

# HOW TO MEASURE INDIRECT GENETIC EFFECTS: THE CONGRUENCE OF TRAIT-BASED AND VARIANCE-PARTITIONING APPROACHES

Joel W. McGlothlin<sup>1,2</sup> and Edmund D. Brodie III<sup>1,3</sup>

<sup>1</sup>*Department of Biology, University of Virginia, Charlottesville Virginia 22902*

<sup>2</sup>*E-mail: jmcgloth@virginia.edu*

<sup>3</sup>*E-mail: bbrodie@virginia.edu*

Received December 15, 2008

Accepted February 12, 2009

Indirect genetic effects (IGEs), which occur when phenotypic expression in one individual is influenced by genes in another conspecific individual, may have a drastic effect on evolutionary response to selection. General evolutionary models of IGEs have been developed using two distinct theoretical frameworks derived from maternal effects theory. The first framework is trait-based and focuses on how phenotypes are influenced by specific traits in a social partner, with the strength of interactions defined by the matrix  $\Psi$ . The second framework partitions total genetic variance into components representing direct effects, indirect effects, and the covariance between them, without identifying specific social traits responsible for IGEs. The latter framework has been employed more commonly by empiricists because the methods for estimating variance components are relatively straightforward. Here, we show how these two theoretical frameworks are related to each other and derive equations that can be used to translate between them. This translation leads to a generalized method that can be used to estimate  $\Psi$  via standard quantitative genetic breeding designs or pedigrees from natural populations. This method can be used in a very general set of circumstances and is widely applicable to all IGEs, including maternal effects and other interactions among relatives.

**KEY WORDS:** Animal model, interacting phenotypes, maternal effects, quantitative genetics, social evolution.

Quantitative genetic theory has provided a useful framework for studying the process of phenotypic evolution in both laboratory and natural populations (Falconer and MacKay 1996; Roff 1997; Lynch and Walsh 1998; Kruuk 2004). In standard quantitative genetic theory, phenotypic variance is partitioned into heritable (additive genetic) and nonheritable components. Measures of additive genetic variance, or its multivariate analog, the additive genetic variance–covariance matrix ( $\mathbf{G}$ ), may be used to make short-term predictions about how a population should respond to phenotypic selection (Lande 1979; Lande and Arnold 1983). Furthermore, the pattern of covariation in  $\mathbf{G}$  represents the degree to which traits should evolve together and has been interpreted as a measure of both integration and constraint (Merilä and Björklund 2004).

In certain circumstances, this standard model must be altered to accommodate complex patterns of inheritance that may influence a population's response to selection (Arnold 1994). One such case occurs when trait expression is affected by not only an organism's own genes but also those of a conspecific. Such indirect genetic effects (IGEs) may occur among related or unrelated individuals, and include the special case of maternal effects (Cheverud and Moore 1994; Moore et al. 1997; Mousseau and Fox 1998; Wolf et al. 1998; Räsänen and Kruuk 2007). From the perspective of a given focal individual, an IGE may be thought of as an environmental effect. However, this environment is partially determined by the social partner's genes, and may thus contribute to an evolutionary response to selection. Theoretical models of maternal effects and other IGEs have shown that they

may often have drastic effects on the rate and direction of evolution (Kirkpatrick and Lande 1989; Lande and Kirkpatrick 1990; Moore et al. 1997; Bijma and Wade 2008).

General evolutionary models of IGEs have been constructed within two theoretical frameworks, following a historical dichotomy in maternal effects theory (reviewed in Cheverud and Moore 1994; Lynch and Walsh 1998). The first generalized model of IGEs was developed in a series of papers by Griffing (1967, 1969, 1976, 1981; see also Bijma et al. 2007a; Bijma and Wade 2008). Griffing's approach to indirect, or "associative," genetic effects resembles maternal effects theory developed by Willham (1963, 1972), and more recently, by Cheverud (1984; Cheverud and Moore 1994) and Lynch (1987). As in Willham's models, Griffing's framework partitions total heritable variance into a direct component, which arises from an individual's own genotype, and an indirect component, which arises from interactions among individuals that may or may not be related. A second approach was developed by Moore et al. (1997), following maternal-effects theory by Falconer (1965) and Kirkpatrick and Lande (1989). This framework was trait-based, defining IGEs as variation in the expression of a focal individual's traits caused by one or more specific heritable traits in an interacting individual. Together, the traits expressed in the two individuals are called interacting phenotypes. The strength of IGEs in the trait-based framework is determined by  $\Psi$ , a square matrix of regression coefficients that is analogous to  $\mathbf{M}$ , the matrix of maternal effect coefficients (Kirkpatrick and Lande 1989; Moore et al. 1997). Either approach may be used to predict the relative importance of direct and indirect sources of genetic variance influencing a character, and each has its advantages and disadvantages (Cheverud and Moore 1994; Wolf et al. 1998). For example, the variance-components approach has the advantage of not requiring the identification of specific traits that influence expression and so may be less sensitive to the exclusion of unknown or unmeasured traits. However, trait-based formulations are required to analyze how interactions among specific traits affect phenotypic evolution.

The variance-components framework has been more commonly employed in empirical studies of maternal effects, because relevant parameters may be estimated using laboratory breeding designs or natural pedigrees (Lynch and Walsh 1998; Roff 1998; Räsänen and Kruuk 2007). Muir (2005) and Bijma et al. (2007b) have demonstrated that analogous methods allow for the measurement of IGEs among unrelated individuals. To date, most of the empirical studies attempting to measure such IGEs have used this framework (e.g., Wolf 2003; Muir 2005; Petfield et al. 2005; Bergsma et al. 2008; Brommer and Rattiste 2008; Ellen et al. 2008; Wilson et al. 2009). Although measurements of  $\mathbf{M}$ ,  $\Psi$ , or their components could provide great insight into the complex pattern of covariance among interacting phenotypes, only a few studies have attempted to estimate these parameters (Schluter

and Gustafsson 1993; Roff 1998; Kent et al. 2008; Bleakley and Brodie 2009). One potential reason for the paucity of such measurements is that a generalized, easily applicable empirical method has not yet been proposed. For example, the method suggested by Lande and Price (1989) for measuring  $\mathbf{M}$  relies on differences between mother-offspring and father-offspring regressions, and thus cannot be applied when parental phenotypes are unavailable or when interactants are not parents and offspring (i.e., it cannot be used to estimate  $\Psi$ ). Furthermore, the single experimental approach developed specifically to measure  $\Psi$  is not generally applicable to most laboratory or natural populations because it relies on testing with multiple inbred lines (Bleakley and Brodie 2009).

A second reason that the trait-based estimates have been less common is that connections between the two theoretical frameworks are not immediately obvious. Whereas components of  $\mathbf{M}$  can be shown to be functions of empirically estimable variance components (Lynch and Walsh 1998), thus demonstrating the equivalence of the two frameworks, as of yet it has been unclear whether the generalized IGE models are also equivalent. An apparent difficulty arises from the potential for feedback loops that may occur when one trait affects itself in a social partner or when two traits in different individuals reciprocally affect one another (Moore et al. 1997; Kölliker et al. 2005). Such feedback is modeled explicitly in the trait-based framework but appears only implicitly in the variance-components framework.

In this article, we briefly summarize the trait-based and variance-components approaches for modeling IGEs and demonstrate that the two approaches are compatible. Models of  $\Psi$  for social interactions of more than two individuals are derived. We then show that the equivalence between trait-based and variance-components approaches leads to a statistical method for the estimation of  $\Psi$  or  $\mathbf{M}$ , given an appropriate experimental design.

## Models of IGEs

Moore et al. (1997) defined a phenotype in a focal individual ( $z_i$ ) as the sum of its own additive genetic and environmental components ( $a_i$  and  $e_i$ , respectively) as well as the phenotype of a social partner ( $z'_j$ ). Here, the prime denotes that the phenotype occurs in another individual, and the subscript  $j$  denotes that it is a different trait than trait  $i$ . This effect is scaled by the parameter  $\psi_{ij}$ , which is a regression coefficient describing the strength and direction of the effect of trait  $j$  on trait  $i$ . The social phenotype may also be broken down into heritable and environmental components, giving

$$z_i = a_i + e_i + \psi_{ij}a'_j + \psi_{ij}e'_j. \quad (1)$$

Following standard quantitative genetic theory, genetic and environmental effects are assumed to be uncorrelated, and environmental effects are assumed to have zero mean (Falconer and

MacKay 1996). In the case represented by equation (1), trait  $i$  does not feed back upon trait  $j$ . If such feedback occurs, the definition of  $z_i$  is more complicated

$$z_i = \frac{a_i + e_i + \psi_{ij}a'_j + \psi_{ij}e'_j}{(1 - \psi_{ij}\psi_{ji})}. \tag{2}$$

The denominator of equation (2) may be very small when there is strong positive feedback (i.e.,  $\psi_{ij}$  and  $\psi_{ji}$  have the same sign), or very large when there is strong negative feedback ( $\psi_{ij}$  and  $\psi_{ji}$  are of opposite sign).

Moore et al. (1997) calculate an individual's total breeding value ( $A$ ) from its average effect on the population (Falconer and MacKay 1996). When focal individuals also act as social partners, their own social effect is included in their breeding value, thus,

$$A_i = \frac{a_i + \psi_{ij}a_j}{(1 - \psi_{ij}\psi_{ji})}. \tag{3}$$

Note the absence of the prime on the social trait,  $a_j$ . This total breeding value can then be used to calculate the response to selection (Moore et al. 1997).

In contrast, Griffing's models do not attribute social effects to a certain phenotype. Instead, they define a focal individual's phenotype as a simple sum of direct and social components (Griffing 1967; Bijma et al. 2007a). Each of these components may be decomposed into heritable and nonheritable components:

$$z = a_D + e_D + a'_S + e'_S, \tag{4}$$

where subscripts  $D$  and  $S$  represent direct and social effects, respectively. Again, primes are used to show that an effect derives from the phenotype of a social partner on the focal individual. Although this approach easily extends to multiple social partners, we will initially concentrate on a single partner for simplicity and to follow the approach of Moore et al. (1997), saving the treatment of larger groups for a later section. As before, an individual's total breeding value,  $A$ , may be represented by the sum of its direct breeding value and its own social breeding value,

$$A = a_D + a_S. \tag{5}$$

In the absence of feedback, the analogy between the two frameworks is clear. The direct breeding values and environmental values are identical, and  $a_S = \psi_{ij} a'_j$ . However, if feedback occurs, as in equation (3),  $a_D \neq a_i$ . Instead, it appears that  $a_D = \frac{a_i}{(1 - \psi_{ij}\psi_{ji})}$ . Thus, depending on the signs and relative magnitudes of  $\psi_{ij}$  and  $\psi_{ji}$ , an individual's direct breeding value may be greater or less than its additive genetic value. In turn, the social breeding value for trait  $i$  appears to be  $a_S = \frac{\psi_{ij}a_j}{(1 - \psi_{ij}\psi_{ji})}$ .

The total amount of heritable variation in the population, or the variance of total breeding values, can be calculated by taking the expected variance of equation (5)

$$\sigma_A^2 = \sigma_{a_D}^2 + 2\sigma_{a_D a_S} + \sigma_{a_S}^2 \tag{6}$$

(Bijma et al. 2007a). In other words, the variance of total breeding values is equal to the sum of the variances of direct and IGEs, as well as twice the covariance between the two. Similarly, we can calculate the same value in the trait-based framework by taking the expected variance of equation (3)

$$\sigma_{A_i}^2 = \frac{G_{ii} + 2\psi_{ij}G_{ij} + \psi_{ij}^2 G_{jj}}{(1 - \psi_{ij}\psi_{ji})^2}. \tag{7}$$

Here,  $G$  is used rather than  $\sigma_a^2$  to emphasize that these values are elements of the additive genetic variance-covariance matrix  $\mathbf{G}$  (Lande 1979; Moore et al. 1997).

Equation (7) suggests that even in the presence of feedback, we should be able to partition the variance in total breeding values into components due to direct and IGEs, and the covariance between the two, as in equation (6). Setting the components from the right-hand sides of equations (6) and (7) equal to each other, we find

$$\sigma_{a_D}^2 = \frac{G_{ii}}{(1 - \psi_{ij}\psi_{ji})^2} \tag{8a}$$

$$\sigma_{a_D a_S} = \frac{\psi_{ij}G_{ij}}{(1 - \psi_{ij}\psi_{ji})^2} \tag{8b}$$

$$\sigma_{a_S}^2 = \frac{\psi_{ij}^2 G_{jj}}{(1 - \psi_{ij}\psi_{ji})^2}. \tag{8c}$$

## Empirical Estimation of IGEs

### THEORY

Because the components on the left-hand sides of equations (8a–c) are estimable (Muir 2005; Bijma et al. 2007b), these relationships immediately suggest that  $\psi$  coefficients should be estimable as a function of these variance components. The simplest, univariate case, where  $z_i$  is only affected by itself, can be explored by setting  $j = i$  in equations (8a–c). In this case,

$$\psi_{ii} = \frac{\sigma_{a_D a_S}}{\sigma_{a_D}^2}. \tag{9}$$

However, this is probably an uncommon situation. To derive a generalized method for measuring  $\Psi$  and its components, we must first expand both frameworks to incorporate multiple traits.

A multivariate version of the trait-based framework has been derived by Moore et al. (1997), and here we briefly summarize their results while deriving the multivariate version of the variance-components model. Following Moore et al. (1997), we define  $\mathbf{z}$  as a column vector of phenotypic values and  $\Psi$  as a square matrix with elements  $\psi_{ij}$ , or

$$\mathbf{z} = (\mathbf{I} - \Psi\Psi)^{-1}(\mathbf{a} + \mathbf{e} + \Psi\mathbf{a}' + \Psi\mathbf{e}') \quad (10a)$$

In the variance-components framework,

$$\mathbf{z} = \mathbf{a}_D + \mathbf{e}_D + \mathbf{a}'_S + \mathbf{a}'_S. \quad (10b)$$

The vector of total breeding values  $\mathbf{A}$  is then

$$\mathbf{A} = (\mathbf{I} - \Psi\Psi)^{-1}(\mathbf{a} + \Psi\mathbf{a}) \quad (11a)$$

or

$$\mathbf{A} = \mathbf{a}_D + \mathbf{a}_S. \quad (11b)$$

Taking variances,

$$\mathbf{G}_A = (\mathbf{I} - \Psi\Psi)^{-1}(\mathbf{G} + \mathbf{G}\Psi^T + \Psi\mathbf{G} + \Psi\mathbf{G}\Psi^T)(\mathbf{I} - \Psi^T\Psi^T)^{-1}, \quad (12a)$$

where T denotes matrix transposition, or

$$\mathbf{G}_A = \mathbf{G}_D + \mathbf{G}_{DS} + \mathbf{G}_{SD} + \mathbf{G}_S \quad (12b)$$

Additively partitioning  $\mathbf{G}_A$ , we can show that

$$\mathbf{G}_D = (\mathbf{I} - \Psi\Psi)^{-1}\mathbf{G}(\mathbf{I} - \Psi^T\Psi^T)^{-1} \quad (13a)$$

$$\mathbf{G}_{DS} = (\mathbf{I} - \Psi\Psi)^{-1}\mathbf{G}\Psi^T(\mathbf{I} - \Psi^T\Psi^T)^{-1} \quad (13b)$$

$$\mathbf{G}_{SD} = (\mathbf{I} - \Psi\Psi)^{-1}\Psi\mathbf{G}(\mathbf{I} - \Psi^T\Psi^T)^{-1} \quad (13c)$$

$$\mathbf{G}_S = (\mathbf{I} - \Psi\Psi)^{-1}\Psi\mathbf{G}\Psi^T(\mathbf{I} - \Psi^T\Psi^T)^{-1}. \quad (13d)$$

From these equations, we can calculate

$$\Psi = \mathbf{G}_{SD}\mathbf{G}_D^{-1} = (\mathbf{G}_D^{-1}\mathbf{G}_{DS})^T. \quad (14)$$

This relationship is proved in the Appendix.

Note that the relationship described by equation (14) applies equally well to maternally affected traits. If we assume no change in the mean across generations and that maternal phenotypes are not affected by offspring phenotypes (such as parental provisioning influenced by begging, Kölliker et al. 2005), it can be shown that  $\Psi$  can be replaced by  $\mathbf{M}$  in equations (13) and (14). In the maternal case, however, the multipliers  $(\mathbf{I} - \mathbf{M}\mathbf{M})^{-1}$  and  $(\mathbf{I} - \mathbf{M}^T\mathbf{M}^T)^{-1}$  arise not from feedback but rather from the accumulation of maternal effects over multiple generations. Relaxation of these assumptions is likely to introduce some difficulty into the model (Kirkpatrick and Lande 1989; Kölliker et al. 2005).

Disappointingly, it cannot be proven mathematically that this method of measuring  $\mathbf{M}$  is equivalent to that introduced by Lande and Price (1989). This is because the derivation of the earlier method relies upon  $\mathbf{P}$ , the phenotypic variance-covariance, which cannot be defined explicitly in maternal effects theory

(Kirkpatrick and Lande 1989). Demonstrating equivalence between the two methods will have to await sufficient empirical data.

Because the elements of  $\Psi$  are regression coefficients, their magnitudes will depend upon the means and variances of the traits involved. For a single trait that affects itself, or for two traits with identical direct genetic variances, the expected range of  $\psi$  is between  $-1$  and  $1$ . However, when variances of the two traits differ, the expected limits can be breached. To compare the elements of  $\Psi$  across studies and between traits within a study, we recommend that each trait be standardized to zero mean and unit variance prior to analysis so that standardized estimates of  $\Psi$  will be generated.

### VALIDATION OF METHODOLOGY

To validate equations (9) and (14) numerically, we simulated datasets with the desired parameters and then analyzed them using restricted maximum likelihood and an animal model (Kruuk 2004; Bijma et al. 2007b). First, we simulated base phenotypes (i.e., phenotypes that were not affected by social interactions) using WOMBAT (Meyer 2006) and a two-generation pedigree. The parental generation consisted of 100 sires and 200 dams. Each sire was mated to two unique dams to produce 200 families of 10 offspring each (2000 total). In simulation 1, WOMBAT generated phenotypic values for one trait with a mean of 0, genetic variance of 0.3, and environmental variance of 0.7. Ten replicate populations were generated. In simulation 2, we simulated two traits each with a mean of 0, genetic variance of 0.3, and environmental variance of 0.7. Genetic and environmental covariance was set equal to 0.1.

These base phenotypes and a range of values of  $\psi$  were then used to calculate interacting phenotypes in the program ASReml 2.0 (Gilmour et al. 2006). First, each individual was randomly paired with another individual from its own generation. Then, phenotypic values were calculated using equation (10a). Each pair was represented twice in each dataset, with each of the two interactants acting as the focal individual. In simulation 1, we varied  $\psi_{11}$  from  $-0.8$  to  $0.8$  by increments of  $0.2$ . In simulation 2, we varied  $\psi_{12}$  and  $\psi_{21}$ , using the 15 unique combinations of  $-0.6, -0.2, 0, 0.2,$  and  $0.6$ .

We then used ASReml to perform a quantitative genetic analysis of these new phenotypic data. Using restricted maximum likelihood and an animal model, phenotypic variance was partitioned into direct additive genetic and social additive genetic components ( $\mathbf{G}_D$  and  $\mathbf{G}_S$ ), covariance between direct and social breeding values ( $\mathbf{G}_{SD}$ ), environmental covariance between social partners, and a residual component. Environmental covariance was modeled by including a random environmental effect of pair. The expected value for this component is

**Table 1.** Predicted and observed variance/covariance components and interaction coefficient,  $\psi_{11}$ , from one-trait simulation. No observed components fell outside  $\pm 3$  SE of the predicted value.

Predicted				Observed			
$\psi_{11}$	$\sigma_{a_D}^2$	$\sigma_{a_D a_S}$	$\sigma_{a_S}^2$	$\psi_{11}$	$\sigma_{a_D}^2$	$\sigma_{a_D a_S}$	$\sigma_{a_S}^2$
-0.8	2.315	-1.852	1.481	-0.794 (0.007)	2.457 (0.136)	-1.959 (0.121)	1.556 (0.109)
-0.6	0.732	-0.439	0.264	-0.601 (0.009)	0.807 (0.033)	-0.487 (0.025)	0.288 (0.021)
-0.4	0.425	-0.170	0.068	-0.396 (0.011)	0.453 (0.021)	-0.181 (0.013)	0.067 (0.011)
-0.2	0.326	-0.065	0.013	-0.207 (0.012)	0.347 (0.015)	-0.072 (0.006)	0.009 (0.006)
0	0.300	0.000	0.000	-0.011 (0.012)	0.319 (0.013)	-0.004 (0.004)	-0.006 (0.005)
0.2	0.326	0.065	0.013	0.185 (0.011)	0.344 (0.013)	0.063 (0.004)	0.006 (0.005)
0.4	0.425	0.170	0.068	0.384 (0.011)	0.446 (0.017)	0.170 (0.007)	0.060 (0.007)
0.6	0.732	0.439	0.264	0.585 (0.009)	0.761 (0.028)	0.445 (0.018)	0.254 (0.015)
0.8	2.315	1.852	1.481	0.790 (0.006)	2.380 (0.089)	1.880 (0.074)	1.480 (0.066)

$$\begin{aligned} \mathbf{E}_{pair} &= \text{Cov}[(\mathbf{I} - \Psi\Psi)^{-1}(\mathbf{e} + \Psi\mathbf{e}'), (\mathbf{e}^T + \mathbf{e}^T\Psi^T)(\mathbf{I} - \Psi^T\Psi^T)^{-1}] \\ &= (\mathbf{I} - \Psi\Psi)^{-1}(\Psi\mathbf{E} + \mathbf{E}\Psi^T)(\mathbf{I} - \Psi^T\Psi^T)^{-1}, \end{aligned} \tag{15}$$

where  $\mathbf{E}$  is the variance-covariance matrix of environmental effects. Including this covariance term controls for indirect environmental effects that are mediated by social interactions (Bijma et al. 2007b). In an experimental situation, this term would also control for sources of common environmental influence on the members of a pair. Because this matrix consists entirely of covariances, the diagonal terms were allowed to take on negative values. Using equation (A6) of Moore et al. (1997) for the phenotypic variance-covariance matrix  $\mathbf{P}$ , the expected residual term is

$$\mathbf{R} = (\mathbf{I} - \Psi\Psi)^{-1}(\mathbf{E} + \Psi\mathbf{E}\Psi^T - \Psi\mathbf{E} - \mathbf{E}\Psi^T)(\mathbf{I} - \Psi^T\Psi^T)^{-1}. \tag{16}$$

Genetic variance components in the model were compared to their expected values generated by equations (13a-d). We then compared calculated values of  $\psi_{11}$  or  $\Psi$  from equation (9) or (14), and compared these values with those used to generate the data.

Analysis of both simulations produced accurate, unbiased estimates of genetic variance components (Tables 1-4). In only three cases did the predicted value of a parameter fall outside  $\pm 3$  SE of the mean estimate. Equations (9) and (14) also produced accurate and unbiased estimates of  $\psi_{11}$  and  $\Psi$  (Tables 1 and 5). In simulation 1, predicted and calculated  $\psi_{11}$  were highly

**Table 2.** Predicted and observed direct genetic variance-covariance matrices from two-trait simulation. No observed components fell outside  $\pm 3$  SE of the predicted value.

Predicted				Observed			
$\psi_{12}, \psi_{21}$	$\mathbf{G}_D(1,1)$	$\mathbf{G}_D(1,2)$	$\mathbf{G}_D(2,2)$	$\mathbf{G}_D(1,1)$	$\mathbf{G}_D(1,2)$	$\mathbf{G}_D(2,2)$	
-0.6, -0.6	0.732	0.244	0.732	0.782 (0.042)	0.245 (0.027)	0.720 (0.033)	
-0.6, -0.2	0.387	0.129	0.387	0.415 (0.022)	0.127 (0.013)	0.382 (0.017)	
-0.6, 0	0.300	0.100	0.300	0.321 (0.017)	0.097 (0.010)	0.296 (0.014)	
-0.6, 0.2	0.239	0.080	0.239	0.256 (0.013)	0.076 (0.007)	0.235 (0.011)	
-0.2, -0.2	0.326	0.109	0.326	0.345 (0.014)	0.105 (0.010)	0.323 (0.014)	
-0.2, 0	0.300	0.100	0.300	0.320 (0.013)	0.096 (0.009)	0.297 (0.014)	
0, 0	0.300	0.100	0.300	0.319 (0.012)	0.095 (0.009)	0.297 (0.014)	
0.2, -0.2	0.277	0.092	0.277	0.289 (0.012)	0.089 (0.009)	0.275 (0.012)	
0.2, 0	0.300	0.100	0.300	0.313 (0.013)	0.095 (0.009)	0.297 (0.014)	
0.2, 0.2	0.326	0.109	0.326	0.339 (0.014)	0.102 (0.009)	0.321 (0.016)	
0.6, -0.6	0.162	0.054	0.162	0.162 (0.009)	0.051 (0.006)	0.159 (0.007)	
0.6, -0.2	0.239	0.079	0.239	0.239 (0.014)	0.074 (0.008)	0.236 (0.010)	
0.6, 0	0.300	0.100	0.300	0.299 (0.017)	0.093 (0.010)	0.296 (0.014)	
0.6, 0.2	0.387	0.129	0.387	0.386 (0.022)	0.120 (0.012)	0.381 (0.019)	
0.6, 0.6	0.732	0.244	0.732	0.728 (0.044)	0.223 (0.023)	0.713 (0.042)	

**Table 3.** Predicted and observed social-direct genetic covariance matrices from two-trait simulation. One observed component, indicated by an asterisk, fell outside  $\pm 3$  SE of the predicted value.

Predicted					Observed			
$\psi_{12}, \psi_{21}$	$G_{SD}(1,1)$	$G_{SD}(1,2)$	$G_{SD}(2,1)$	$G_{SD}(2,2)$	$G_{SD}(1,1)$	$G_{SD}(1,2)$	$G_{SD}(2,1)$	$G_{SD}(2,2)$
-0.6, -0.6	-0.146	-0.439	-0.439	-0.146	-0.156 (0.021)	-0.428 (0.023)	-0.473 (0.033)	-0.152 (0.021)
-0.6, -0.2	-0.077	-0.232	-0.077	-0.026	-0.082 (0.011)	-0.230 (0.011)	-0.086 (0.010)	-0.028 (0.008)
-0.6, 0	-0.060	-0.180	0.000	0.000	-0.064 (0.009)	-0.179 (0.008)	-0.003 (0.006)	-0.002 (0.006)
-0.6, 0.2	-0.048	-0.143	0.048	0.016	-0.051 (0.007)	-0.144 (0.006)	0.049 (0.005)	0.014 (0.005)
-0.2, -0.2	-0.022	-0.065	-0.065	-0.022	-0.026 (0.006)	-0.066 (0.006)	-0.076 (0.007)	-0.023 (0.007)
-0.2, 0	-0.020	-0.060	0.000	0.000	-0.024 (0.005)	-0.062 (0.005)	-0.007 (0.005)	-0.002 (0.006)
0, 0	0.000	0.000	0.000	0.000	-0.004 (0.004)	-0.002 (0.005)	-0.008 (0.005)	-0.002 (0.006)
0.2, -0.2	0.018	0.055	-0.055	-0.018	0.014 (0.003)	0.056 (0.006)	-0.067 (0.005)	-0.020 (0.006)
0.2, 0	0.020	0.060	0.000	0.000	0.016 (0.003)	0.059 (0.007)	-0.010 (0.005)	-0.002 (0.004)
0.2, 0.2	0.022	0.065	0.065	0.022	0.017 (0.003)	0.062 (0.007)	0.058 (0.006)	0.019 (0.007)
0.6, -0.6	0.032	0.097	-0.097	-0.032	0.029 (0.003)	0.098 (0.005)	-0.107 (0.005)	-0.033 (0.004)
0.6, -0.2	0.048	0.143	-0.048	-0.016	0.043 (0.004)	0.142 (0.008)	-0.060 (0.004)*	-0.018 (0.005)
0.6, 0	0.060	0.180	0.000	0.000	0.054 (0.006)	0.177 (0.011)	-0.014 (0.005)	0.002 (0.006)
0.6, 0.2	0.077	0.232	0.077	0.026	0.069 (0.007)	0.227 (0.015)	0.062 (0.009)	0.022 (0.008)
0.6, 0.6	0.146	0.439	0.439	0.146	0.130 (0.013)	0.422 (0.033)	0.415 (0.032)	0.132 (0.021)

correlated ( $r = 0.998$ , Fig. 1). Calculated values of  $\psi_{ij}$  generated in simulation 2 were less precise, but still highly correlated with predicted values ( $r = 0.979$ , Fig. 2).

### Extension to Larger Groups

Although the variance-components model of IGEs has been derived for groups of any size, the trait-based model of Moore et al. (1997) considers only pairs of individuals. However, there are

many cases in which exploring trait-based interactions in larger groups may be of interest. For example, advertising males may adjust their display behavior based on the displays of a number of neighboring males, siblings within a nest may compete for resources, or aggregations of insect larvae may affect the antipredator response of individuals. To account for such cases here, we expand the trait-based model to include multiple social partners. We also show that our experimental method to measure  $\Psi$  can be applied to larger groups.

**Table 4.** Predicted and observed social genetic variance-covariance matrices from two-trait simulation. Two observed components, indicated by asterisks, fell outside  $\pm 3$  SE of the predicted value.

Predicted				Observed		
$\psi_{12}, \psi_{21}$	$G_S(1,1)$	$G_S(1,2)$	$G_S(2,2)$	$G_S(1,1)$	$G_S(1,2)$	$G_S(2,2)$
-0.6, -0.6	0.264	0.088	0.264	0.248 (0.020)	0.099 (0.017)	0.278 (0.027)
-0.6, -0.2	0.139	0.015	0.015	0.132 (0.010)	0.020 (0.007)	0.010 (0.006)
-0.6, 0	0.108	0.000	0.000	0.102 (0.008)	0.003 (0.005)	-0.008 (0.003)
-0.6, 0.2	0.086	-0.010	0.010	0.082 (0.006)	-0.007 (0.004)	0.002 (0.003)*
-0.2, -0.2	0.013	0.004	0.013	0.008 (0.005)	0.008 (0.005)	0.010 (0.005)
-0.2, 0	0.012	0.000	0.000	0.007 (0.005)	0.003 (0.004)	-0.007 (0.003)
0, 0	0.000	0.000	0.000	-0.006 (0.005)	0.002 (0.004)	-0.007 (0.003)
0.2, -0.2	0.011	-0.004	0.011	0.006 (0.005)	-0.001 (0.004)	0.008 (0.004)
0.2, 0	0.012	0.000	0.000	0.006 (0.006)	0.002 (0.004)	-0.007 (0.003)
0.2, 0.2	0.013	0.004	0.013	0.007 (0.006)	0.005 (0.005)	0.003 (0.003)*
0.6, -0.6	0.058	-0.019	0.058	0.055 (0.006)	-0.017 (0.003)	0.063 (0.005)
0.6, -0.2	0.086	-0.010	0.010	0.081 (0.009)	-0.007 (0.005)	0.008 (0.003)
0.6, 0	0.108	0.000	0.000	0.102 (0.012)	0.007 (0.006)	-0.007 (0.003)
0.6, 0.2	0.139	0.015	0.015	0.131 (0.015)	0.015 (0.008)	0.002 (0.005)
0.6, 0.6	0.264	0.088	0.264	0.244 (0.028)	0.080 (0.015)	0.229 (0.005)

**Table 5.** Predicted and observed interaction matrix,  $\Psi$ , from two-trait simulation. No observed components fell outside  $\pm 3$  SE of the predicted value.

Predicted				Observed			
$\psi_{11}$	$\psi_{12}$	$\psi_{21}$	$\psi_{22}$	$\psi_{11}$	$\psi_{12}$	$\psi_{21}$	$\psi_{22}$
0	-0.6	-0.6	0	-0.015 (0.010)	-0.586 (0.012)	-0.598 (0.012)	-0.004 (0.009)
0	-0.6	-0.2	0	-0.015 (0.015)	-0.595 (0.017)	-0.199 (0.016)	-0.004 (0.011)
0	-0.6	0	0	-0.014 (0.018)	-0.602 (0.021)	-0.0001 (0.018)	-0.004 (0.014)
0	-0.6	0.2	0	-0.011 (0.021)	-0.613 (0.026)	0.199 (0.020)	-0.003 (0.016)
0	-0.2	-0.2	0	-0.013 (0.014)	-0.198 (0.018)	-0.219 (0.015)	0.0004 (0.014)
0	-0.2	0	0	-0.011 (0.015)	-0.205 (0.019)	-0.020 (0.016)	0.001 (0.015)
0	0	0	0	-0.010 (0.014)	-0.003 (0.020)	-0.028 (0.015)	0.003 (0.015)
0	0.2	-0.2	0	-0.012 (0.014)	0.206 (0.020)	-0.237 (0.017)	0.003 (0.016)
0	0.2	0	0	-0.009 (0.014)	0.199 (0.020)	-0.035 (0.016)	0.004 (0.016)
0	0.2	0.2	0	-0.007 (0.014)	0.192 (0.021)	0.165 (0.014)	0.005 (0.016)
0	0.6	-0.6	0	-0.027 (0.028)	0.630 (0.032)	-0.683 (0.036)	0.006 (0.025)
0	0.6	-0.2	0	-0.015 (0.021)	0.609 (0.023)	-0.268 (0.028)	0.007 (0.019)
0	0.6	0	0	-0.011 (0.018)	0.601 (0.021)	-0.062 (0.025)	0.007 (0.017)
0	0.6	0.2	0	-0.007 (0.015)	0.595 (0.019)	0.144 (0.021)	0.007 (0.015)
0	0.6	0.6	0	-0.002 (0.011)	0.585 (0.018)	0.558 (0.016)	0.006 (0.012)

Following Griffing (1967) and Bijma et al. (2007a), we assume that the population of interest is subdivided into a number of distinct groups. For simplicity, we assume that all groups are of the same size, all interactions occur only within these groups, and that within groups, all possible interactions between group members occur simultaneously and with equal strength. As in the variance-components model, the phenotype of a focal individual is partitioned into a direct genetic component, a direct residual

component, and the sum of the social effects of all the members of its group,

$$\mathbf{z} = \mathbf{a} + \mathbf{e} + (n - 1)\Psi\bar{\mathbf{z}}, \tag{17}$$

where  $\bar{\mathbf{z}}$  represents the mean phenotype of the focal individual's social partners and  $n$  represents the group size (i.e., the focal individual plus the number of social partners). Note that as defined here,  $\Psi$  represents the strength of each interaction between two individuals in a group. If one wished to measure the strength of an interaction between one individual and the rest of its group, a new variable could be defined such that

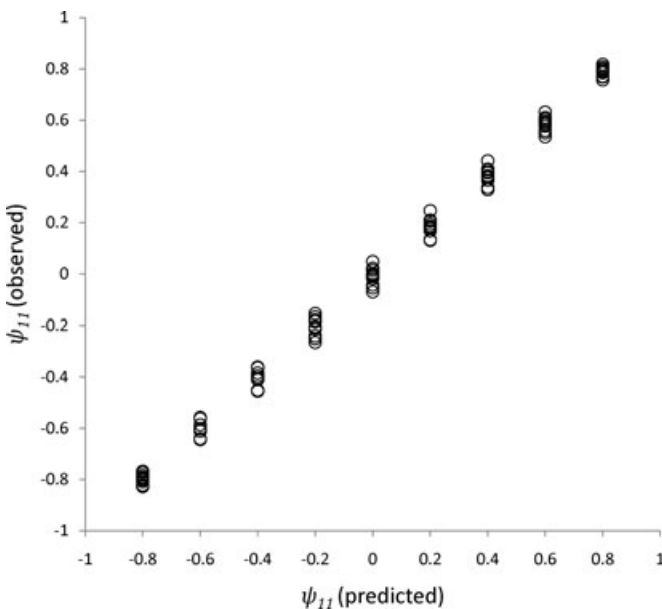
$$\Psi_{group} = (n - 1)\Psi \tag{18}$$

This identity can be used to transform the following derivation so that group-level interactions will be measured. A graphical depiction of the difference between  $\Psi$  and  $\Psi_{group}$  is provided in Figure 3. In some cases, the  $\Psi_{group}$  formulation may be desirable because it more accurately describes the dynamics of the interaction. For instance, it may be easier to describe the behavior of a single fish as affected by an emergent property of its school.

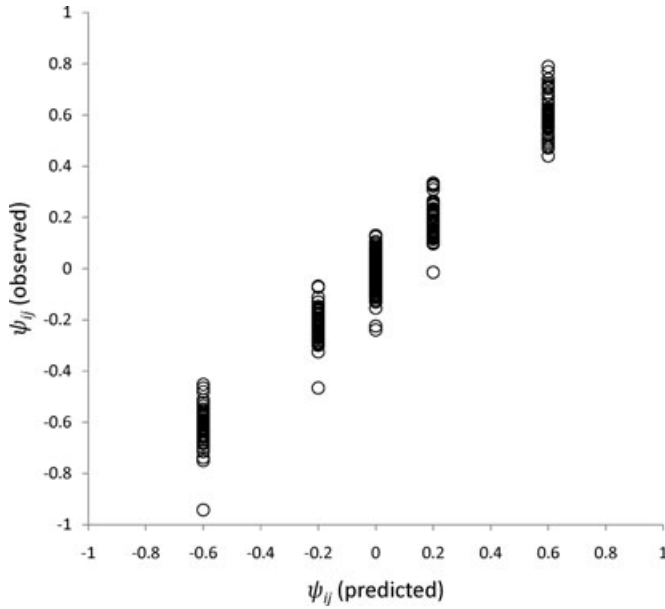
The expression of traits in the social partners will be affected by their interaction with the focal individual as well as with the other group members. Thus, we can define the vector of social partner phenotypes as

$$\mathbf{Z}^i = \mathbf{a}^i + \mathbf{e}^i + (n - 1)\Psi\bar{\mathbf{z}} - \Psi\bar{\mathbf{z}}^i + \Psi\mathbf{z}, \tag{19}$$

where the superscript  $i$  indicates an individual social partner. The last two terms in equation (19) demonstrate that each social partner



**Figure 1.** Predicted and observed interaction coefficients,  $\psi_{11}$ , from one-trait simulation.



**Figure 2.** Predicted and observed interaction coefficients,  $\psi_{ij}$ , from two-trait simulation.

interacts with the focal individual from equation (17), but not with itself. We can solve for  $\bar{z}'$  by taking the expectation of equation (19),

$$\bar{z}' = \bar{a}' + \bar{e}' + (n - 1)\Psi\bar{z}' - \Psi\bar{z}' + \Psi z.$$

Simplifying,

$$\bar{z}' = \bar{a}' + \bar{e}' + (n - 2)\Psi\bar{z}' + \Psi z$$

and

$$\bar{z}' = [\mathbf{I} - (n - 2)\Psi]^{-1}[\bar{a}' + \bar{e}' + \Psi z], \tag{20}$$

we can now solve for an explicit definition of  $z$  by substituting equation (20) into equation (17),

$$z = a + e + (n - 1)\Psi[\mathbf{I} - (n - 2)\Psi]^{-1}[\bar{a}' + \bar{e}' + \Psi z]$$

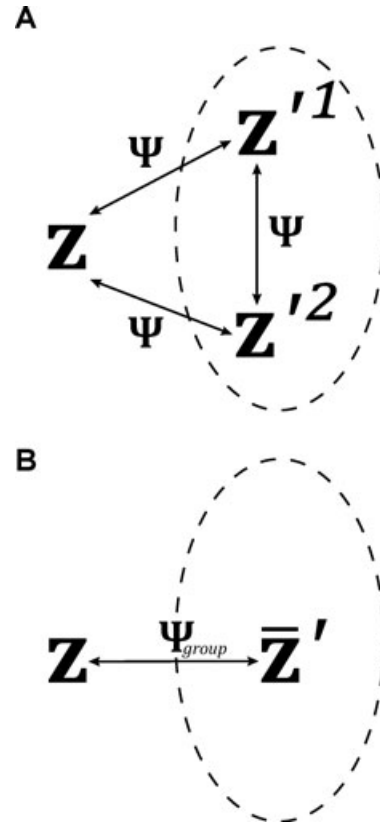
$$z = [\mathbf{I} - (n - 1)\Psi[\mathbf{I} - (n - 2)\Psi]^{-1}\Psi]^{-1} \cdot [a + e + (n - 1)\Psi[\mathbf{I} - (n - 2)\Psi]^{-1}(\bar{a}' + \bar{e}')]. \tag{21}$$

To simplify notation, we write

$$\mathbf{U} = \mathbf{I} - (n - 1)\Psi[\mathbf{I} - (n - 2)\Psi]^{-1}\Psi \tag{22a}$$

$$\mathbf{V} = \mathbf{I} - (n - 2)\Psi \tag{22b}$$

$$z = \mathbf{U}^{-1}[a + e + (n - 1)\Psi\mathbf{V}^{-1}(\bar{a}' + \bar{e}')] \tag{22c}$$



**Figure 3.** In the model for IGEs in groups larger than 2, it is assumed that all interactions occur symmetrically and simultaneously between group members. These illustrations depict groups of  $n = 3$ . (A) Here, interactions are depicted as occurring between individuals of the group. The arbitrarily chosen focal individual is shown outside the ellipse. The focal individual's traits ( $z$ ) are affected directly by the traits of its social partners ( $z'$ ) within the ellipse as well indirectly by the interactions among these social partners. The strength of each individual interaction is measured by  $\Psi$  (as shown in eq. 17). (B) Equivalently, this situation can be depicted as a single interaction between a focal individual's traits and the average trait values of its social group ( $\bar{z}'$ ). Here, the strength of the interaction is measured by  $\Psi_{group}$ , which is equal to  $(n - 1)\Psi$  (eq. 18). In this formulation, interactions among individuals within the social group are implicit.

Then, from the expectation across all social groups, we can find the total breeding value of the focal individual,

$$\mathbf{A} = \mathbf{U}^{-1}[a + (n - 1)\Psi\mathbf{V}^{-1}a]. \tag{23}$$

To measure  $\Psi$ , we must partition  $\mathbf{A}$  into its components as before,

$$a_D = \mathbf{U}^{-1}a \tag{24a}$$

$$(n - 1)a_S = \mathbf{U}^{-1}(n - 1)\Psi\mathbf{V}^{-1}a$$

$$a_S = \mathbf{U}^{-1}\Psi\mathbf{V}^{-1}a. \tag{24b}$$

Taking variances of equations (23a–b) and the covariance between them, we find that the variance components estimated by an animal model are

$$\mathbf{G}_D = \mathbf{U}^{-1} \mathbf{G} (\mathbf{U}^T)^{-1} \quad (25a)$$

$$\mathbf{G}_{DS} = \mathbf{U}^{-1} \mathbf{G} (\mathbf{V}^T)^{-1} \boldsymbol{\Psi}^T (\mathbf{U}^T)^{-1} \quad (25b)$$

$$\mathbf{G}_{SD} = \mathbf{U}^{-1} \boldsymbol{\Psi} \mathbf{V}^{-1} \mathbf{G} (\mathbf{U}^T)^{-1} \quad (25c)$$

$$\mathbf{G}_S = \mathbf{U}^{-1} \boldsymbol{\Psi} \mathbf{V}^{-1} \mathbf{G} (\mathbf{V}^T)^{-1} \boldsymbol{\Psi}^T (\mathbf{U}^T)^{-1}. \quad (25d)$$

From these identities, it can be shown that:

$$\boldsymbol{\Psi} = \mathbf{G}_{SD} [\mathbf{G}_D + (n - 2) \mathbf{G}_{SD}^{-1}]. \quad (26)$$

A proof is given in the Appendix.

Equation 26 suggests that the maximum absolute value of the components of  $\boldsymbol{\Psi}$  should decrease as group size increases. However, note that by equation (18), the total strength of the group interaction,  $\boldsymbol{\Psi}_{group}$ , still has the potential to be large.

It must be noted here that the equations derived in this section are precisely applicable only to populations that fit the assumptions discussed above. Although these assumptions are generally met in experimental designs used to measure IGEs (Bijma et al. 2007b; Bleakley and Brodie 2009), the relationships derived here should be applied with caution elsewhere. One major complication is that in natural populations, patterns of interaction may be complex, and “groups” may be difficult to define. Social network analysis may be used to assist in the description of these patterns (Wey et al. 2008), but this complexity has not yet been incorporated into quantitative genetic models.

Furthermore, measurements of  $\boldsymbol{\Psi}$  or  $\boldsymbol{\Psi}_{group}$  are specific to a given group size and do not translate to parameters for groups of different sizes. One reason this is true is that as group size increases, any given individual’s effect on its group members will be more diffuse. Under our assumptions, this means that the maximum covariance between any two individuals in the group will decrease with group size, leading to differences in observed  $\boldsymbol{\Psi}$  with group size. Another reason is that observed  $\boldsymbol{\Psi}$  depends not only upon direct interactions between a given focal individual and its  $n - 1$  social partners, but also upon the indirect effects of interactions among the social partners (Fig. 3A). As  $n$  increases, the number of interactions between pairs of group members increases by  $n!/(n - 2)!$ , and each of these interactions is expected to have a smaller effect on the phenotype of a given group member. It is possible that an extension of the theory presented here, in combination with statistical methods described by Hadfield and Wilson (2007), may be used to examine the effects of variation in group size on  $\boldsymbol{\Psi}$ .

## Discussion

We have shown that the two major IGE models provide equivalent results, and that the relationships between the two can be used to estimate the matrix  $\boldsymbol{\Psi}$  (eqs. 9, 14, and 26), which measures the strength of IGEs in the framework of Moore et al. (1997). By extension, our methods are also applicable to the maternal effects matrix  $\mathbf{M}$  (Kirkpatrick and Lande 1989). Although both theoretical frameworks can be used to provide evolutionary predictions, as we will demonstrate elsewhere (J. W. McGlothlin and E. D. Brodie III, unpubl. ms.), measurements of  $\boldsymbol{\Psi}$  and  $\mathbf{M}$  are crucial for dissecting the potentially complex pattern of covariance among interacting phenotypes. Similar to selection gradients, these measurements may be used to generate hypotheses guiding the design of further experiments (Lande and Arnold 1983; Wade and Kalisz 1990).

The direct and indirect components of variance in breeding values that are necessary to estimate interaction coefficients,  $\boldsymbol{\Psi}$ , are readily estimable with available statistical programs. Many large-scale quantitative genetic studies, both from laboratory and natural populations, are likely to fit the requirements for estimating IGEs, which are thoroughly discussed by Bijma et al. (2007b). The primary criteria are the abilities to distinguish additive genetic variance and to separate direct from indirect genetic variance. Most paternal half-sibling breeding designs or multi-generational pedigree can be used to accomplish these goals. For studies of maternal effects, achieving the second criteria is more delicate, often requiring extra generations or complicated cross-fostering schemes to separate maternal and direct genetic effects (Cheverud and Moore 1994; Lynch and Walsh 1998). However, studies of IGEs that occur within a generation and/or between unrelated individuals are less complicated. Here, designs must simply include pairs or groups of interacting individuals that are assigned (or occur in nature) at random, or nearly so (Bijma et al. 2007b). It is important to note that there need be no special distinction between “focal individuals” and “social partners” in these experiments. What is important is that both members of a pair (or all members of a group) have known pedigrees.

Most studies measuring IGEs have concentrated on a single phenotype, such as body size (Wolf 2003) or have measured multiple traits but considered them separately (Wilson et al. 2009). However, to estimate  $\boldsymbol{\Psi}$ , all traits must be considered together in a multivariate analysis. The reason behind this is simple. When two traits interact with each other, that is, when  $\psi_{12}$  and  $\psi_{21}$  are not equal to zero, the covariances  $\text{Cov}(a_{D1}, a_{S2})$  and  $\text{Cov}(a_{D2}, a_{S1})$  are both likely to be large, whereas the covariances  $\text{Cov}(a_{D1}, a_{S1})$  and  $\text{Cov}(a_{D2}, a_{S2})$  may be equal to zero if there is no additive genetic correlation between the traits. Univariate analyses measure only the latter pair of covariances, and thus may fail to detect evidence for IGEs even when strong IGEs are present.

IGEs are expected to play a substantial role in the evolution of all traits involved in interactions among conspecifics, from competitive ability in plants to aggression and dominance in animal societies (Moore et al. 1997; Wolf et al. 1998). Furthermore, IGEs are expected to be a key to understanding multilevel selection and the evolution of social interactions among nonrelatives (Griffing 1967, 1981; Muir 1996; Wolf et al. 1999; Bijma et al. 2007a; Bijma and Wade 2008; Bleakley and Brodie 2009). In many cases, the key questions hinge upon the sign and magnitude of interaction coefficients and the direct-social covariances of specific trait combinations. Unfortunately, the empirical study of IGEs has lagged behind theory, due to both the difficulty of collecting data and the lack of a statistically clear estimation approach. The translation of readily estimable direct and social variance components to parameters in trait-based phenotypic models presented here should provide the route for the expansion of our empirical understanding of the importance of IGEs in natural systems.

#### ACKNOWLEDGMENTS

We thank C. Goodnight, W. Muir, B. H. Bleakley, V. Formica, and A. Moore for helpful discussions and comments on the manuscript. This research was supported by NSF grant DEB-0650078.

#### LITERATURE CITED

- Arnold, S. J. 1994. Multivariate inheritance and evolution: a review of concepts. Pp. 17–48 in C. R. Boake, ed. *Quantitative genetic studies of behavioral evolution*. Univ. of Chicago Press, Chicago.
- Bergsma, R., E. Kanis, E. F. Knol, and P. Bijma. 2008. The contribution of social effects to heritable variation in finishing traits of domestic pigs (*Sus scrofa*). *Genetics* 178:1559–1570.
- Bijma, P., and M. J. Wade. 2008. The joint effects of kin, multilevel selection and indirect genetic effects on response to genetic selection. *J. Evol. Biol.* 21:1175–1188.
- Bijma, P., W. M. Muir, and J. A. M. Van Arendonk. 2007a. Multilevel selection I: quantitative genetics of inheritance and response to selection. *Genetics* 175:277–288.
- Bijma, P., W. M. Muir, E. D. Ellen, J. B. Wolf, and J. A. M. Van Arendonk. 2007b. Multilevel selection 2: estimating the genetic parameters determining inheritance and response to selection. *Genetics* 175:289–299.
- Bleakley, B. H., and E. D. Brodie III. 2009. Indirect genetic effects influence antipredator behavior in guppies: estimates of the coefficient of interaction  $\psi$  and the inheritance of reciprocity. *Evolution* DOI: 10.1111/j.1558-5646.2009.00672.x
- Brommer, J. E., and K. Rattiste. 2008. “Hidden” reproductive conflict between mates in a wild bird population. *Evolution* 62:2326–2333.
- Cheverud, J. M. 1984. Evolution by kin selection: a quantitative genetic model illustrated by maternal performance in mice. *Evolution* 38:766–777.
- Cheverud, J. M., and A. J. Moore. 1994. Quantitative genetics and the role of the environment provided by relatives in the evolution of behavior. Pp. 60–100 in C. R. Boake, ed. *Quantitative Genetic Studies of Behavioral Evolution*. Univ. of Chicago Press, Chicago.
- Ellen, E. D., J. Visscher, J. A. M. van Arendonk, and P. Bijma. 2008. Survival of laying hens: genetic parameters for direct and associative effects in three purebred layer lines. *Poult. Sci.* 87:233–239.
- Falconer, D. S. 1965. Maternal effects and selection response. *Proc. Int. Cong. Genet.* 3:763–774.
- Falconer, D. S., and T. F. C. MacKay. 1996. *Introduction to quantitative genetics*. Prentice Hall, Harlow, England.
- Gilmour, A. R., B. J. Gogel, B. R. Cullis, and R. Thompson. 2006. *ASReml user guide release 2.0*. VSN International Ltd, Hemel Hempstead, UK.
- Griffing, B. 1967. Selection in reference to biological groups. I. Individual and group selection applied to populations of unordered groups. *Aust. J. Biol. Sci.* 20:127–139.
- . 1969. Selection in reference to biological groups. III. Generalized results of individual and group selection in terms of parent-offspring covariances. *Aust. J. Biol. Sci.* 21:1171–1178.
- . 1976. Selection in reference to biological groups. V. Analysis of full-sib groups. *Genetics* 82:703–722.
- . 1981. A theory of natural selection incorporating interaction among individuals. I. The modeling process. *J. Theor. Biol.* 89:635–658.
- Hadfield, J. D., and A. J. Wilson. 2007. Multilevel selection 3: modeling the effects of interacting individuals as a function of group size. *Genetics* 17:667–668.
- Kent, C., R. Azanchi, B. Smith, A. Formosa, and J. D. Levine. 2008. Social context influences chemical communications in *D. melanogaster* males. *Curr. Biol.* 18:1384–1389.
- Kirkpatrick, M., and R. Lande. 1989. The evolution of maternal characters. *Evolution* 43:485–503.
- Kölliker, M., E. D. Brodie III, and A. J. Moore. 2005. The coadaptation of parental supply and offspring demand. *Am. Nat.* 166:506–516.
- Kruuk, L. E. B. 2004. Estimating genetic parameters in natural populations using the ‘animal model’. *Philos. Trans. R. Soc. Lond. B* 359:873–890.
- Lande, R. 1979. Quantitative genetic analysis of multivariate evolution, applied to brain:body size allometry. *Evolution* 33:402–416.
- Lande, R., and S. J. Arnold. 1983. The measurement of selection on correlated characters. *Evolution* 37:1210–1226.
- Lande, R., and M. Kirkpatrick. 1990. Selection response in traits with maternal inheritance. *Genet. Res.* 55:189–197.
- Lande, R., and T. Price. 1989. Genetic correlations and maternal-effect coefficients obtained from offspring-parent regression. *Genetics* 122:915–922.
- Lynch, M. 1987. Evolution of intrafamilial interactions. *Proc. Natl. Acad. Sci. USA* 84:8507–8511.
- Lynch, M., and B. Walsh. 1998. *Genetics and analysis of quantitative traits*. Sinauer Associates, Sunderland, MA.
- Merilä, J., and M. Björklund. 2004. Phenotypic integration as a constraint and adaptation. Pp. 107–129 in M. Pigliucci, and K. Preston, eds. *Phenotypic Integration: studying the ecology and evolution of complex phenotypes*. Oxford Univ. Press, Oxford.
- Meyer, K. 2006. WOMBAT – A program for mixed model analyses by restricted maximum likelihood. User notes. Animal Genetics and Breeding Unit, Armidale, New South Wales.
- Moore, A. J., E. D. Brodie III, and J. B. Wolf. 1997. Interacting phenotypes and the evolutionary process: I. Direct and indirect genetic effects of social interactions. *Evolution* 51:1352–1362.
- Mousseau, T. A., and C. W. Fox, eds. 1998. *Maternal effects as adaptations*. Oxford Univ. Press, New York.
- Muir, W. M. 1996. Group selection for adaptation to multiple-hen cages: selection program and direct responses. *Poult. Sci.* 75:447–458.
- . 2005. Incorporation of competitive effects in forest tree or animal breeding programs. *Genetics* 170:1247–1259.
- Petfield, D., S. F. Chenoweth, H. D. Rundle, and M. W. Blows. 2005. Genetic variance in female condition predicts indirect genetic variance in male sexual display traits. *Proc. Natl. Acad. Sci. USA* 102:6045–6050.
- Räsänen, K., and L. E. B. Kruuk. 2007. Maternal effects and evolution at ecological time-scales. *Funct. Ecol.* 21:408–421.
- Roff, D. A. 1997. *Evolutionary quantitative genetics*. Chapman & Hall, New York.

———. 1998. The detection and measurement of maternal effects. Pp. 83–96 in T. A. Mousseau, and C. W. Fox, eds. *Maternal effects as adaptations*. Oxford Univ. Press, New York.

Schluter, D., and L. Gustafsson. 1993. Maternal inheritance of condition and clutch size in the collared flycatcher. *Evolution* 47:658–667.

Wade, M. J., and S. Kalisz. 1990. The causes of natural selection. *Evolution* 44:1947–1955.

Wey, T., D. T. Blumstein, W. Shen, and F. Jordán. 2008. Social network analysis of animal behaviour: a promising tool for the study of sociality. *Anim. Behav.* 75:333–344.

Willham, R. L. 1963. The covariance between relatives for characters composed of components contributed by related individuals. *Biometrics* 19:18–27.

———. 1972. The role of maternal effects in animal breeding. III. Biometrical aspects of maternal effects in animals. *J. Anim. Sci.* 35:1288–1293.

Wilson, A. J., U. Gelin, M.-C. Perron, and D. Réale. 2009. Indirect genetic effects and the evolution of aggression in a vertebrate system. *Proc. R. Soc. Lond. B* 276:533–541.

Wolf, J. B. 2003. Genetic architecture and evolutionary constraint when the environment contains genes. *Proc. Natl. Acad. Sci. USA* 100:4655–4660.

Wolf, J. B., E. D. Brodie III, J. M. Cheverud, A. J. Moore, and M. J. Wade. 1998. Evolutionary consequences of indirect genetic effects. *Trends Ecol. Evol.* 13:64–69.

Wolf, J. B., E. D. Brodie III, and A. J. Moore. 1999. Interacting phenotypes and the evolutionary process. II. Selection resulting from social interactions. *Am. Nat.* 153:254–266.

Associate Editor: C. Goodnight

## Appendix

### PROOFS

To prove equation (14), we substitute equations (13a) and (13c) for  $G_{SD}$  and  $G_D$ .

$$\Psi = (\mathbf{I} - \Psi\Psi)^{-1}\Psi\mathbf{G}(\mathbf{I} - \Psi^T\Psi^T)^{-1} \cdot [(\mathbf{I} - \Psi\Psi)^{-1}\mathbf{G}(\mathbf{I} - \Psi^T\Psi^T)^{-1}]^{-1}$$

$$\Psi = (\mathbf{I} - \Psi\Psi)^{-1}\Psi\mathbf{G}(\mathbf{I} - \Psi^T\Psi^T)^{-1}(\mathbf{I} - \Psi^T\Psi^T)\mathbf{G}^{-1}(\mathbf{I} - \Psi\Psi)$$

$$\Psi = (\mathbf{I} - \Psi\Psi)^{-1}\Psi(\mathbf{I} - \Psi\Psi)$$

$$\Psi = (\mathbf{I} - \Psi\Psi)^{-1}(\Psi - \Psi\Psi\Psi)$$

$$\Psi = (\mathbf{I} - \Psi\Psi)^{-1}(\mathbf{I} - \Psi\Psi)\Psi$$

$$\Psi = \Psi.$$

Similarly, to prove equation (26), we substitute equations (25a) and (25c) for  $G_{SD}$  and  $G_D$ .

$$\Psi = \mathbf{U}^{-1}\Psi\mathbf{V}^{-1}\mathbf{G}(\mathbf{U}^T)^{-1}[\mathbf{U}^{-1}\mathbf{G}(\mathbf{U}^T)^{-1} + (n - 2)\mathbf{U}^{-1}\Psi\mathbf{V}^{-1}\mathbf{G}(\mathbf{U}^T)^{-1}]^{-1}$$

$$\Psi = \mathbf{U}^{-1}\Psi\mathbf{V}^{-1}[\mathbf{U}^{-1} + (n - 2)\mathbf{U}^{-1}\Psi\mathbf{V}^{-1}]^{-1}$$

$$\Psi = \mathbf{U}^{-1}\Psi\mathbf{V}^{-1}[\mathbf{I} + (n - 2)\Psi\mathbf{V}^{-1}]^{-1}\mathbf{U}$$

$$\Psi = \mathbf{U}^{-1}\Psi[\mathbf{V} + (n - 2)\Psi]^{-1}\mathbf{U}$$

$$\Psi = \mathbf{U}^{-1}\Psi[\mathbf{I} - (n - 2)\Psi + (n - 2)\Psi]^{-1}\mathbf{U}$$

$$\Psi = \mathbf{U}^{-1}\Psi\mathbf{U}$$

$$\Psi = \mathbf{U}^{-1}\Psi[\mathbf{I} - (n - 1)\Psi[\mathbf{I} - (n - 2)\Psi]^{-1}\Psi]$$

$$\Psi = \mathbf{U}^{-1}[\Psi - (n - 1)\Psi\Psi[\mathbf{I} - (n - 2)\Psi]^{-1}\Psi]$$

$$\Psi = \mathbf{U}^{-1}[\mathbf{I} - (n - 1)\Psi\Psi[\mathbf{I} - (n - 2)\Psi]^{-1}]\Psi$$

$$\Psi = \mathbf{U}^{-1}[\mathbf{I} - (n - 1)\Psi[\Psi^{-1} - (n - 2)\mathbf{I}]^{-1}]\Psi$$

$$\Psi = \mathbf{U}^{-1}\mathbf{U}\Psi$$

$$\Psi = \Psi.$$