), we see that the components of covariance change because of changes in the genetic variances and fitness variances within and between groups.

Michod (1982) in his discussion of group selection adopts the Uyenoyama and Feldman (1981) definition of a group as the smallest collection of individuals within a population such that the genotypic fitnesses within each group are not a function of the composition of any other group. The groups defined by the local selection regimes in the hard selection models conform to this definition. In addition, gene frequency changes do occur because of the differential productivity of these groups as shown above. Processes of gene-frequency change of this sort are traditionally viewed as group selection.

Only a single locus was treated in this paper but the extension to multiple loci is straightforward (Wade, in prep). However, even without invoking epistasis for fitness between loci, some of the important features of Sewall Wright's shifting balance theory of evolution are manifest in the one-locus model. In particular, in the hard selection models, it is clear that even when local genotypic fitnesses are not frequency dependent, the average absolute fitness of a local deme can often be frequency dependent and, thus, the global genotypic fitnesses are frequency dependent. These local differences in absolute fitness are brought about by individual or mass selection within groups. They can result in a component of covariance between local mean relative fitness and local gene frequency (for further discussion of this point see Wright [1959, 1978]). One possible consequence of these differences in local average absolute fitness is the differential dispersion of individuals from groups with high values of average absolute fitness into groups with low values. Such a covariance between local gene frequency and local average absolute fitness is the mechanism for interdemic selection in Wright's theory. These simple one-locus models thus provide an elementary example of Wright's shifting balance theory: local mass selection creates between-group variation in average absolute fitness that then gives rise to interdemic selection by differential dispersion of individuals from localities with high values of average absolute fitness. Because the local variations in mean absolute fitness covary with local variations in gene frequency, this differential dispersion results in a between-group component of gene-frequency change. In Wright's theory as opposed to most later discussions of group selection, local mass selection and interdemic selection were not considered to be opposing forces but rather complementary ones. Thus, it is not necessary that the within- and between-group components of covariance have opposing signs. In a multilocus model with epistasis for fitness, the combination of local mass selection and interdemic selection could lead to the spread of the gene combination with the most favorable effect on global fitness. In contrast, local selection alone could locally maximize mean fitness but would not provide a way to select among the different local maxima.

In the covariance derivations of Price (1970, 1972), both individuals and groups were implicitly assumed to reproduce themselves exactly. That is, a particular model for the inheritance of individual phenotypes and for the inheritance of group mean phenotypes was made by assumption. Without invoking a mechanism of heredity, the covariance formulation of Price is a very general description of the change in the value of the mean phenotype within a generation. However, to determine how much of that change produced by selection will be propagated across generations the assumption of an hereditary mechanism is necessary (Arnold and Wade 1984*a*, 1984*b*). The role of heredity is best brought out by the regression formulas (eqq. [6]) which permit comparison of the additive diploid and haploid trait-group models (Wilson 1980) (or, if desired, the diploid and haploid soft and hard selection models [Gliddon and Strobeck 1975]). In the haploid model the average within-group genic variance is equal to $q(1 - q) - s_p^2$ but in the diploid model it is equal to $p(1 - p) - s_p^2 - G_2$, where G_2 is the average frequency of heterozygotes.

CONCLUSION AND SUMMARY

The models of soft selection, hard selection, kin selection, and group selection can be represented as variations of a common general model that expresses the total gene frequency change, itself a covariance, as the sum of two covariance components: (1) the covariance within groups between individual relative fitness and individual gene frequency averaged over all groups; and (2) the covariance between group mean relative fitness and group mean gene frequency. The general model is a formal partitioning of covariance that makes no assumptions concerning the distribution of fitnesses among genotypes or the distributions of genotypes within and among groups. The different models of selection change these components of covariance by their assumptions. The general model was used to examine the models of Wilson's trait-group selection model, the family-structured kin selection models, and a group selection model involving three levels of biological organization, and to illustrate the approach. The relationship of the hard and soft selection models to Wright's shifting balance theory of evolution was also discussed.

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