

Multilevel Selection 4: Modeling the Relationship of Indirect Genetic Effects and Group Size

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ABSTRACT

Indirect genetic effects (IGE) occur when individual trait values depend on genes in others. With IGEs, heritable variance and response to selection depend on the relationship of IGEs and group size. Here I propose a model for this relationship, which can be implemented in standard restricted maximum likelihood software.

SOcial interactions among individuals are abundant in life (FRANK 2007). Trait values of individuals may, therefore, depend on genes in other individuals, a phenomenon known as indirect genetic effects (IGE; WOLF *et al.* 1998) or associative effects (GRIFFING 1967; MUIR 2005). IGEs may have drastic effects on the rate and direction of response to selection. Moreover, with IGEs, heritable variance and response to selection depend on the size of the interaction group, hereafter denoted group size (GRIFFING 1967; BIJMA *et al.* 2007; MCGLOTHLIN *et al.* 2010). The magnitude of the IGEs themselves, however, may also depend on group size, because interactions between a specific pair of individuals are probably less intense in larger groups (ARANGO *et al.* 2005). The relationship between the magnitude of IGEs and group size is relevant because it affects the dynamics of response to selection, heritable variation, and group size, determining, *e.g.*, whether or not selection is more effective with larger groups. Moreover, a model for this relationship is required to estimate IGEs from data containing varying group sizes. HADFIELD and WILSON (2007) proposed a model for the relationship between IGEs and group size. Here I present an alternative.

With IGEs, the trait value of focal individual i is the sum of a direct effect rooted in the focal individual itself, $P_{D,i}$, and the sum of the indirect effects, $P_{S,j}$, of each of its $n - 1$ group mates j ,

$$z_i = P_{D,i} + \sum_{j=1}^{n-1} P_{S,j} = (A_{D,i} + E_{D,i}) + \left(\sum_{j=1}^{n-1} A_{S,j} + \sum_{j=1}^{n-1} E_{S,j} \right), \quad (1)$$

where A and E represent the heritable and nonheritable component of the full direct and indirect effect, respectively, and n denotes group size (GRIFFING 1967). When IGEs are independent of group size, total heritable variance in the trait equals (BIJMA *et al.* 2007)

$$\sigma_{TBV}^2 = \sigma_{A_D}^2 + 2(n-1)\sigma_{A_{DS}} + (n-1)^2\sigma_{A_S}^2. \quad (2)$$

For a fixed $\sigma_{A_S}^2$, σ_{TBV}^2 becomes very large with large groups. This is unrealistic because an individual's IGE on a single recipient probably becomes smaller in larger groups. The decrease of IGEs with group size, referred to as dilution here, will depend on the trait of interest. With competition for a finite amount of feed per group, for example, an individual consuming 1 kg has an average indirect effect of $P_{S,i} = -1/(n-1)$ on feed intake of each of its group mates. Hence, the indirect effect is inversely proportional to the number of group mates, indicating full dilution. The other extreme of no dilution may be illustrated by alarm-calling behavior, where an individual may warn all its group mates when a predator appears, irrespective of group size. Here the indirect effect each group mate receives is independent of group size, indicating no dilution. The degree of dilution is an empirical issue, which may be trait and population specific, and needs to be estimated.

Here I propose to model dilution of indirect effects as

$$P_{S,i,n} = \frac{1}{(n-1)^d} P_{S,i,2}, \quad (3)$$

where $P_{S,i,n}$ is the indirect effect of individual i in a group of n members, $P_{S,i,2}$ the indirect effect of i in a group of two members, and d the degree of dilution. With no dilution, $d = 0$, indirect effects do not depend on group size, $P_{S,i,n} = P_{S,i,2}$, as with alarm-calling

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behavior. With full dilution, $d = 1$, indirect effects are inversely proportional to the number of group mates, $P_{S,i,n} = P_{S,i,2}/(n - 1)$, as with competition for a finite amount of feed. Equation 3 is an extension of the model of ARANGO *et al.* (2005), who used $d = 1$.

Assuming that IGEs are diluted in the same manner as the full indirect effect, the indirect genetic variance for groups of n members equals

$$\sigma_{A_{S,n}}^2 = \frac{1}{(n - 1)^{2d}} \sigma_{A_{S,2}}^2, \quad (4)$$

and total heritable variance equals

$$\sigma_{TBV,n}^2 = \sigma_{A_D}^2 + 2(n - 1)^{1-d} \sigma_{A_{DS,2}}^2 + (n - 1)^{2-2d} \sigma_{A_{S,2}}^2. \quad (5)$$

Hence, for $\sigma_{A_{DS}} = 0$, total heritable variance increases with group size as long as dilution is incomplete ($d < 1$). Total heritable variance is independent of group size with full dilution ($d = 1$). Phenotypic variance also depends on group size. With unrelated group members,

$$\sigma_z^2 = \sigma_{P_D}^2 + (n - 1)^{1-2d} \sigma_{P_{S,2}}^2, \quad (6)$$

which increases with group size for $d < 0.5$, is independent of group size for $d = 0.5$, and decreases with group size for $d > 0.5$.

The degree of dilution can be estimated from data containing variation in group size, by using a mixed model with restricted maximum likelihood and evaluating the likelihood for different fixed values of d (ARANGO *et al.* 2005; CANARIO *et al.* 2010). With Equation 3, however, the estimated genetic (co)variances and breeding values for indirect effect refer to a group size of two individuals, which is inconvenient when actual group size differs considerably. Estimates of A_S , $\sigma_{A_S}^2$, and $\sigma_{A_{DS}}$ referring to the average group size may be obtained from the following mixed model,

$$\mathbf{z} = \mathbf{X}\mathbf{b} + \mathbf{Z}_D\mathbf{a}_D + \mathbf{Z}_{S(d)}\mathbf{a}_{S,\bar{n}} + \mathbf{Z}_g\mathbf{g} + \mathbf{e}, \quad (7)$$

where $\mathbf{z}$ is a vector of observations, $\mathbf{X}\mathbf{b}$ are the usual fixed effects, $\mathbf{Z}_D\mathbf{a}_D$ are the direct genetic effects, $\mathbf{Z}_g\mathbf{g}$ are random group effects, and $\mathbf{e}$ is a vector of residuals. The $\mathbf{a}_{S,\bar{n}}$ is a vector of IGEs referring to the average group size, and $\mathbf{Z}_{S(d)}$ is the incidence matrix for IGEs, which depends on the degree of dilution; dilution being specified relative to the average group size. Elements of $\mathbf{Z}_{S(d)}$ are

$$\begin{aligned} \mathbf{Z}_{S(d)}(i, j) &= \left(\frac{\bar{n} - 1}{n - 1}\right)^d \quad \text{when } j \text{ is a group mate of } i, \\ \mathbf{Z}_{S(d)}(i, j) &= 0 \quad \text{otherwise,} \end{aligned} \quad (8)$$

where $\bar{n}$ denotes average group size. This model is equivalent to Equation 3, but yields estimates of genetic parameters and breeding values referring to the average

group size because $[(\bar{n} - 1)/(n - 1)]^d = 1$ for $n = \bar{n}$. When the magnitude of IGEs depends on group size, the group and residual variance in Equation 7 will depend on group size:

$$\sigma_{g,n}^2 = 2\left(\frac{\bar{n} - 1}{n - 1}\right)^d \sigma_{E_{DS,\bar{n}}}^2 + (n - 2)\left(\frac{\bar{n} - 1}{n - 1}\right)^{2d} \sigma_{E_{S,\bar{n}}}^2 \quad (9a)$$

$$\sigma_{e,n}^2 = \sigma_{E_D}^2 - 2\left(\frac{\bar{n} - 1}{n - 1}\right)^d \sigma_{E_{DS,\bar{n}}}^2 + \left(\frac{\bar{n} - 1}{n - 1}\right)^{2d} \sigma_{E_{S,\bar{n}}}^2. \quad (9b)$$

Hence, to obtain unbiased estimates of the genetic parameters and d , it may be required to fit a separate group and residual variance for each group size.

To account for the relationship between IGEs and group size, HADFIELD and WILSON (2007; HW07) proposed including an additional IGE. In their model, an individual's full IGE is the sum of an effect independent of group size, and an effect regressed by the reciprocal of the number of group mates,

$$A_{S,i,HW07} = A_{S,i} + \frac{1}{n - 1} A_{S_R,i}. \quad (10)$$

There are a number of differences between both models. First, Equation 3 specifies the relationship between the magnitude of IGEs and group size on the population level, which is sufficient to remedy the problem of increasing variance with group size. The HW07 model, in contrast, specifies the relationship between the magnitude of IGEs and group size on the individual level. In the HW07 model, the absolute value of $(1/(n - 1))A_{S_R,i}$ decreases with group size, while $A_{S,i}$ is constant. Consequently, the relationship between an individual's full IGE and group size depends on the relative magnitudes of its $A_{S,i}$ and $A_{S_R,i}$; the IGEs of individuals with greater $|A_{S_R,i}|$ show greater change when group size varies. This alters the IGE ranking of individuals when group size varies. The HW07 model, therefore, not only scales IGEs with group size, but also allows for IGE-by-group-size interaction, whereas Equation 3 scales IGEs of all individuals in the same way. Second, the interpretation of the genetic parameters differs between both models. In the HW07 model, $\lim_{n \rightarrow \infty} A_{S,i,HW07} = A_{S,i}$ meaning that $\text{Var}(A_S)$ represents the variance in IGEs when group size is infinite. With Equation 3 or 7, in contrast, $\sigma_{A_S}^2$ refers to groups of two individuals or to the average group size. Third, in the HW07 model, the dilution of IGEs with group size is implicitly incorporated in the magnitudes of $\text{Var}(A_S)$ and $\text{Var}(A_{S_R})$, greater $\text{Var}(A_{S_R})$ implying greater dilution. Equation 3, in contrast, has a single parameter for the degree of dilution, expressed on a 0–1 scale. Finally, implementing the HW07 model involves estimating three additional covariance parameters, $\text{Var}(A_{S_R})$, $\text{Cov}(A_D, A_{S_R})$, and $\text{Cov}(A_S, A_{S_R})$, whereas implementing the model proposed here involves estimating a single

additional fixed effect, which is simpler. In conclusion, the HW07 model has greater flexibility than the model proposed here, but is also more difficult to implement and interpret.

Note added in proof: See P. BIJMA (pp. 1013–1028) in this issue, for a related work.

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