

## On Indirect Genetic Effects in Structured Populations

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**ABSTRACT:** Indirect genetic effects (IGEs) occur when the phenotype of an individual, and possibly its fitness, depends, at least in part, on the genes of its social partners. The effective result is that environmental sources of phenotypic variance can themselves evolve. Simple models have shown that IGEs can alter the rate and direction of evolution for traits involved in interactions. Here we expand the applicability of the theory of IGEs to evolution in metapopulations by including nonlinear interactions between individuals and population genetic structure. Although population subdivision alone generates some dramatic and nonintuitive evolutionary dynamics for interacting phenotypes, the combination of nonlinear interactions with subdivision reveals an even greater importance of IGEs. The presence of genetic structure links the evolution of interacting phenotypes and the traits that influence their expression ("effector traits") even in the absence of genetic correlations. When nonlinear social effects occur in subdivided populations, evolutionary response is altered and can even oppose the direction expected due to direct selection. Because population genetic structure allows for multilevel selection, we also investigate the role of IGEs in determining the response to individual and group selection. We find that nonlinear social effects can cause interference between levels of selection even when they act in the same direction. In some cases, interference can be so extreme that the actual evolutionary response to multilevel selection is opposite in direction to that predicted by summing selection at each level. This theoretical result confirms empirical data that show higher levels of selection cannot be ignored even when selection acts in the same direction at all levels.

**Keywords:** population subdivision, indirect genetic effects, group selection, evolutionary rate.

The traditional partitioning of causal influences on phenotypic expression into genetic and environmental factors

(e.g., Falconer and Mackay 1996; Lynch and Walsh 1998) ignores the possibility that some influences are simultaneously genetic and environmental. Such is the case when complex phenotypes are determined, at least in part, by the social environment that an individual experiences. Whether this environment is a direct behavioral interaction among individuals in a dominance hierarchy or the competitive environment generated by a stand of neighboring plants, interactions among conspecifics can have profound influences on the expression of phenotypic traits and, thereby, on the evolutionary process (Waddington 1942; Wolf et al. 1998). Because genetic factors play a role in determining the environment provided by conspecifics, causal pathways exist linking genes in one individual with phenotypic traits expressed by other, even unrelated, individuals (Moore et al. 1997; Wolf et al. 1998). These "indirect genetic effects," or IGEs, contribute to the inheritance of complex phenotypes and to the coevolution of phenotypes and environment.

Interacting phenotypes are characters that require or are influenced by interactions with conspecifics (Moore et al. 1997 and references therein). Though many different types of traits are affected by such interactions (see "Discussion"), interacting phenotypes have mainly been studied in the contexts of social evolution and kin selection. Though we have learned much about the evolution of social behaviors from optimality-oriented (Maynard Smith 1982; Grafen 1991) and selection-oriented (e.g., Hamilton 1964*a*, 1964*b*; West-Eberhard 1979, 1983) models, these approaches often do not explicitly consider genetics. Consequently, such models tell us little about the rate and trajectory of evolution in multivariate phenotypic space. In contrast, the IGE approach (Moore et al. 1997; Wolf et al. 1998), as with other studies based on Price's equation (Lynch 1987; Kirkpatrick and Lande 1989; Queller 1992*a*, 1992*b*; Cheverud and Moore 1994; Frank 1997, 1998), separates the role of genetics from the role of selection in social evolution. Indirect genetic effects models focus first on how the phenotype is generated from an individual's own genotype as well as the genotype of its social partner(s) and second on how this phenotype influences fitness. As discussed elsewhere (Moore et al. 1997; Wolf et al. 1998, 1999), this approach provides a framework for

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the empirical study of social evolution based on the tools of quantitative genetics (Lynch and Walsh 1998) and phenotypic selection analysis (Lande and Arnold 1983; Wade and Kalisz 1990). Traditional approaches (e.g., Hamilton 1964*a*, 1964*b*; Michod 1982) that map genotype directly onto fitness make empirical investigation difficult.

The importance of IGEs in evolutionary processes comes about from two main sources (Wolf et al. 1998). First, IGEs act as an environmental component of variation that itself evolves, allowing concomitant evolutionary change with the interacting phenotypes they affect. Second, the existence of IGEs alters the genotype-phenotype relationship, such that, in order to predict a focal individual's phenotype, it is necessary to know not only that individual's genotype but also the genotypes of individuals with which the focal individual interacts. Like the coevolution of traits in different species (Gomulkiewicz et al. 2000), the specific evolutionary consequences of IGEs depend on the sign, magnitude, and reciprocity of the interactions among individuals and traits. Generally, IGEs are expected to alter the rate of evolutionary change for both interacting phenotypes and the traits that influence them and, under certain circumstances, allow for evolutionary change in characters that have no direct additive genetic variance (Griffing 1977, 1981; Moore et al. 1997, 1998; Wolf et al. 1998; Wolf 2000).

Prior models of IGEs among unrelated individuals make the standard assumptions of a large panmictic population with random interactions among individuals and purely linear effects of the interactions among individuals (Moore et al. 1997). Although these assumptions may be reasonable approximations for some systems, they clearly fail to encompass many of the kinds of biological interactions that generate IGEs (Wolf 2000). If individuals are distributed into social groups or physical neighborhoods, then population subdivision exists, and interactions may be more likely to occur within demes than among them (Wade 1996). Such population structure is a biological reality for many, perhaps most, organisms (Loveless and Hamrick 1984; Avise et al. 1987; Whitlock 1992; Hanski and Gilpin 1997; Kelly 1997; Wade and Goodnight 1998) and allows for selection to act at multiple levels. Other approaches to the study of social evolution have shown that population structure can be important (e.g., the evolution of cooperation in viscous populations [Taylor 1992; Queller 1994*a*, 1994*b*; van Baalen and Rand 1998]). Wade (1982, 1985) showed the relationship between genetic subdivision,  $F_{ST}$ , and relatedness,  $r$ , demonstrating that population structure provides another way to satisfy Hamilton's rule.

Also, different individuals may respond differently in the same social setting. Many animal contests undoubtedly involve decisions based on comparisons among individuals

for traits such as body size and display rate. This sort of relative rule of interaction (e.g., "if bigger do  $x$ , if smaller do  $y$ ") introduces a nonlinear component to the effects of interaction among individuals. Although important in the outcome of any specific interaction, the contribution of such nonlinearities to evolutionary response in panmictic populations is limited (Wade and Goodnight 1998) for two reasons. First, in large homogeneous populations, the positive and negative effects of nonlinear interactions tend to average out across the population. Second, under panmixis, the particular social environment experienced by an individual is not the same as that experienced by its parents. The effects of nonlinear interactions are most important when populations are genetically structured because individuals tend to experience a local social context (rather than the average global social environment) and this local social context is experienced similarly by an individual and its parents. When nonlinear effects have been built into models of altruism between relatives, Hamilton's rule no longer holds true (e.g., Charlesworth 1978; Uyenoyama and Feldman 1982; Queller 1992*b*; Frank 1998).

We extend the applicability of models of the evolution of interacting phenotypes by including nonlinear effects of interactions among individuals and population structure. We evaluate the importance of these elements in the presence of individual and group selection on interacting phenotypes. We find that population subdivision generally inflates the role of IGEs in evolution. The existence of IGEs not only can alter the response to the individual selection in the absence of intergroup selection but also can generate interference among the levels of selection, even when they operate in the same direction. Finally, IGEs generate different responses to two different forms of individual selection (i.e., hard vs. soft) in structured populations.

### The Model

We consider the evolution of two traits,  $z_1$  and  $z_2$ . The first trait,  $z_1$ , is a standard quantitative trait, by which we mean that the value of  $z_1$  for a given individual depends only on that individual's additive genetic value for the trait,  $a_1$ , as well as its environmental (residual) effect,  $e_1$ :

$$z_1 = \alpha_1 a_1 + e_1. \quad (1a)$$

The parameter  $\alpha_1$  measures the effect of the additive genetic value on the phenotype. When the trait is a simple linear function of its additive genetic value and does not depend on interactions with other individuals (as is true for  $z_1$ ),  $\alpha_1$  can be assumed to equal unity with no loss of generality. In this case, equation (1a) collapses to the traditional equation

$$z_1 = a_1 + e_1. \quad (1b)$$

Throughout this article, we use equation (1b) to describe  $z_1$ , the “effector trait” or “effector phenotype.”

Unlike  $z_1$ , the value of the second trait,  $z_2$  (the “interacting phenotype”), is affected by the phenotypes of its social partners (Moore et al. 1997). In particular,  $z_2$  is affected by the value of  $z_1$  in its social partners. We assume that the individual interacts with many other individuals from within its deme but never with individuals from outside of its deme. Thus,  $z_2$  is a function of the average value of  $z_1$  within the deme,  $\bar{z}_1$ . The value of the second trait is defined by

$$z_2 = \alpha_2 a_2 + \psi_{21} \bar{z}_1 + \xi_{21} a_2 \bar{z}_1 + e_2. \quad (2a)$$

The first term describes the linear effect of an individual’s additive genetic value on its own phenotype. Note that this additive genetic value does not represent the breeding value in the usual sense but rather an underlying additive basis to the direct genetic effect (Moore et al. 1997). The second term describes the linear effect of the social partners’ phenotypes within the deme. The coefficient  $\psi_{21}$  weights the importance of  $z_1$  in local social partners on the value of  $z_2$  in the focal individual. The third term in equation (2a) describes the nonlinear interaction between the genotype of the focal individual and the phenotype of its local social partners. If the global means of  $z_1$  and  $z_2$  are both 0, then the third term represents a pure interaction effect in the statistical sense. When both global means are not 0, this term contains both linear and interaction effects. The fourth term represents the environmental deviation of this trait.

Unlike linear models, nonlinear models cannot always be rescaled such that the global means of  $z_1$  and  $z_2$  are both 0 while holding the parameters  $\alpha$ ,  $\psi$ , and  $\xi$  constant. For tracking evolution over multiple generations, there are two solutions. First, these global means can be rescaled to 0 every generation, but the value of the parameters  $\alpha$ ,  $\psi$ , and  $\xi$  will no longer be constants and must themselves be reevaluated each generation. Alternatively, the parameters  $\alpha$ ,  $\psi$ , and  $\xi$  can be held constant, but the appropriate global mean values for each generation must be incorporated into the equations for evolutionary change. In the equations that follow, we assume that all the global means are initially 0, so these simplified equations only apply to the first generation of change. In this case,  $\alpha$  and  $\psi$  can be simply interpreted as coefficients of linear effects, while  $\xi$  is the coefficient of the pure interaction effect. The appendix provides more complete versions of all the relevant equations without making any assumptions about mean values.

To investigate the evolutionary interaction between the

effector and interacting phenotypes, we begin by substituting equation (1b) into equation (2a):

$$z_2 = \alpha_2 a_2 + \psi_{21} \bar{a}_1 + \xi_{21} a_2 (\bar{a}_1) + \psi_{21} \bar{e}_1 + \xi_{21} a_2 \bar{e}_1 + e_2. \quad (2b)$$

The second and third terms of this expansion show that  $z_2$  is affected indirectly by genes in its social partners (i.e., IGEs exist in this model). These two terms will be responsible for all of the interesting evolutionary results of IGEs.

### Variance in Structured Populations

The variance among demes in a heritable social context determines, in large part, the special evolutionary effects of IGEs. Population structure influences the partitioning of genetic variance within and among demes. We assume that the genes contributing to the intrinsic additive genetic values,  $a_1$  and  $a_2$ , are purely additive at the physiological level (sensu Cheverud and Routman 1995). In this case, the average variance for additive genetic values within demes is (Hartl and Clark 1997)

$$V_{wi}(a) = (1 - F_{ST})V_{F_0}(a), \quad (3)$$

where  $F_{ST}$  is Wright’s measure of population subdivision and  $V_{F_0}(a)$  is the total variance in  $a$  in a hypothetical population with no subdivision (i.e.,  $F_{ST} = 0$ ). The variance in  $a$  among populations is (Hartl and Clark 1997)

$$V_{am}(a) = 2F_{ST}V_{F_0}(a). \quad (4)$$

### Evolutionary Response to Selection

We define the relative fitness of an individual as

$$w = \beta_{11} z_1 + \beta_{12} z_2 + \beta_{G1} \bar{z}_1 + \beta_{G2} \bar{z}_2 + k. \quad (5)$$

The first two terms show that the fitness of an individual is a linear function of its own values for traits 1 and 2 (individual-level selection components,  $\beta_i$ ’s). The second two terms show that the fitness of an individual is also a linear function of the average value of these two traits in its deme (group-level selection components,  $\beta_G$ ’s). The  $\beta$ ’s quantify the strength of directional selection on each component (Lande and Arnold 1983). For example, setting  $\beta_{G1}$  and  $\beta_{G2}$  equal to 0 indicates that selection acts only on individual phenotypes and not on the group mean trait values. The last term ( $k$ ) is a constant.

Because we make the common assumption that the abi-

otic environment remains constant across generations, the change in the global mean phenotype across a generation depends only on the expected changes in genetic values (Price 1972; Frank 1998)

$$\Delta \bar{\bar{z}}_1 = E(\Delta a_1) \quad (6)$$

and

$$\begin{aligned} \Delta \bar{\bar{z}}_2 = & \alpha_2 E(\Delta a_2) + \psi_{21} E(\Delta \bar{a}_1) + \xi_{21} E(\Delta a_2) E(\Delta \bar{a}_1) \\ & + \xi_{21} \Delta C(\bar{a}_1, a_2). \end{aligned} \quad (7)$$

The double bar symbols (e.g.,  $\bar{\bar{z}}$ ) refer to the global means over the entire metapopulation, averaging first within demes and then over all demes. The variable  $E(\Delta a)$  is the expected change in individual genetic values, while  $E(\Delta \bar{a})$  is the expected change in deme average genetic values. Although the distinction between individual and deme values is made in equation (7) for clarity,  $E(\Delta a) = E(\Delta \bar{a})$ . The final term in equation (7) equals the change in the among-deme covariance of  $a_1$  and  $a_2$ . The evolution of variances and covariances is not well understood (e.g., Turelli 1988, 1990), and these second moments are often assumed to be constant (e.g., Lande 1979). In panmictic populations, second moments can be assumed to be constant when selection is weak relative to recombination (Lande 1979). Similarly, in structured populations, second moments as well as  $F_{ST}$  can be assumed constant when selection is weak relative to the joint action of recombination and migration. We will assume that there is no change in the among-deme covariance. Assuming that selection is the only evolutionary force, we can calculate the changes in the means of the genetic terms by taking the covariance of each genetic term with relative fitness (Price 1972; Frank 1997, 1998):

$$E(\Delta a) = C(a, w). \quad (8)$$

It is important to note that these changes only apply to changes within a generation. It is only through the assumption of a totally additive genetic system that these same equations can be used to describe changes across generations. To evaluate these covariances, we need to describe fitness as a function of genetic values. Substituting equations (1b) and (2b) into equation (5) and ignoring the environmental terms that will not contribute to the covariances of interest, we find

$$\begin{aligned} w = & \beta_{11} a_1 + \beta_{12} \alpha_2 a_2 + (\beta_{12} \psi_{21} + \beta_{G1} + \beta_{G2} \psi_{21}) \bar{a}_1 \\ & + \beta_{G2} \bar{a}_2 + \beta_{12} \xi_{21} a_2 \bar{a}_1 + \beta_{G2} \xi_{21} \bar{a}_2 \bar{a}_1 + k. \end{aligned} \quad (9)$$

Using equations (3), (4), (8), and (9), and assuming that  $a_1$  and  $a_2$  are bivariate normally distributed, we can evaluate the expected changes in genetic values as

$$\begin{aligned} E(\Delta a_1) = & [\beta_{11}(1 + F_{ST}) \\ & + 2(\beta_{12} \psi_{21} + \beta_{G1} + \beta_{G2} \psi_{21}) F_{ST}] \\ & \times V_{F_0}(a_1) + \beta_{12} \alpha_2 C_{wi}(a_1, a_2) \\ & + (\beta_{12} + \beta_{G2}) \alpha_2 C_{am}(a_1, a_2) \end{aligned} \quad (10)$$

and

$$\begin{aligned} E(\Delta a_2) = & [\beta_{12}(1 + F_{ST}) + 2\beta_{G2} F_{ST}] \\ & \times \alpha_2 V_{F_0}(a_2) + \beta_{11} C_{wi}(a_1, a_2) \\ & + (\beta_{11} + \beta_{12} \psi_{21} + \beta_{G1} + \beta_{G2} \psi_{21}) \\ & \times C_{am}(a_1, a_2). \end{aligned} \quad (11)$$

As with most quantitative genetic models, our response to selection equations strictly apply only to short-term evolution as  $F_{ST}$  and genetic (co)variances may change over longer evolutionary periods.

Indirect genetic effects affect evolution primarily in two ways. First, they create new sources of covariance between genetic factors and fitness and thus contribute to selection response within a generation. All the terms in equations (10) and (11) with the coefficient  $\psi_{21}$  contribute to this element of change. Second, IGEs affect how the individual phenotypes are constructed in the following generation (via the terms in eq. [7] containing the coefficients  $\psi_{21}$  or  $\xi_{21}$ ). Note the importance of population structure on IGEs. All of the terms in equations (11) and (12) with the coefficient  $\psi_{21}$  are weighted explicitly or implicitly by  $F_{ST}$  (the covariance of  $a_1$  and  $a_2$  among demes implicitly includes  $F_{ST}$  because  $C_{am}[a_1, a_2] = 2\rho_{am} F_{ST} [V_{F_0}(a_1)V_{F_0}(a_2)]^{1/2}$ , where  $\rho_{am}$  is the correlation of  $a_1$  and  $a_2$  among demes). This dependence on  $F_{ST}$  is the reason that population structure augments the evolutionary importance of IGEs in natural populations.

### *Evolution of Interacting Phenotypes under Individual Selection*

We now explore evolutionary change in the interacting phenotype,  $z_2$ , when selection acts only on this trait. We begin with the simplest case and gradually relax assumptions to gain insight into how IGEs affect evolution in structured populations. We assume throughout that  $a_1$  and  $a_2$  are bivariate normally distributed so that third moments are 0 and fourth moments can be expressed in terms of second moments. Consider the case in which selection acts

only at the individual level on  $z_2$ : there is no population structure, and the effect of social partners on the interacting phenotype is linear. We will call this list of assumptions “condition A,” which is summarized as

$$\text{condition A: } \beta_{12} \neq 0, \beta_{G2} = 0, F_{ST} = 0, \xi_{21} = 0.$$

Under condition A, the response to selection is

$$\Delta \bar{z}_2|_A = \beta_{12} \alpha_2^2 V_{F_0}(a_2) + \beta_{12} \alpha_2 \psi_{21} C_{wi}(a_1, a_2). \quad (12)$$

For this simplest case, we recover the result of Moore et al. (1997; see their eq. [8]): the change in the mean of the interacting trait depends on its correlation with the effector trait weighted by the social effect coefficient.

When we allow for nonlinear forms of social interactions, we have condition B:

$$\text{condition B: } \beta_{12} \neq 0, \beta_{G2} = 0, F_{ST} = 0, \xi_{21} \neq 0.$$

The response to selection is now changed to

$$\Delta \bar{z}_2|_B = \Delta \bar{z}_2|_A + \beta_{12}^2 \alpha_2^2 \xi_{21} V_{F_0}(a_2) C_{wi}(a_1, a_2). \quad (13)$$

The second term in the above equation shows the added part of the response that is owing to the nonlinear component of the social interaction.

If we again assume that the effect of social partners is purely linear but now allow for population structure, we have condition C:

$$\text{condition C: } \beta_{12} \neq 0, \beta_{G2} = 0, F_{ST} \neq 0, \xi_{21} = 0.$$

The response to selection under condition C is

$$\begin{aligned} \Delta \bar{z}_2|_C &= \Delta \bar{z}_2|_A + \beta_{12} \alpha_2 \\ &\times [\alpha_2 F_{ST} V_{F_0}(a_2) + \psi_{21} C_{am}(a_1, a_2)] \\ &+ \beta_{12} \psi_{21} [2\psi_{21} F_{ST} V_{F_0}(a_1) + \alpha_2 C_{am}(a_1, a_2)]. \end{aligned} \quad (14)$$

The second two terms show the added part of the response that is owing to population structure. Recall that all of those components of this equation that include the coefficient  $\psi_{21}$  indicate ways in which IGEs contribute to the evolutionary response. Note that equation (12) shows that when there is no population structure (i.e.,  $F_{ST} = 0$ ), IGEs can contribute to the response to selection on trait 2 only when there is a correlation between  $a_1$  and  $a_2$ . In contrast, equation (14) demonstrates that IGEs contribute to the response even in the absence of a correlation between  $a_1$  and  $a_2$ , as long as there is some degree of population structure (i.e.,  $F_{ST} > 0$ ).

We now allow for nonlinear forms of social interactions in structured populations:

$$\text{condition D: } \beta_{12} \neq 0, \beta_{G2} = 0, F_{ST} \neq 0, \xi_{21} \neq 0.$$

The response to selection is given by

$$\begin{aligned} \Delta \bar{z}_2|_D &= \Delta \bar{z}_2|_C + \beta_{12}^2 \xi_{21} \\ &\times \{[\alpha_2(1 + F_{ST})V_{F_0}(a_2) + \psi_{21}C_{am}(a_1, a_2)] \\ &\times [2\psi_{21}F_{ST}V_{F_0}(a_1) + \alpha_2C_{am}(a_1, a_2) \\ &+ \alpha_2C_{wi}(a_1, a_2)]\}. \end{aligned} \quad (15)$$

Comparing equation (13) with equation (15), we see that nonlinear social interactions have a larger effect in genetically structured populations than in panmictic ones. In panmictic populations, all individuals experience the same average social environment regardless of the nature of social interactions. However, in subdivided populations, demes can differ with respect to local social environment, and these differences among demes can be amplified into even larger differences when the social interactions are nonlinear in their phenotypic effects.

#### *Evolution of Interacting Phenotypes under Group Selection*

In the next set of conditions, we focus on the response of  $z_2$  to group selection in the absence of individual selection. We begin with the simplest case of no population structure and only linear effects of social interactions:

$$\text{condition E: } \beta_{12} = 0, \beta_{G2} \neq 0, F_{ST} = 0, \xi_{21} = 0.$$

In this case, there can be no response to selection because there is no among-group heritable variance (Wade 1978):

$$\Delta \bar{z}_2|_E = 0. \quad (16)$$

Allowing for nonlinear forms of social interactions,

$$\text{condition F: } \beta_{12} = 0, \beta_{G2} \neq 0, F_{ST} = 0, \xi_{21} \neq 0,$$

does not change the outcome:

$$\Delta \bar{z}_2|_F = 0. \quad (17)$$

The reason is simple. Just as an evolutionary response to individual selection requires genetic variation among individuals, a response to group selection requires genetic variation among groups (i.e., nonzero values of  $F_{ST}$ ).

We now allow for among-deme genetic variation but temporarily ignore nonlinear forms of social interactions:

$$\text{condition G: } \beta_{12} = 0, \beta_{G2} \neq 0, F_{ST} \neq 0, \xi_{21} = 0.$$

Here, we find a response to intergroup selection:

$$\begin{aligned} \Delta \bar{z}_2|_G &= \beta_{G2} \alpha_2 [2\alpha_2 F_{ST} V_{F_0}(a_2) + \psi_{21} C_{am}(a_1, a_2)] \\ &+ \beta_{G2} \psi_{21} [2\psi_{21} F_{ST} V_{F_0}(a_1) + \alpha_2 C_{am}(a_1, a_2)]. \end{aligned} \quad (18)$$

When we include nonlinear social interactions,

$$\text{condition H: } \beta_{12} = 0, \beta_{G2} \neq 0, F_{ST} \neq 0, \xi_{21} \neq 0,$$

we find that the response to group selection changes to

$$\begin{aligned} \Delta \bar{z}_2|_H &= \Delta \bar{z}_2|_G \\ &+ \xi_{21} \beta_{G2}^2 [2\alpha_2 F_{ST} V_{F_0}(a_2) + \psi_{21} C_{am}(a_1, a_2)] \\ &\times [2\psi_{21} F_{ST} V_{F_0}(a_1) + \alpha_2 C_{am}(a_1, a_2)]. \end{aligned} \quad (19)$$

Finally, in the case where both individual and group selection on the interacting phenotype occurs,

$$\text{condition I: } \beta_{12} \neq 0, \beta_{G2} \neq 0, F_{ST} \neq 0, \xi_{21} \neq 0,$$

the response is

$$\begin{aligned} \Delta \bar{z}_2|_I &= \Delta \bar{z}_2|_D + \Delta \bar{z}_2|_H - \xi_{21} \beta_{12} \beta_{G2} \\ &\times \{2\alpha_2 \psi_{21} C_{am}^2(a_1, a_2) \\ &+ 2\alpha_2 F_{ST} V_{F_0}(a_2) \\ &\times [\alpha_2 C_{wi}(a_1, a_2) \\ &+ \psi_{21}(1 + 3F_{ST})V_{F_0}(a_1)] \\ &+ C_{am}(a_1, a_2) \\ &\times [\alpha_2^2(1 + 3F_{ST})V_{F_0}(a_2) \\ &+ \alpha_2 \psi_{21} C_{wi}(a_1, a_2) \\ &+ 4\psi_{21}^2 F_{ST} V_{F_0}(a_1)]\}. \end{aligned} \quad (20)$$

When both forms of selection occur, the response cannot be predicted simply by summing the predicted response from each type of selection alone. That is,  $\Delta \bar{z}_2$  does not equal the sum of individual selection ( $\Delta \bar{z}_2|_D$ ) and group selection ( $\Delta \bar{z}_2|_H$ ). The final term in equation (20)

results from an interaction between the two levels of selection, as signified by the product  $\beta_{12}\beta_{G2}$ . Note that this term is a function of the coefficient of nonlinearity in social interactions,  $\xi_{21}$ . If the effects of social interactions are completely linear, then no interaction occurs between the two levels of selection.

### Interference between Levels of Selection

Is the response to selection at the individual level independent of the response to selection at the group level? If so, then the response to selection acting simultaneously at both levels should equal the sum of the response to individual selection alone and the response to group selection alone. We can measure the degree of interference as the difference between this sum and the actual response:

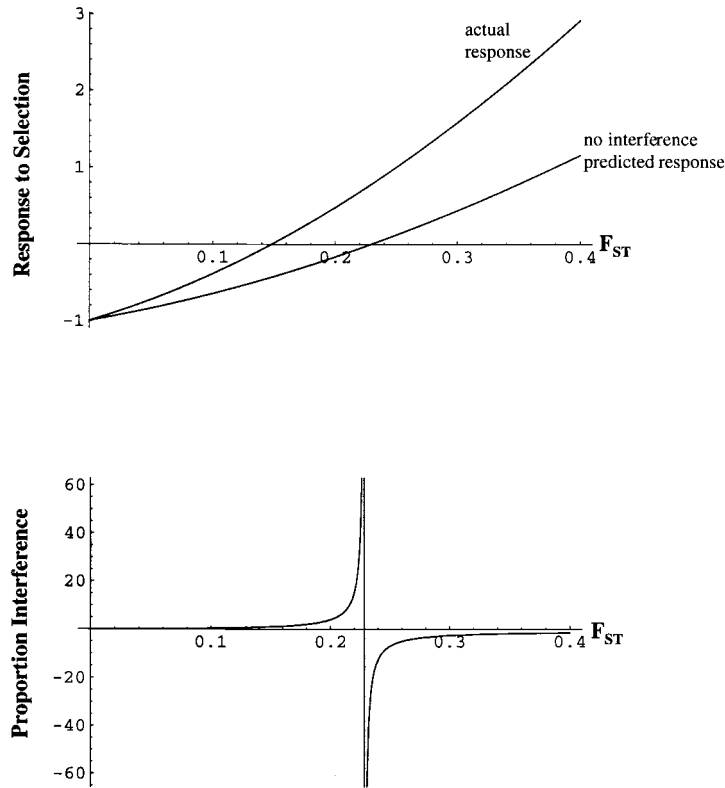
$$I = \text{Abs}(\Delta \bar{z}_2^*) - \frac{\Delta \bar{z}_2^*}{\text{Abs}(\Delta \bar{z}_2^*)} \Delta \bar{z}_2|_I, \quad (21)$$

where “Abs” refers to the absolute value and  $\Delta \bar{z}_2^*$  is the predicted response assuming no interference:

$$\Delta \bar{z}_2^* = \Delta \bar{z}_2|_D + \Delta \bar{z}_2|_H. \quad (22)$$

The variable  $I$  is the difference between the predicted response assuming no interference and the actual response, adjusting for sign of the predicted response. Note that the magnitude of interference is completely determined by the third term in equation (20). When  $I = 0$ , there is no interference between the two levels of selection. When  $I > 0$ , then the response to selection is less than expected assuming no interference (fig. 1). If  $I$  is large enough (i.e.,  $I > \Delta \bar{z}_2|_D + \Delta \bar{z}_2|_H$ ), then the actual response can be in the opposite direction to that predicted assuming no interference (see fig. 1 for values of  $F_{ST}$  between 0.15 and 0.23). When  $I < 0$ , then the response to selection is greater than expected assuming no interference.

The magnitude of interference can be large when expressed as a proportion of the response expected assuming no interference (figs. 1–3). When group selection is relatively weak ( $\beta_1 > \beta_G$ ), then the interference rises almost linearly with the degree of population genetic subdivision,  $F_{ST}$  (see fig. 2). When intergroup selection is relatively strong ( $\beta_1 = \beta_G$ ), then the interference rises somewhat faster with the degree of population genetic subdivision,  $F_{ST}$  (see figs. 1, 2). With nonlinear social interactions, the magnitude of interference is augmented considerably, even for small values of  $F_{ST}$  (see fig. 3).



**Figure 1:** *Top*, Magnitude and sign of the response to selection changes as a function of population subdivision,  $F_{ST}$ . The actual response is compared to the predicted response assuming no interference. For  $F_{ST}$  values in the range of approximately 0.0–0.15, the actual response is in the same direction as the predicted response but is of reduced magnitude. For  $F_{ST}$  values in the range of approximately 0.15–0.23, the direction of evolution is different between the two response curves. For  $F_{ST}$  values in the range of approximately 0.23–1, the actual response is in the same direction as the predicted response but of greater magnitude. Parameter values for this plot are  $\alpha_2 = \psi_{21} = \xi_{21} = V_{F_0}(a_1) = V_{F_0}(a_2) = \beta_{G2} = 1$ ,  $\beta_{I2} = -1$ , and  $C_{am}(a_1, a_2) = C_{wi}(a_1, a_2) = 0$ . *Bottom*, Interference as a proportion of the predicted response assuming no interference. Note the scale of the interference axis. Large values of interference occur near  $F_{ST} \approx 0.23$  because the predicted response assuming no interference is close to 0.

*Hard and Soft Selection*

In structured populations, so-called individual selection can operate in two different ways. With hard selection, individual phenotypes are selected relative to the global mean phenotype. In contrast, with soft selection, individual phenotypes are selected relative to the local mean. These distinctions are important in multilevel selection models because they determine when group selection can and cannot operate (see also Kelly 1997). By setting  $\beta_{I1} = \beta_{G1} = \beta_{G2} = 0$ , as in conditions A–D, the fitness function of equation (5) describes selection on  $z_2$  acting at the individual level across the entire metapopulation (i.e., hard selection).

An alternative fitness function to equation (5), which permits us to capture these distinctions between hard and soft selection, is

$$w = \beta_{W1}(z_1 - \bar{z}_1) + \beta_{W2}(z_2 - \bar{z}_2) + \beta_{A1}\bar{z}_1 + \beta_{A2}\bar{z}_2 + k, \tag{23}$$

where  $\beta_{Wi}$  is the average within-deme directional selection gradient on  $z_i$  and  $\beta_{Ai}$  is the among-deme directional selection gradient on  $z_i$ .

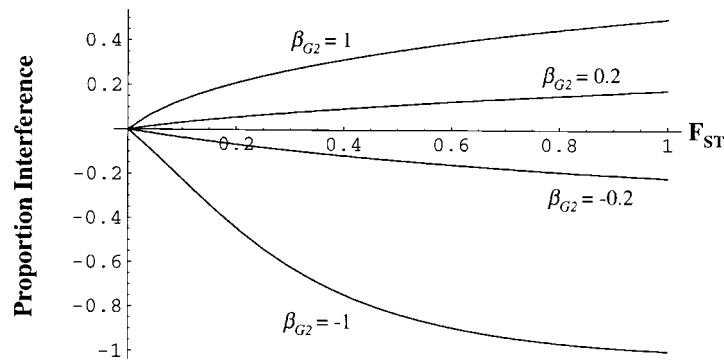
Note the selection gradients in equation (23) are related to the selection gradients in equation (5),

$$\beta_{Ii} = \beta_{Wi} \tag{24a}$$

and

$$\beta_{Gi} = \beta_{Ai} - \beta_{Wi}. \tag{24b}$$

Soft selection on  $z_2$  occurs when  $\beta_{W1} = \beta_{A1} = \beta_{A2} = 0$ . Equation (24b) shows that under these conditions,



**Figure 2:** The effect of group selection on interference. Interference is expressed as a proportion of the predicted response assuming no interference. Note that interference is asymmetrically influenced by group selection; strong group selection opposing individual selection ( $\beta_{G2} = -1$ ) generates proportionally higher interference. Interference is plotted for four values of the group selection gradient,  $\beta_{G2}$ . Parameter values for this plot are  $\alpha_2 = \psi_{21} = \xi_{21} = V_{F_0}(a_1) = V_{F_0}(a_2) = \beta_{12} = 1$  and  $C_{\text{am}}(a_1, a_2) = C_{\text{wi}}(a_1, a_2) = 0$ .

$\beta_{G2} = -\beta_{w2} = -\beta_{12}$ . In other words, soft selection represents a specific type of group selection as defined by equation (5) (see also Goodnight and Stevens 1997; Wade et al. 1999). Under soft selection, the response to selection on  $z_2$  is

$$\begin{aligned} \Delta \bar{z}_2 = & \beta_{12} \alpha_2^2 (1 - F_{ST}) V_{F_0}(a_2) + \beta_{12} \alpha_2 \psi_{21} C_{\text{wi}}(a_1, a_2) \\ & + \beta_{12}^2 \xi_{21} \alpha_2^2 (1 - F_{ST}) V_{F_0}(a_1) C_{\text{wi}}(a_1, a_2). \end{aligned} \quad (25)$$

When there is no population subdivision ( $F_{ST} = 0$ ), the results for soft selection (eq. [25]) and hard selection (eq. [12]–[15]) are identical. This result is unsurprising as the definitions of these two forms of selection are equivalent in the absence of subdivision (the phenotypic mean within a deme equals the grand phenotypic mean when  $F_{ST} = 0$ ).

When no among-deme covariance exists ( $C_{\text{am}}[a_1, a_2] = 0$ ) and the effect of social partners on the phenotype is additive ( $\xi_{21} = 0$ ), the difference in the response to hard selection (eq. [14]) versus the response to soft selection (eq. [25]) is

$$\begin{aligned} \Delta \bar{z}_2(\text{HARD}) - \Delta \bar{z}_2(\text{SOFT}) = & 2\beta_{12} \alpha_2^2 F_{ST} V_{F_0}(a_2) \\ & + 2\beta_{12} \psi_{21}^2 F_{ST} V_{F_0}(a_2). \end{aligned} \quad (26)$$

Under these conditions, this equation shows that the response to hard selection is always greater than the response to soft selection. The first term in equation (26) would apply to any character, whereas the second term is a unique result of indirect genetic effects in subdivided populations. (Additional terms contribute to the difference between the response to hard and soft selection when there is either

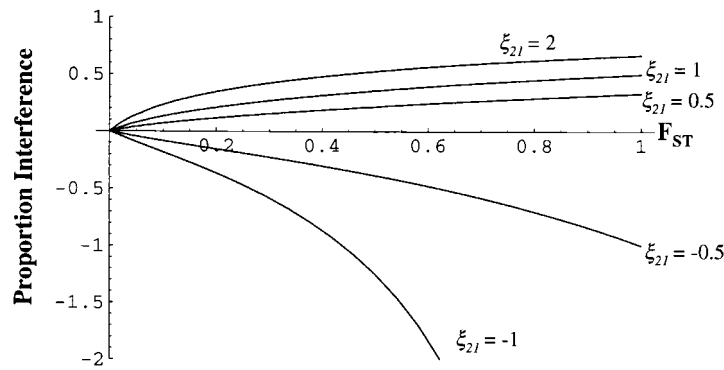
some among-deme covariance or some degree of non-linearity in the effect of social partners.)

## Discussion

Our model builds from the pioneering efforts of Moore et al. (1997, p. 1358), who noted that “interacting phenotypes differ from other traits because they are determined in part by an environment that can evolve, that is, traits of other individuals.” Even though their model assumed no population structure and simple, linear-additive effects of social interactions, Moore et al. (1997) found that IGEs altered predictions about evolutionary change. For example, they showed that, when selection acts only on an effector trait (e.g., our  $z_1$ ), there is evolution in an interacting phenotype,  $z_2$ , because the effector represents an environmental effect on the mean of the other character. Furthermore, this effect occurs even when there is no genetic variation for the interacting phenotype,  $z_2$ , because, as the mean of  $z_1$  changes, the average environment experienced during development changes for  $z_2$ . They also showed how genetic variation in the effector trait,  $z_1$ , contributes to the response to selection acting only on  $z_2$ , the interacting trait, whenever there is a genetic correlation between them. The evolution of the interacting phenotype changes the environment in which it is expressed (i.e., the mean of  $z_1$ ) via the genetic correlation. (If we assume  $F_{ST} = \xi_{21} = 0$  [eq. (12)], then our model is equivalent to that of Moore et al. [1997].)

The incorporation of population structure extends the range of evolutionary influence of IGEs. In metapopulations with genetic subdivision ( $F_{ST} > 0$ ), we find that IGEs have a much greater influence on the evolution of interacting phenotypes. For example, with population structure





**Figure 3:** The effect of nonlinearity in social interactions on interference. Interference is expressed as a proportion of the predicted response assuming no interference. Note that interference is asymmetrically influenced by nonlinearity of social interaction; negative values of the interaction coefficient ( $\xi_{21} < 0$ ) contribute proportionally more to interference. Interference is plotted for five different values of the coefficient of nonlinearity,  $\xi_{21}$ . Parameter values for this plot are  $\alpha_2 = \psi_{21} = V_{F_0}(a_1) = V_{F_0}(a_2) = \beta_{12} = \beta_{G2} = 1$  and  $C_{am}(a_1, a_2) = C_{wi}(a_1, a_2) = 0$ .

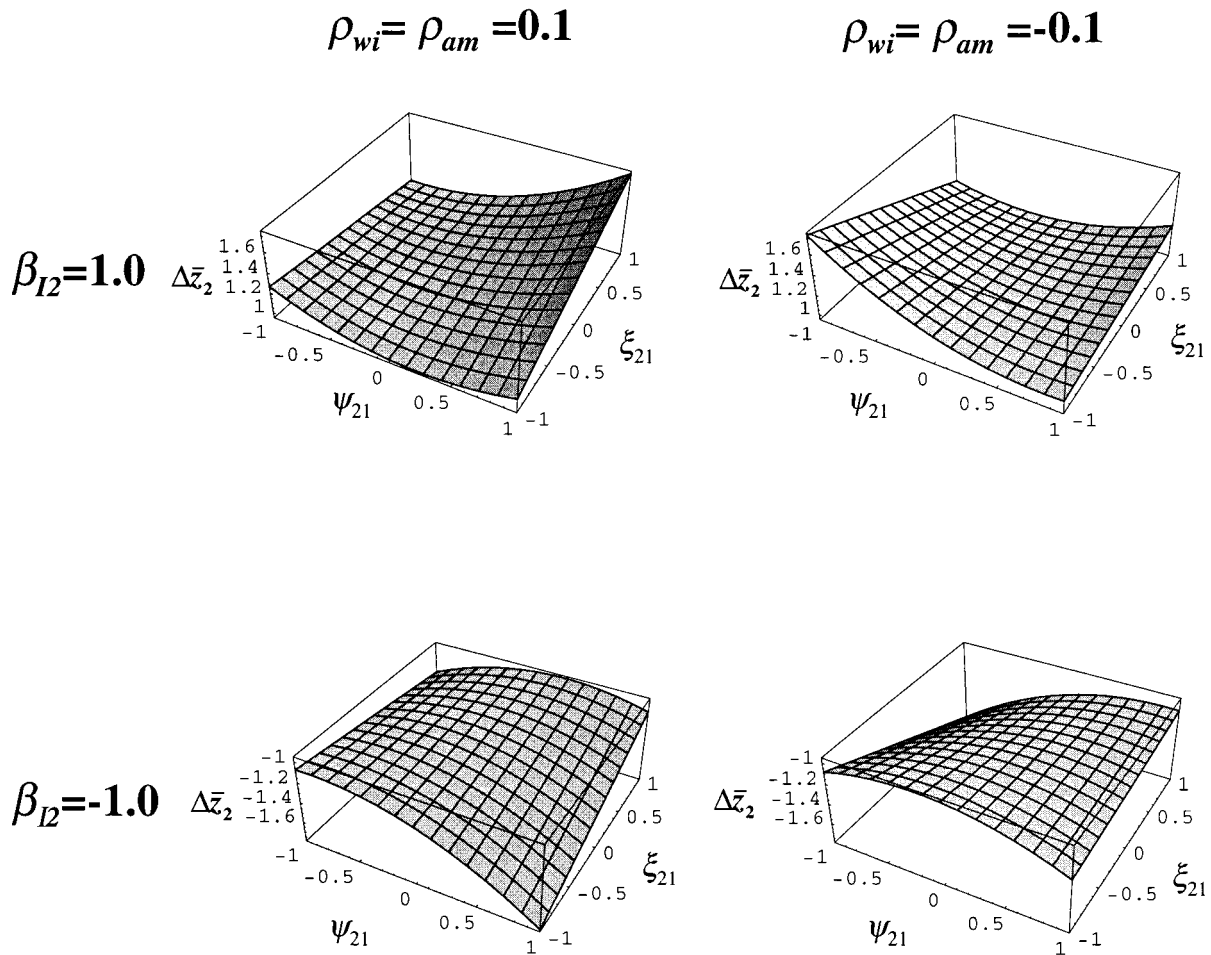
but no genetic correlation between effector and interacting trait ( $\rho_{12} = 0$ ), an unselected effector trait ( $\beta_{11} = \beta_{G1} = 0$ ) still contributes to the response to selection of the interacting trait, whether selection occurs at the individual ( $\beta_{12} \neq 0$  in eq. [15]) or group ( $\beta_{G2} \neq 0$  in eq. [18]) levels. Furthermore, an interacting phenotype without any genetic variation ( $V_{F_0}[a_2] = 0$ ) will respond to direct individual selection operating on either trait ( $\beta_{12} \neq 0$  in eq. [14] or  $\beta_{11} \neq 0$  in eq. [10] and [11]). Individual selection alone causes this effect because changing the mean of the effector trait changes the mean of the interacting phenotype, which “perceives” the mean of the effector as part of its environment. In metapopulations, the reciprocal environmental effects between an IGE and its effector(s) cannot be set and held equal to 0 as is commonly done with nonevolving environmental effects in standard quantitative genetics theory. Thus, the evolution of an interacting phenotype, whether it is heritable or not, is always affected by the evolution of the effector trait.

What happens to the evolution of an effector trait,  $z_1$ , when there is selection on the trait it influences,  $z_2$ ? First, we emphasize that, unlike the IGE, the effector trait must be heritable (eq. [10]). Given that it is heritable ( $V_{F_0}[a_1] \neq 0$ ), an unselected effector trait ( $\beta_{11} = \beta_{G1} = 0$ ) will respond to selection acting on the interacting trait ( $\beta_{12} \neq 0$  or  $\beta_{G2} \neq 0$ ). Unlike standard quantitative genetics theory, no genetic correlation is required for this effect (i.e.,  $\rho_{12} = 0$ ) as long as there is population genetic structure ( $F_{ST} > 0$ ). Thus, the evolution of a heritable effector trait is always affected by the evolution of an interacting trait in a subdivided population.

Reciprocally, the evolutionary response of the interacting trait,  $z_2$ , is altered by the role of the effector trait,  $z_1$ , in determining the direction and strength of the local social

interactions. In figure 4, for a weakly subdivided population ( $F_{ST} = 0.10$ ), we graph the evolutionary response surface of the mean of the interacting phenotype,  $\bar{z}_2$ , as the joint function of  $\psi_{21}$ , the strength of the social effect mediated by the mean of  $z_1$ , and of  $\xi_{21}$ , the nonlinearity of the social effect. We emphasize three important features of figure 4. First, within each of the four graphs, there is an interaction between the strength of social context ( $\psi_{21}$ ) and the degree of nonlinearity of social effect ( $\xi_{21}$ ). Second, changes in the sign of the genetic correlation between  $z_1$  and  $z_2$  have a profound effect on the evolutionary response surface (compare the right-hand with the left-hand graphs in fig. 4). Third, the genetic correlation between  $z_1$  and  $z_2$  ( $\rho$ ) interacts with the direction of selection on  $z_2$  ( $\beta_{12}$ ) to change the response surface of the  $z_2$  mean (compare the upper with the lower graphs of fig. 4). Even in the absence of direct selection on the effector trait,  $z_1$ , it is almost impossible to predict the rate of response of the interacting trait to direct individual selection in weakly subdivided populations. These effects are only enhanced with greater degrees of population genetic subdivision.

In figure 5, we show that the interacting trait evolves when there is direct selection, linear social effects, or nonlinear social effects separately (fig. 5A) and in combination (fig. 5B) as a function of the degree of population genetic subdivision,  $F_{ST}$ . In figure 5, there is no genetic correlation between the interacting and effector traits, and there is no intergroup selection. In all cases, the effect on the rate of evolution of  $z_2$  increases linearly (or very nearly so) with increasing population structure. Figure 6 shows these same effects when there are the genetic correlations between the two traits. In all cases, the effect on the rate of evolution is nonlinear. Indeed, with nonlinear social effects of opposite sign to the direct effects, the direction of evolution



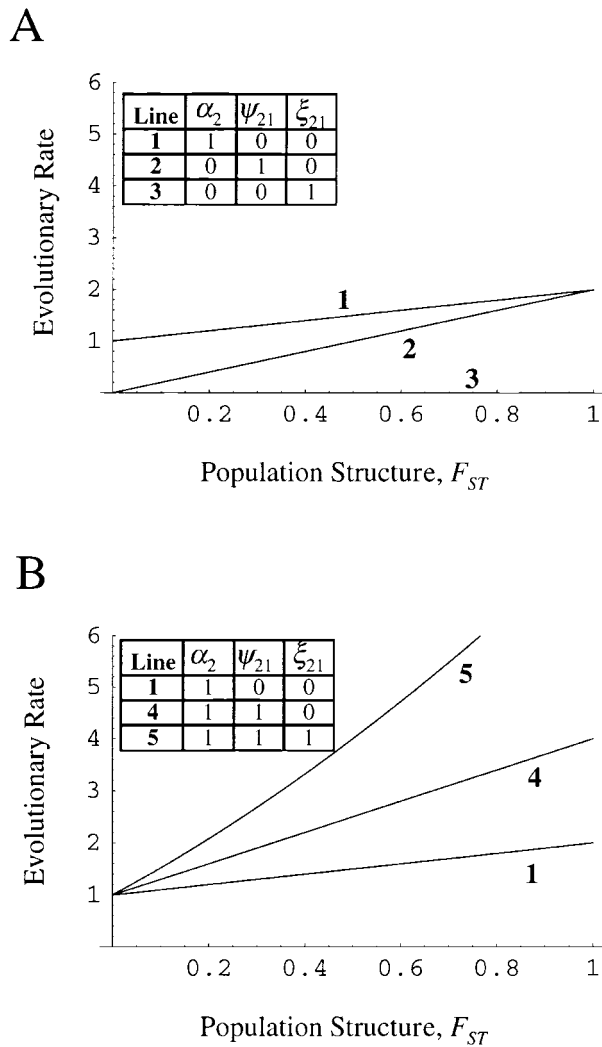
**Figure 4:** Evolutionary rate,  $\Delta \bar{z}_2$ , as a function of the coefficients of social effects. The selection response to positive directional selection (*top row*) is contrasted with negative directional selection (*bottom row*), and the effect of small positive genetic correlations (*left column*) is contrasted with the effect of small negative genetic correlations (*right column*);  $\rho_{wi}$  and  $\rho_{am}$  are the genetic correlations within and among demes, respectively. Taken together, these four plots show that the coefficients of social interactions interact with the direction of selection and the sign of the genetic correlations. Parameter values for these plots are  $\alpha_2 = V_{F_0}(a_1) = V_{F_0}(a_2) = 1$ ,  $F_{ST} = 0.1$ , and  $\beta_{I1} = \beta_{G1} = \beta_{G2} = 0$ .

of  $z_2$  can be the opposite of that expected under direct selection. Note that this change in direction depends on  $F_{ST}$  and that it occurs despite an absence of intergroup selection.

In our model, we assume that an individual's value of the interacting phenotype,  $z_2$ , is affected by the local (i.e., demic) mean value,  $\bar{z}_1$ , of the effector trait,  $z_1$ . This is analogous to assuming that every individual interacts equally with all members of its deme. When individuals interact randomly but with only a single individual or subset of individuals within the deme, the phenotypic variance is larger than when individuals interact equally with all members of the deme. Nevertheless, the response to selection given by our model also holds for these situations. If  $z'_j$  is the value of the  $j$ th trait in a randomly chosen

social partner or the mean of a randomly chosen subset of social partners, the expected values of the relevant covariances remain the same:  $\text{Cov}(z_i, z'_j) = \text{Cov}(z_i, \bar{z}_j) = 2\rho_{am}F_{ST}[V_{F_0}(a_i)V_{F_0}(a_j)]^{1/2}$ . (By analogy, the average within-deme  $\text{Cov}[z_2, \bar{z}_1]$  could be estimated by resampling random subsets from within demes.) When individuals preferentially interact with some subset of other deme members, as is the case with genetic relatives in kin selection models, then the covariance terms of our model must be recalculated to reflect these interaction biases.

Perhaps most interesting, we find that, whenever selection acts at multiple levels simultaneously, selection at one level can interfere with selection at another level if there are nonlinear effects of social interactions ( $\xi_{21} \neq 0$ ; see figs. 1–3). The actual total response to multilevel selection



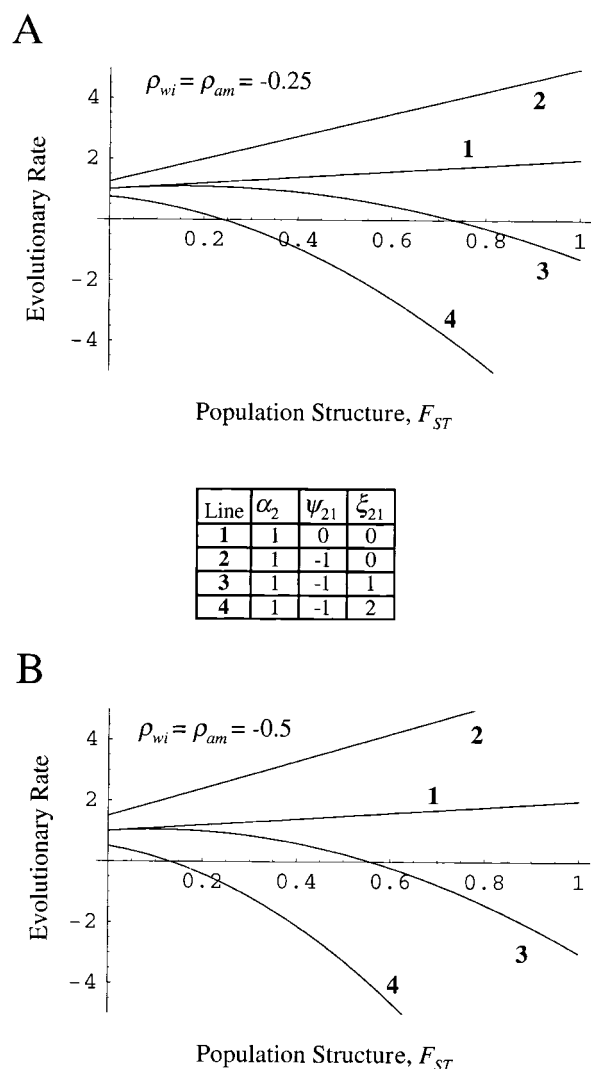
**Figure 5:** Evolutionary rate,  $\Delta\bar{z}$ , with different types of genetic effects. **A**, Role of each type of genetic effect in isolation. The response to selection is shown when the trait,  $z_2$ , is determined only by direct genetic effects (line 1),  $z_2$  is determined only by linear indirect genetic effects (line 2), and  $z_2$  is determined only by nonlinear indirect genetic effects (line 3). **B**, Consequences of combining social effects. The response to selection is shown when  $z_2$  is determined only by direct genetic effects (line 1; as in **A**),  $z_2$  is determined by direct genetic effects and linear indirect genetic effects (line 4), and  $z_2$  is determined by direct genetic effects as well as linear and nonlinear indirect genetic effects (line 5). When no genetic correlations exist, linear indirect genetic effects contribute only to the response to selection when there is population structure (see line 2). Nonlinear indirect genetic effects only contribute to the response to selection when there is population structure as well as direct genetic effects and linear indirect genetic effects (cf. lines 3 and 5). The parameter values for these plots are  $\beta_{12} = V_{F_0}(a_1) = V_{F_0}(a_2) = 1$  and  $\beta_{11} = \beta_{G1} = \beta_{G2} = C_{am}(a_1, a_2) = C_{wi}(a_1, a_2) = 0$ .

can be greater or less than the predicted response assuming no interference. In fact, the actual total response can even be opposite in sign to the predicted response. In this framework, soft selection, when  $\xi_{21} \neq 0$ , represents a type of intergroup selection, and so interference can occur in this situation (see Wade et al. 1999 for a discussion of soft selection in relation to intergroup selection in the absence of social effects, i.e., the cases where  $\xi_{21} = \psi_{21} = 0$ ).

*Empirical Studies of the Levels of Selection*

Interference between different levels of selection was first detected in experimental studies of group and individual selection for emigration rate in the flour beetle, *Tribolium castaneum* (Craig 1982), and for leaf area in the cress, *Arabidopsis thaliana* (Goodnight 1985). In both studies, the response to selection, when individual and group selection were acting in the same direction, was significantly different from the response expected based on treatments with each level of selection acting alone. Both authors interpreted this surprising finding in terms of the different kinds of genetic variance available to selection at the different levels, especially the novel variance components available to intergroup selection. Goodnight (1985) also suggested that directional selection within demes might reduce the heritable among-deme variance created by random genetic drift and, thus, also contribute to the observed interference between the levels of selection. Our model does not examine either of these possibilities but rather suggests that interference between levels will be a common feature associated with selection in metapopulations whenever there are IGEs. In order to measure the extent of the interference between levels of selection, replicated, factorial experiments with both selection level and selection intensity as treatment factors would have to be conducted (Wade and Kalisz 1990). Interactions can only be detected through such a factorial design.

In regard to these experiments, we note that both emigration rate and leaf area are traits with a conspicuous social component. In *Tribolium*, an individual's emigratory phenotype is determined both by its own genotype and by local population density. In addition, genotype and social context interact because different genotypes are differentially sensitive to density (Wade and McCauley 1980; Craig 1982). In *Arabidopsis*, the leaf area of a focal plant is determined by its own genotype as well as by the leaf areas (and, hence, genotypes) of its surrounding neighbors. In this case, the interaction between an individual's genotype and that of its neighbors is such that individual selection alone for increased leaf area produces instead a decline in the mean of leaf area (Goodnight 1985). Our theory shows that interference between the levels of selection should be the norm for both of these traits and



**Figure 6:** Evolutionary rate ( $\Delta\bar{z}_2$ ), social effects, and genetic correlations. The magnitude and direction of the response to selection,  $\Delta\bar{z}_2$ , depends on social influences as well as the population genetic architecture (i.e., population structure and genetic correlations within  $[\rho_{wi}]$  and among  $[\rho_{am}]$  demes). Weak negative genetic correlations (A,  $\rho_{wi} = \rho_{am} = -0.25$ ) are contrasted with stronger negative genetic correlations (B,  $\rho_{wi} = \rho_{am} = -0.5$ ). In this example, the direction of evolution can be reversed if there are nonlinear indirect genetic effects, genetic correlations, and population structure. The level of population structure required for this reversal decreases with increasing non-linearity of IGEs (cf. lines 3 and 4) and increasing magnitude of the genetic correlation (cf. A and B). The parameter values for these plots are  $\beta_{12} = V_{F_0}(a_1) = V_{F_0}(a_2) = 1$  and  $\beta_{11} = \beta_{G1} = \beta_{G2} = 0$ .

many other interacting phenotypes. Griffing (1981) found similar results in his theoretical models of traits affected by competitive interactions.

Unlike the laboratory studies, interference between the

levels of selection has not been detected in any field studies of intergroup selection (reviewed in Goodnight and Stevens 1997). However, the kinds of methodologies employed in studies to date are inadequate for detecting interference. For example, in an experimental investigation of selection within and between kin groups affecting cannibalism in the willow leaf beetle, *Plagioderma versicolora*, Breden and Wade (1989) estimated the strength of each level of selection acting alone in separate experiments and then additively combined the separate estimates into an overall picture of selection. Furthermore, McCauley et al. (1988) showed that the degree of genetic subdivision within this species varied spatially as well as temporally. Based on our theoretical results, the additive combination of these two levels of selection probably does not give an accurate picture of total selection on the interacting phenotype of cannibalism. Furthermore, the levels of selection would combine in different ways as the population structure changed temporally and spatially. These field experiments were not designed to detect an interaction between the levels of selection. Very different experimental procedures with a larger number of treatments measuring each level of selection alone and together in a variety of combinations would be necessary to permit detection of the kinds of interactions we modeled here (Wade and Kalisz 1990).

In addition to manipulative experiments, contextual analysis, a method based on linear regression, is commonly used to detect the possibility of among-group selection in natural populations by statistically detecting an effect of group mean on the fitness of focal individuals (Goodnight et al. 1992). Although such group effects have been detected in several studies (reviewed in Goodnight and Stevens 1997), interactions are particularly difficult to detect statistically by linear regression, especially so in unmanipulated natural populations where the patterns of individual and group variation may not be factorially combined. Indeed, if interactions were present, the method of linear regression would assign as much of the interaction variance as possible to each of the main effects of individual and group mean. Although the total response to selection might be quite accurately detected, its component parts would not.

#### Interacting Phenotypes: Beyond Social Traits

Interacting phenotypes are characters that require or are influenced by interactions with conspecifics and are common in nature (Moore et al. 1997 and references therein). Although overt social behaviors in animals such as aggression and courtship displays may be the most obvious examples, interacting phenotypes also exist for many other kinds of traits and taxa. For example, plant growth rate,

biomass, and form are all influenced by light and soil qualities, which in turn are affected by the phenotypes of individual plants in the immediate neighborhood (e.g., Wilson 1977; Griffing 1989). Morphological structures including body size, genitalia, and ornamentation can be influenced by social and competitive interactions with conspecifics (e.g., Conner 1989; Stern and Emlen 1999). Life-history characters such as age and size at maturity, egg size, and fecundity are also affected by the identity and actions of members of social groups. Indirect genetic effects, then, may have much greater importance than previously appreciated for an extremely broad range of traits and taxa.

Morphological traits involved in the expression of social traits will also evolve differently in metapopulations. For example, in flour beetles, it is likely that the cannibalism of eggs by larvae is mediated by mandible size and egg size (Teleky 1980): eggs exceeding the gape of small larvae cannot be cannibalized. The phenotype “egg cannibalism” is thus jointly influenced by an individual’s own mandible size, the same individual’s propensity toward cannibalism, and the egg size phenotypes expressed by other individuals. The level of cannibalism expressed by an individual is a nonlinear function of the interaction of these phenotypes (as well as many others, like tunneling speed). Mandible morphology is a taxonomically important trait in many groups, but it is not typically considered a social trait in any respect. Egg size, too, is not typically considered a social trait. However, because the social trait of cannibalism links mandible size with egg size via egg viability, the evolution of both mandible size and egg size will be affected by population genetic structure, whether they share a genetic correlation or not. Purely environmental effects on mandible size (e.g., those caused by the nutritional or

thermal environment) will affect the evolution of egg size because it will influence the mean level of egg cannibalism across a metapopulation as well as the variance in cannibalism among demes in different environments. Reciprocally, environmental effects on egg size will affect the evolution of mandible size. This example demonstrates how apparently nonsocial morphological traits can be involved in the expression of interacting traits and thus become subject to the evolutionary consequences of selection of IGEs in metapopulations. Hence, the simple distinction between purely social and purely asocial traits is blurred by IGEs and metapopulation structure.

The existence of IGEs in structured populations changes the way in which interacting and effector traits evolve. The mean value of one trait within a deme becomes an important component of the environment experienced by the other trait. Because each of the traits can evolve, the environments they experience can also evolve (Wolf et al. 2001). Our model illustrates how IGEs in metapopulations can add a component of context to apparently “pure” individual selection. As asserted by Goodnight and Wade (2000, p. 322), “Multilevel selection is far more common in nature than previously believed, and ‘pure’ individual selection is far less common.” With nonlinear social effects, interactions between levels of selection can become important. Context appears inescapable in metapopulations.

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#### Appendix

Nonlinear social effects contribute to the linear effects when trait means are nonzero. There are two ways to deal with this problem. First, we can relax the assumption that the global mean genetic values are 0 and explicitly include the trait means, making equations (7), (10), and (11) become, respectively,

$$\bar{\bar{z}}_2 = \alpha_2 E(\Delta a_2) + \psi_{21} E(\Delta \bar{a}_1) + \xi_{21} E(\Delta a_2) E(\Delta \bar{a}_1) + \xi_{21} \bar{\bar{a}}_2 E(\Delta \bar{a}_1) + \xi_{21} \bar{\bar{a}}_1 E(\Delta \bar{a}_2) + \xi_{21} \Delta C(\bar{a}_1, a_2), \quad (\text{A1})$$

$$E(\Delta a_1) = \{\beta_{11}(1 + F_{ST}) + 2[\beta_{12}(\psi_{21} + \xi_{21} \bar{\bar{a}}_2) + \beta_{G1} + \beta_{G2}(\psi_{21} + \xi_{21} \bar{\bar{a}}_2)]F_{ST}\}V_{F_0}(a_1) \\ + \beta_{12}(\alpha_2 + \xi_{21} \bar{\bar{a}}_1)C_{wi}(a_1, a_2) + (\beta_{12} + \beta_{G2})(\alpha_2 + \xi_{21} \bar{\bar{a}}_1)C_{am}(a_1, a_2), \quad (\text{A2})$$

and

$$E(\Delta a_2) = [\beta_{12}(1 + F_{ST}) + 2\beta_{G2}F_{ST}](\alpha_2 + \xi_{21}\bar{a}_1)V_{F_0}(a_2) + \beta_{11}C_{wi}(a_1, a_2) \\ + [\beta_{11} + \beta_{12}(\psi_{21} + \xi_{21}\bar{a}_2) + \beta_{G1} + \beta_{G2}(\psi_{21} + \xi_{21}\bar{a}_2)]C_{am}(a_1, a_2). \quad (A3)$$

All the other results can be rederived using these three equations and equation (6).

Alternatively, the global genetic means can be rescaled to 0, but the value of the parameters  $\alpha$  and  $\psi$  must be reevaluated such that

$$\alpha_2^* = \alpha_2 + \xi_{21}\bar{a}_1 \quad (A4)$$

and

$$\psi_{21}^* = \psi_{21} + \xi_{21}\bar{a}_2, \quad (A5)$$

where  $\alpha_2^*$  and  $\psi_{21}^*$  are the rescaled values and  $\alpha_2$  and  $\psi_{21}$  are the original values that apply when the global genetic means are 0. Note that when the global genetic means are 0, the rescaled values are the same as the original values. Equations (A4) and (A5) show that when the global genetic means are not 0, nonlinear social effects contribute to linear direct and indirect genetic effects. These rescaled parameter values can be substituted into the equations in the text to evaluate the response to selection when the global genetic means are nonzero.

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