Lecture 10:
Associate effects models, kin/group selection, inclusive fitness

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Introduction to Mixed Models
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More details in WL (2018) Chapter 22,
On Course website

Associative effects models

- A very powerful recent development in quantitative genetics (although the idea dates back to Griffin's work in the 1960s) is the notion of direct vs. associative (or social, or indirect genetic) effects
- This idea unifies kin and group selection, offers models for the evolution of social (group-level) traits, and shows why selection can often fail
- The basic idea is that the phenotype of a target individual is a function of some intrinsic direct value and also the phenotypes of those individuals with which it interacts.

Direct & Associative effects

- Consider egg production from chickens raised in cages. Production is a function of both a chicken's own genetics and the environment (her other cage-mates)
  - Direct effects = intrinsic egg production
  - Associative effects = competitive ability
- Suppose our focal individual (i) interacts with n-1 others in a group

$$z_i = P_{d,i} + \sum_{j \neq i}^{n} P_{j,s}$$

Direct and associative effects can be antagonistic

- Consider a plant with a trait that allows it to more efficiently garner resources
- This gives it a high direct effect but a negative associative effect --- it reduces the trait values in those individuals with which it interacts
- Thus, the best performing single plants can have very low average plot performance

Example 20.1. This point was made in a classic paper by Weibe et al (1976), who examined yield in mixed- versus single-genotype plots of barley. They observed that genotypes which yielded well in mixed stands had poorer yield in pure stands, while those genotypes that did poorly in mixed stands had the highest yield in pure stands. In our framework, we could imagine that lines which do well in mixed stands have both high direct effects and high negative associative effects, suppressing the phenotypes of their neighbors. When grown in a pure strand, the high negative associative effects suppress plot yield. Conversely, lines that perform poorly in mixed strands might have low direct effects but high positive associative effects, so that the phenotypes of their neighbors are enhanced (or atleast not hindered). When grown as a pure strand, these high positive associative effects more than compensate for the low direct effects, increasing yield.

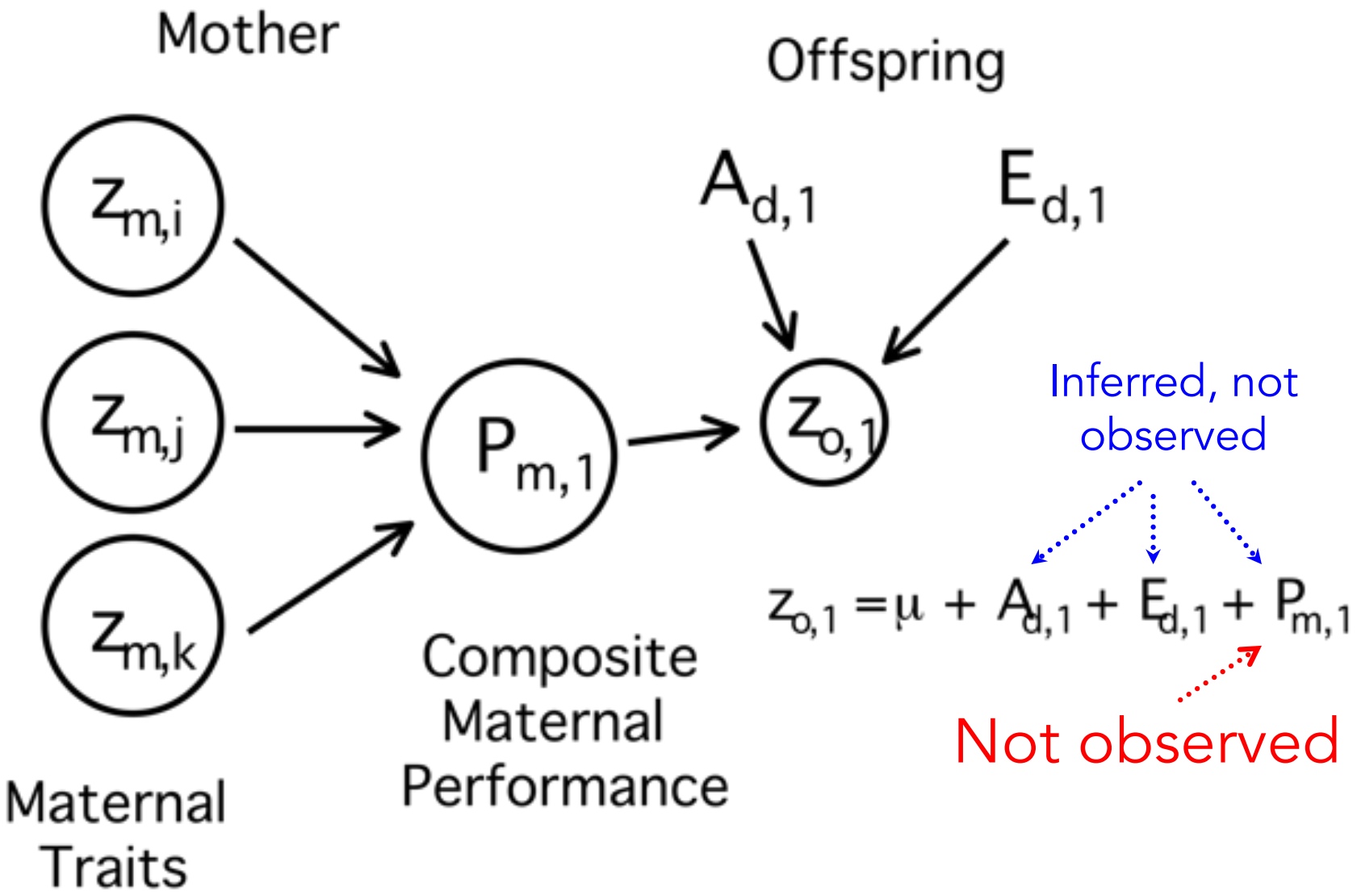
Roots of associative-effects models trace to maternal effects

- Maternal effects are a classic example of associative effects (maternal performance).
- Two different approaches to model maternal effects
  - Falconer model: an observed trait value (e.g., litter size) influences offspring. Trait-based
  - Willham model: Maternal performance is a latent (unobserved) variable, and hence we don't need to specify it. Variance-component based. We focus on these models here.

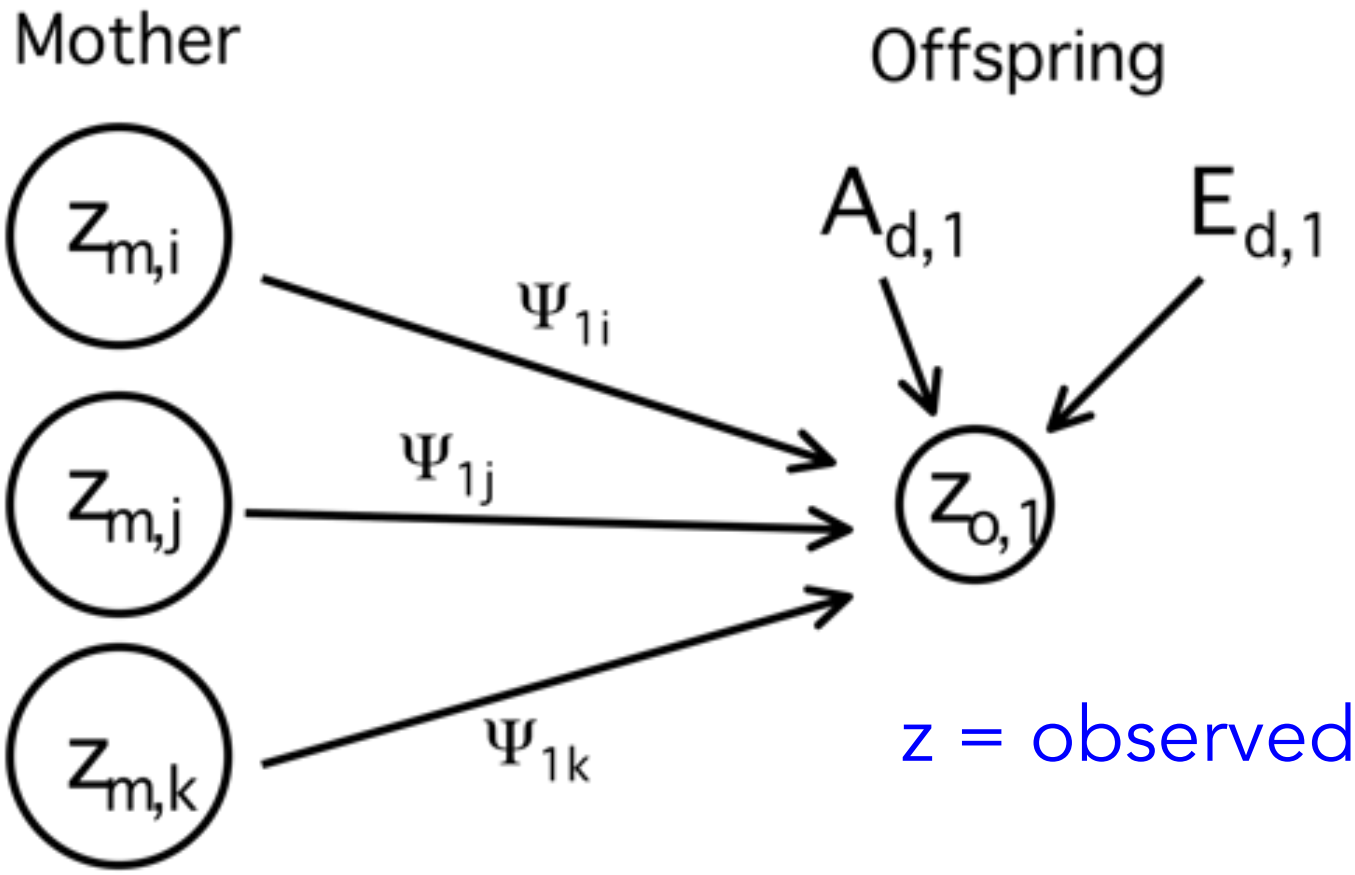
Trait-based vs. variance-component models

- Trait based:
  - Trait values of associative effects in group members are observed
- Variance-component models
  - A composite latent (unmeasured) variable for associative effects is created

Variance components



Trait-based models



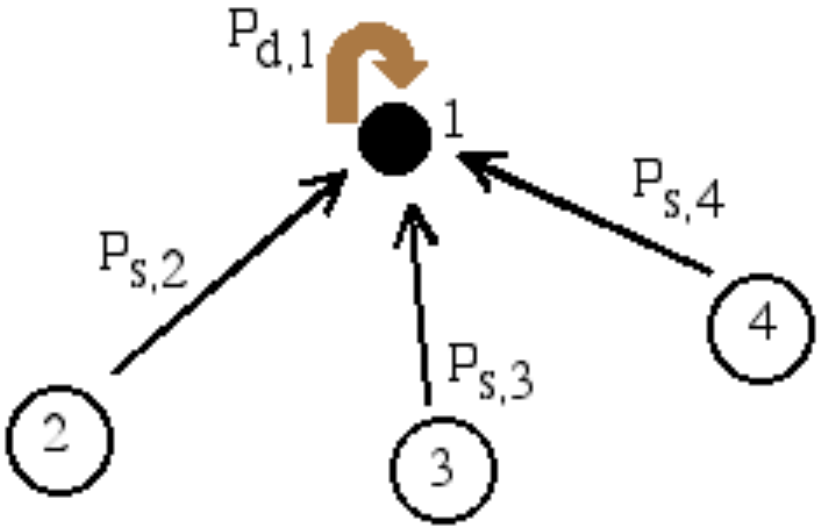
$$z_{o,1} = \mu + A_{d,1} + E_{d,1} + \Psi_{1,i} z_{m,i} + \Psi_{1,j} z_{m,j} + \Psi_{1,k} z_{m,k}$$

Decomposition

- Consider the phenotype of a focal individual
- Sum of a direct effect and an associative effect
- Both of these can have a breeding value and an environment (residual) deviation
- The breeding values of the direct & associative effects can be correlated
- This is a multiple-traits problem

$$z_i = P_{d,i} + \sum_{j \neq i}^{n} P_{j,s}$$

- i's phenotype z_i is the sum of its direct effect ($P_{d,i}$) plus the sum of the associative (or social) effects ($P_{s,j}$) from its n-1 group members



$$z_1 = P_{d,1} + P_{s,2} + P_{s,3} + P_{s,4}$$

Breeding values for direct (A_d) and associative (A_s) effects

- Can express the phenotype of i in terms of its direct breeding value ($A_{d,i}$) and the associative breeding values ($A_{s,j}$) of its group mates

$$z_i = \mu + (A_{d_i} + E_{d_i}) + \sum_{j \neq i} (A_{s_j} + E_{s_j})$$

$$z_i = \mu + A_{d_i} + \sum_{j \neq i} A_{s_j} + e_i, \qquad e_i = E_{d_i} + \sum_{j \neq i} E_{s_j}$$

Total response

The trait mean equals the mean of the direct effects plus the means of the associative effects,

$$\mu_z = \mu_{A_d} + (n-1)\mu_{A_s}$$

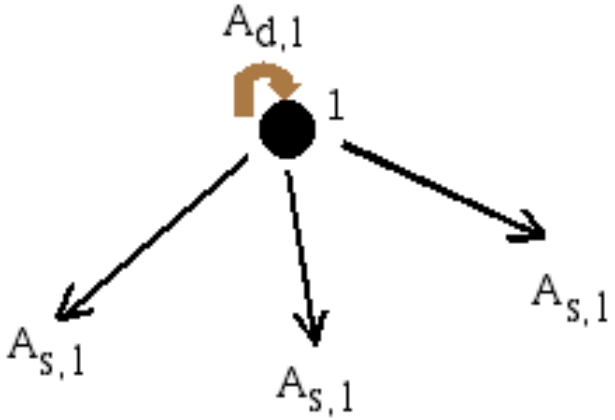
Total response is the sum of the response R_d in the direct breeding values plus the sum of the responses R_s in the associative effects breeding values,

$$R_z = R_d + (n-1)R_s$$

Total breeding value

The key to predicting response is the total breeding value of an individual, where

$$A_{T,i} = A_{d,i} + (n-1)A_{s,i}$$



Note that part ($A_{s,i}$) of the total breeding value of i never appears in its phenotype. Must either use informative from relatives or the group to estimate it.

h^2 and τ^2

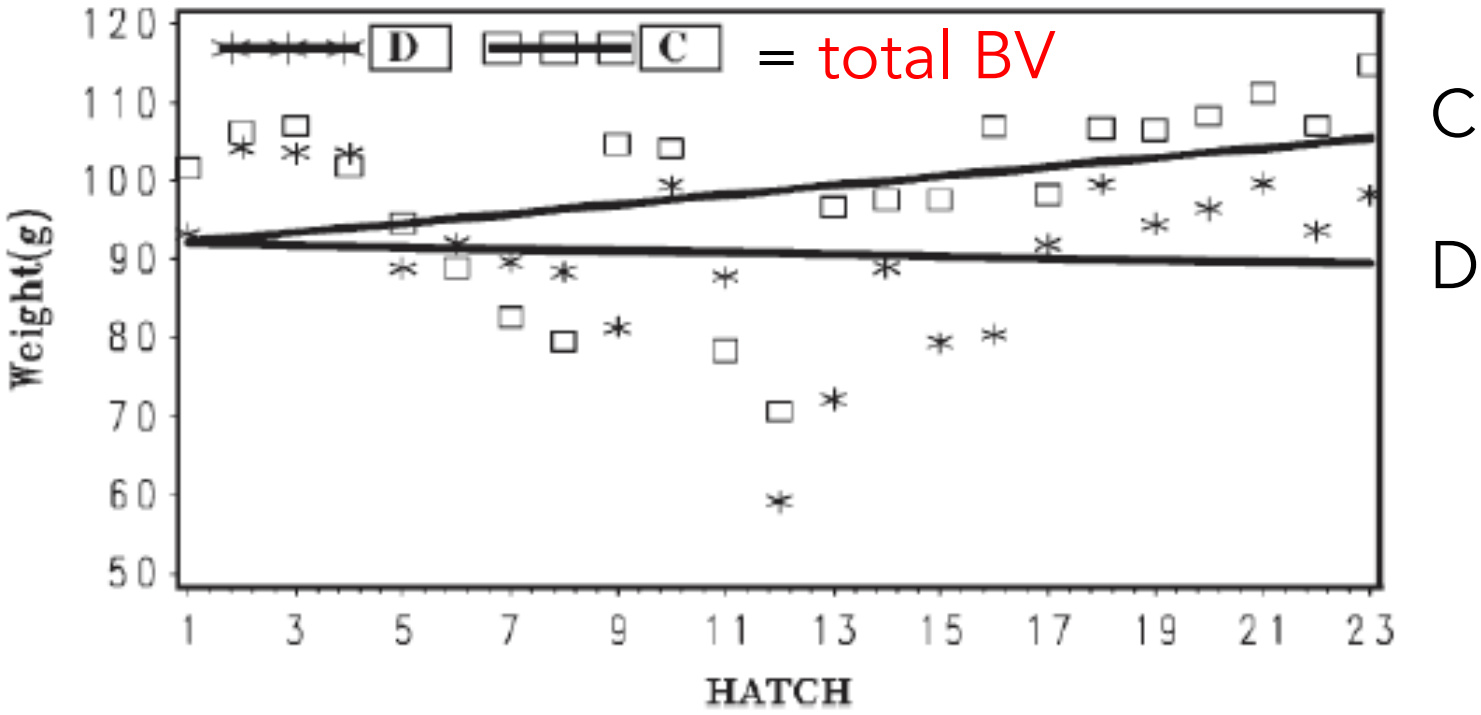
- τ^2, the analog for h^2, is the ratio of the total breeding value to the individual phenotypic variance
  - $\tau^2 = Var(A_T)/Var(z)$
- Note that, unlike h^2, τ^2 can exceed one,
- Why? A potentially large fraction of A_T never appears in z, and hence Var(z)
  - $Var(A_T) = Var(A_d) + (n-1)Var(A_s)$
  - $\tau^2 = Var(A_d)/Var(z) + (n-1)Var(A_s)/Var(z)$
  - $= h^2 + (n-1)Var(A_s)/Var(z)$

BLUP estimation

- While the total breeding value cannot be estimated directly from an individual's phenotype, using an appropriate mixed model, we can obtain
  - BLUPs of Direct breeding values (A_d)
  - BLUPs of Associative (or social) BVs (A_s)
  - REML estimates of $\sigma^2(A_d)$, $\sigma^2(A_s)$, and the direct-associate effects covariance $\sigma(A_d, A_s)$

This works: Muir's result

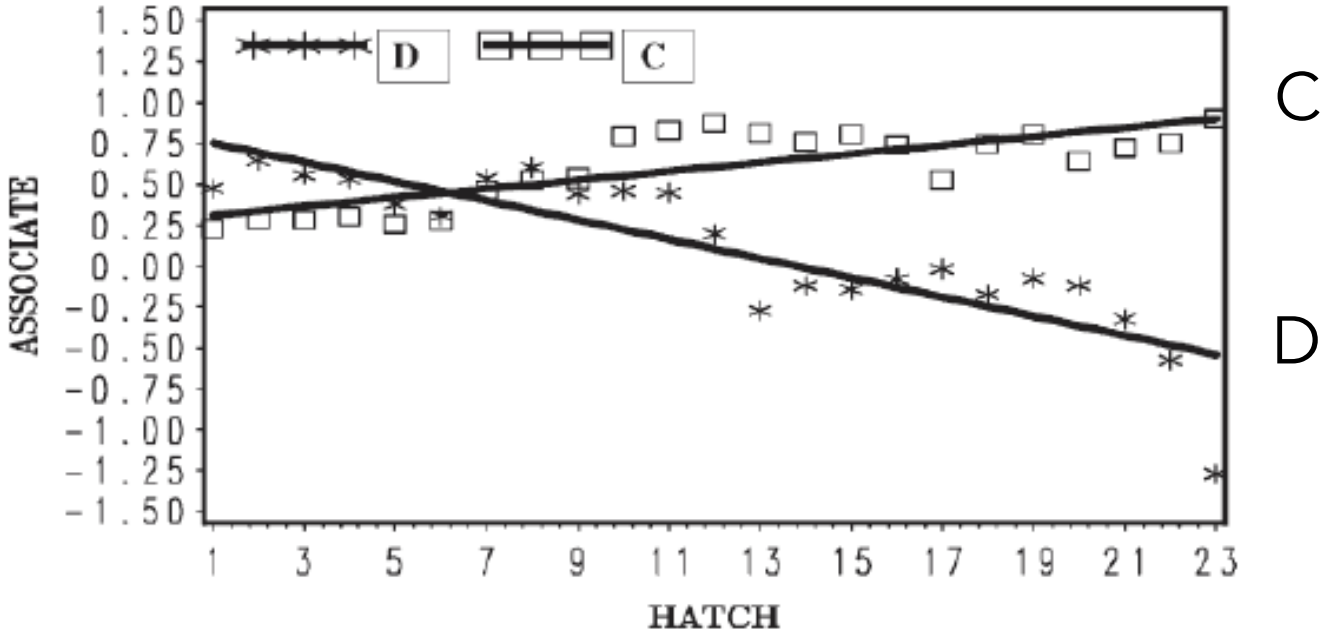
- Bill Muir (Purdue University) selection on six-week weight in Japanese quail over 23 generations using two different schemes
  - BLUP selection on estimated direct BV (D)
    - Denoted by D-BLUP
  - BLUP selection on estimated total BV
    - Denoted by C-BLUP



C

D

Weighted increased under selection using total BV (C), decreased under selection using direct BV (D).



C

D

Under BLUP selection on direct BV (D), significant decline in the mean social value, which over-rode the positive response in the direct value

Under BLUP selection of total BV (C), *both* increase

The mixed model

$$\mathbf{z} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}_d\,\mathbf{a}_d + \mathbf{Z}_s\,\mathbf{a}_s + \mathbf{e}$$

Example: Individuals 1-4 and 5-8 are half sibs from unrelated families

$$\mathbf{A} = \begin{pmatrix} 1 & 0.25 & 0.25 & 0.25 & 0 & 0 & 0 & 0 \\ 0.25 & 1 & 0.25 & 0.25 & 0 & 0 & 0 & 0 \\ 0.25 & 0.25 & 1 & 0.25 & 0 & 0 & 0 & 0 \\ 0.25 & 0.25 & 0.25 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0.25 & 0.25 & 0.25 \\ 0 & 0 & 0 & 0 & 0.25 & 1 & 0.25 & 0.25 \\ 0 & 0 & 0 & 0 & 0.25 & 0.25 & 1 & 0.25 \\ 0 & 0 & 0 & 0 & 0.25 & 0.25 & 0.25 & 1 \end{pmatrix}$$

Filling out Z_s

- Suppose group one contains individuals 1, 2, 5, 6. The resulting values for these individuals become
  - $z_1 = m + A_{d1} + A_{s2} + A_{s5} + A_{s6} + e$
  - $z_2 = m + A_{d2} + A_{s1} + A_{s5} + A_{s6} + e$
  - $z_5 = m + A_{d5} + A_{s1} + A_{s2} + A_{s6} + e$
  - $z_6 = m + A_{d6} + A_{s1} + A_{s2} + A_{s5} + e$
  - The result $\mathbf{Z_d}$ and $\mathbf{Z_s}$ incident matrices become

$$\mathbf{z} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}_d\,\mathbf{a}_d + \mathbf{Z}_s\,\mathbf{a}_s + \mathbf{e}$$

$$\mathbf{z} = \begin{pmatrix} z_1 \\ z_2 \\ z_3 \\ z_4 \\ z_5 \\ z_6 \\ z_7 \\ z_8 \end{pmatrix},\ \mathbf{X} = \begin{pmatrix} 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \end{pmatrix},\ \mathbf{a}_d = \begin{pmatrix} A_{d,1} \\ A_{d,2} \\ A_{d,3} \\ A_{d,4} \\ A_{d,5} \\ A_{d,6} \\ A_{d,7} \\ A_{d,8} \end{pmatrix},\ \mathbf{Z}_d = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix} = \mathbf{I}_8$$

Group one contains individuals 1,2,5,6; while group two contains 3,4,7,8.

$$\mathbf{Z}_s = \begin{pmatrix} 0 & 1 & 0 & 0 & 1 & 1 & 0 & 0 \\ 1 & 0 & 0 & 0 & 1 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 1 & 1 \\ 0 & 0 & 1 & 0 & 0 & 0 & 1 & 1 \\ 1 & 1 & 0 & 0 & 0 & 1 & 0 & 0 \\ 1 & 1 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 1 & 1 & 0 & 0 & 0 & 1 \\ 0 & 0 & 1 & 1 & 0 & 0 & 1 & 0 \end{pmatrix},\qquad \mathbf{a}_s = \begin{pmatrix} A_{s,1} \\ A_{s,2} \\ A_{s,3} \\ A_{s,4} \\ A_{s,5} \\ A_{s,6} \\ A_{s,7} \\ A_{s,8} \end{pmatrix}$$

Lots of hidden variation to exploit

- Bergsma et al. (2008) examined four traits in 14,000 pigs grown in pens of 6-12 animals.
- Heritability for these traits was estimated in a model without social effects,

	Growth	Back fat	Muscle	Intake
$\sigma^2(A)$	2,583	2.83	7.94	41,275
h^2	0.37	0.36	0.25	0.41

Next, a model was fit allowing for heritable social effects, $\mathbf{z} = \mathbf{X}\beta + \mathbf{Z}_d\mathbf{a}_d + \mathbf{Z}_s\mathbf{a}_s + \mathbf{Z}_c\mathbf{c} + \mathbf{e}$, which gave estimates of

	Growth	Back fat	Muscle	Intake
$\sigma^2(A_d)$	1,522	2.75	6.68	16,950
h^2_d	0.21	0.35	0.21	0.17
$\sigma^2(A_s)$	51	0.01	0.03	596
$\sigma^2(A_T)$	5,208	3.19	10.35	68,687
τ^2	0.71	0.41	0.32	0.70

Here $h^2_d = \sigma^2(A_d)/\sigma^2(z)$, while $\tau^2 = \sigma^2(A_T)/\sigma^2(z)$. h^2_d measures the response potential under phenotypic selection, while $\tau^2 \geq h^2_d$ measures the total genetic potential for improvement under specialized selection designs.

	Growth	Back fat	Muscle	Intake
$\sigma^2(A)$	2,583	2.83	7.94	41,275
h^2	0.37	0.36	0.25	0.41

Hence, for growth and food intake, lots of additional genetic variation for trait response lies “hidden” in associative effects.

Consequences

- How can we exploit this variation in breeding?
- What are the consequences for evolutionary biologists?
- Need to consider selection response
  - Has both a direct and associative effects component

$$z_i = \mu + (A_{d_i} + E_{d_i}) + \sum_{j \neq i} (A_{s_j} + E_{s_j}) \tag{20.1b}$$

We can write this compactly as

$$z_i = \mu + A_{d_i} + \sum_{j \neq i} A_{s_j} + e_i, \quad \text{where} \quad e_i = E_{d_i} + \sum_{j \neq i} E_{s_j} \tag{20.1c}$$

Since the environmental values have expected value zero, the mean phenotypic value in the group is just

$$\mu_z = \mu_{A_d} + (n-1)\mu_{A_s} \tag{20.1d}$$

Further, the change in the mean trait value within a group following selection is

$$\Delta\mu_z = \Delta\mu_{A_d} + (n-1)\Delta\mu_{A_s} \tag{20.1e}$$

total response

Direct response

Associative response

Example 20.2. Consider a trait in a group of four (unrelated) individuals, where (for illustrative purposes) we assume no environmental values so that $P_d = A_d$ and $P_s = A_s$. The population mean is 20, and the four group members have the following breeding values for direct, associative, and total effects:

Individual	A_d	A_s	A_T	$\sum_{j \neq i} A_{s_j}$	z
1	9	-4	-3	4	33
2	5	-1	2	1	26
3	-6	2	0	-2	12
4	-8	3	1	-3	9

Since $n = 4$, $A_T = A_d + 3\,A_s$. The sum $\sum_{j \neq i} A_{s_j}$ represents the contribution of the associative effects of the other three individuals to i's value. For example, for individual 1, the contributions from individuals 2 through 4 is -1 + 2 + 3 = 4. From Equation 20.1c, the phenotypic value we would observe is

$$z_i = 20 + A_{d_i} + \sum_{j \neq i} A_{s_j}$$

Individual one has the largest direct effect (9) and the largest observed trait value (33). This individual also has the most unfavorable associative value (-4), and the smallest total breeding value (-3). Conversely, it has the largest contribution (4) to its trait value from the associative effects of the other group members. Its high trait value is due to this combination of a high direct effect and a high contribution from the associative effects of the other group members. Its unfavorable associative effects do not appear in its own phenotype, but rather are expressed in the trait values of the other group members. As a result, its own phenotypic value is a poor predictor of A_T.

Individual	A_d	A_s	A_T	$\sum_{j \neq i} A_{s_j}$	z
1	9	-4	-3	4	33
2	5	-1	2	1	26
3	-6	2	0	-2	12
4	-8	3	1	-3	9

If the next generation is formed by crossing the two individuals (1 and 2) with the largest trait values, the expected offspring mean is 20 + (-3+2)/2 = 19.5, the mean plus their average total breeding values. Although the two largest individuals were chosen, the population mean *decreases*. Conversely, crossing the two smallest individuals gives an expected offspring mean of 20 + (0+1)/2 = 20.5, increasing the mean. While the two smallest individuals have the smallest direct effects, they also have the most favorable associative effects, and hence give a more favorable response. The greatest expected response occurs by crossing the two individuals (2 and 4) with the largest total breeding values, for an expected mean of 20 + (2+1)/2 = 21.5.

Response: It's about covariances

- Selection response is a function of the covariance between our unit u of selection and the total breeding value, $\sigma(A_T, u)$
  - $R = i * \sigma(A_T, u) / \sigma(u)$ (generalized breeder's Eq.)
- The "unit" could be a
  - single individual (individual selection)
  - The group mean (group selection)
  - Some index of these
- Members of a group can be
  - Unrelated
  - Related (kin selection)
- All these considerations influence $\sigma(A_T, u)$

The covariance between an individual's phenotype and total breeding value is

$$\sigma(z_i, A_{T_i}) = \sigma\left(\mu + A_{d_i} + \sum_{j \neq i} A_{s_j} + e_i, A_{d_i} + (n-1)A_{s_i}\right)$$

General expression

$$= \sigma\left(A_{d_i}, A_{d_i} + (n-1)A_{s_i}\right) + \sum_{j \neq i} \sigma\left(A_{s_j}, A_{d_i} + (n-1)A_{s_i}\right) \qquad (20.4a)$$

For now, we assume unrelated group members, in which case the covariances in the summation are all zero, giving

Group members unrelated (r = 0)

$$\sigma(z, A_T) = \sigma^2(A_d) + (n-1)\sigma(A_d, A_s) \qquad (20.4b)$$

If the direct and associative effects are uncorrelated, this reduces to our standard result that the covariance between an individual's phenotype and breeding value is just the additive genetic variance (in this case, of direct effects). By contrast, the variance of the total breeding value becomes

$$\sigma^2(A_T) = \sigma^2[A_d + (n-1)A_s]$$

$$= \sigma^2(A_d) + 2(n-1)\sigma(A_d, A_s) + (n-1)^2\sigma^2(A_s) \qquad (20.4c)$$

$$= \sigma(z, A_T) + (n-1)\left[2\sigma(A_d, A_s) + (n-1)\sigma^2(A_s)\right] \qquad (20.4d)$$

Group members unrelated (r = 0)

Now consider the phenotypic variance,

$$\sigma_z^2 = \sigma^2\left(P_{d_i} + \sum_{j \neq i} P_{s_j}\right). \tag{20.5a}$$

Assuming (for now) that the group members are unrelated, so that $\sigma(P_{d_i}, P_{s_j}) = 0$. For a group of size n Equation 20.5a reduces to

$$\begin{aligned}
\sigma_z^2 &= \sigma^2(P_d) + (n-1)\sigma^2(P_s) && (20.5b)\\
&= \sigma^2(A_d) + (n-1)\sigma^2(A_s) + \sigma^2(E_d) + (n-1)\sigma^2(E_s) && (20.5c)\\
&= \sigma^2(A_d) + (n-1)\sigma^2(A_s) + \sigma^2(e) && (20.5d)
\end{aligned}$$

where e is given by Equation 20.1c. With the phenotypic variance in hand, we can define the heritability of the direct and associative effects as

$$h_d^2 = \frac{\sigma^2(A_d)}{\sigma_z^2}, \qquad \text{and} \qquad h_s^2 = \frac{\sigma^2(A_s)}{\sigma_z^2} \tag{20.6a}$$

Example 20.3. Consider a trait in a group of 10 unrelated individuals, with $\sigma^2_{P_d} = 10$, $\sigma^2_{P_s} = 1$, and both direct and associative effects have modest heritabilities measured on the scale of the effect themselves ($h'^2_d = 0.4, h'^2_s = 0.3$). To simplify matters, assume $\sigma(A_d, A_s) = 0$. Applying Equation 20.5b, the resulting phenotypic variance is

$$\sigma^2_z = \sigma^2_{P_d} + 9 \cdot \sigma^2_{P_s} = 10 + 9 \cdot 1 = 19$$

From Equation 20.4c, the variance in total breeding value becomes

$$\sigma^2_{A_T} = \sigma^2_{A_d} + 9^2 \cdot \sigma^2_{A_s} = h'^2_d \, \sigma^2_{P_d} + 9^2 \cdot h'^2_s \, \sigma^2_{P_s} = 4 + 81 \cdot 0.3 = 28.3,$$

giving T^2 = 28.3 / 18 = 1.57.

A real world example of large potential differences in h^2_d versus T^2 is survival days in chickens (Bijma et al. 2007b). Ignoring associative effects gives a heritability h^2_d of 6.7%, while when using a mixed model that incorporates associative effects (detailed later in the chapter), the estimate of T^2 was 20%, a threefold increase. Hence, under the conditions in the study, roughly two-thirds of the heritable variation in the trait arises from interactions between individuals and is thus hidden from standard analyses which ignore these. As discussed below, this component is only fully accessible under individual selection if the group includes relatives.

One of the key results when associative effects are present is that individual selection can result in a reversed response, while group selection always results in a positive response (although it may be far from optimal). These points were clearly made by Griffing (1967) for the simple case of two interacting, and unrelated, individuals within each group. For selection on individual phenotype, the response becomes

$$R = \frac{\overline{i}}{\sigma_z}\left[\sigma^2(A_d) + \sigma(A_d, A_s)\right] \tag{20.11a}$$

A negative covariance between direct and associative effects reduces the efficiency of selection, and if sufficiently negative, gives a reversed response. This loss of efficiency occurs because the only information an individual's phenotype contains about their breeding value for associative effects is that provided by the covariance between direct and associative breeding values (which can be negative). Conversely, if we select based on the mean of a group, we are selecting on both direct and associative effects to improve trait value. For the case of $n = 2$, Griffing obtained the expected response as

$$R = \frac{\overline{i}}{2\sigma(\overline{z})}\left[\sigma^2(A_d) + 2\sigma(A_d, A_s) + \sigma^2(A_s)\right] = \frac{\overline{i}}{2\sigma(\overline{z})}\,\sigma^2(A_T) \tag{20.11b}$$

While group selection always give a non-negative response, if the associative effects are weak, this approach is very inefficient relative to individual selection. For example, in the absence of associative effects, $\sigma^2(\overline{z}) = \sigma^2(z)/2$, and Equation 20.11b reduces to $\overline{i}\,\sigma(A_d)/[\sqrt{2}\,\sigma(z)]$, or $1/\sqrt{2} = 0.701$ of the response under individual selection.

Covariances with related group members

$$\sigma(z, A_T) = r\sigma^2(A_T) + (1-r)\left[\sigma^2(A_d) + (n-1)\sigma(A_d, A_s)\right]$$

$$\begin{aligned}\sigma^2(z) &= \sigma^2(A_d) + \sigma^2(E_d) + (n-1)\left[\sigma^2(A_s) + \sigma^2(E_s)\right] \\ &\quad + (n-1)r\left[2\sigma(A_s, A_d) + (n-2)\sigma^2(A_d)\right] \\ &= \sigma^2(z \mid r = 0) + (n-1)r\left[2\sigma(A_s, A_d) + (n-2)\sigma^2(A_d)\right]\end{aligned}$$

Group members related (r > 0)

The response to selection is simply the change in the mean total breeding value, which (from Chapter 10) is the within-generation change in the phenotypic mean after selection (the selection differential S) times the slope of the regression of A_T on phenotype z,

$$R = \frac{\sigma(z, A_T)}{\sigma_z^2} S = \frac{\sigma(z, A_T)}{\sigma_z} \bar{\imath} \qquad (20.14)$$

with the second formulation following from the standard identity that $S = \sigma_z \bar{\imath}$ (Equation 10.6a). For $n = 2$ and $r = 0$, we recover Griffin's result (Equation 20.11a).

Example 20.4. Muir (2005) estimated variance components for six-week body weight in Japanese quail (*Cotumix cotumix japonica*) housed in groups of $n = 16$ per cage. REML estimates of the genetic variances were $\sigma^2(A_d) = 33.7$ and $\sigma^2(A_s) = 2.87$, while $\sigma(A_d, A_s) = -5.5$. Under these values, the predicted response to individual selection in a group of 16 unrelated individuals is

$$R = \frac{\bar{i}}{\sigma_z}\left[\sigma^2(A_d) + (n-1)\sigma(A_d, A_s)\right] = \frac{\bar{i}}{\sigma_z}\left[33.7 + 15 \cdot (-5.5)\right] = -48.8\,\frac{\bar{i}}{\sigma_z}$$

The strong negative covariance between direct and social (competitive) effects results in an expected reversed response if directional selection is used, as the positive gain from improvement of direct effects is swamped by the negative effects from the correlated response in social values.

The presence of relatives within the group results in some fraction of $\sigma^2(A_s)$ being incorporated into the response under individual selection. Suppose the group of 16 consists of two half-sib families. In this case, the average relationship is 0.125, and from Equation 20.12d the resulting covariance between phenotype and total breeding values becomes

$$\begin{aligned}\sigma(z, A_T) &= \sigma(z, A_T \mid r = 0) + (n-1)r\left[\sigma(A_s, A_d) + (n-1)\sigma^2(A_s)\right] \\ &= -48.4 + 15 \cdot 0.125\,(-5.5 + 15 \cdot 2.87) = 21.6\end{aligned}$$

Simply by using groups of relatives (as opposed to groups of unrelated individuals) allows individual selection to give an expected positive response.

Individual selection: Direct vs. Associate response

Here unit of selection u = z, the phenotype of an individual

$$R_z = R_d + (n-1)R_s, \quad \text{where} \quad R_d = \frac{\sigma(A_d, z)}{\sigma_z}\bar{\imath} \quad \text{and} \quad R_s = \frac{\sigma(A_s, z)}{\sigma_z}\bar{\imath} \qquad (20.15a)$$

Here

$$\sigma(A_d, z) = \sigma\left(A_d, A_d + \sum_{i \neq j} A_{s,i} + e\right) = \sigma^2(A_d) + r(n-1)\sigma(A_d, A_s) \qquad (20.15b)$$

while

$$\sigma(A_s, z) = \sigma\left(A_s, A_d + \sum_{i \neq j} A_{s,i} + e\right) = \sigma(A_d, A_s) + r(n-1)\sigma^2(A_s) \qquad (20.15c)$$

Unless (i) A_s, A_d correlated OR (ii) group members are relatives, value of z provides information on A_d, but NOT on its A_s value

Example 20.6. Consider the response in a family of half-sibs from Example 20.5, where the expected total response was 15.39 $\bar{\imath}$. What were the contributions from the direct and social response? For the values used in that example,

$$\sigma(A_d, z) = \sigma^2(A_d) + r(n-1)\sigma(A_d, A_s) = 500 + 0.25 \cdot 5 \cdot (-39.5) = 450.63$$

and

$$\sigma(A_s, z) = \sigma(A_d, A_s) + r(n-1)\sigma^2(A_s) = -39.5 + 0.24 \cdot 5 \cdot 50 = 23.0$$

Recalling (for half-sibs) that $\sigma_z^2 = 1350.6$, Equation 20.15 gives the two components of response as

$$R_d = \frac{450.63}{\sqrt{1350.6}}\,\bar{\imath} = 12.26\,\bar{\imath}, \quad \text{and} \quad R_s = \frac{23}{\sqrt{1350.6}}\,\bar{\imath} = 0.63\,\bar{\imath}$$

Hence, 80% (12.26/ 15.39) of the total response was due to response in direct effects, while 20% was from the response in social effects (5·0.63/ 15.39). Under individual selection on half-sib families, both the mean direct and mean social values improved. By contrast, if group members are unrelated, then (Example 20.5) $\sigma_z^2 = 1150$, while

$$\sigma(A_d, z) = \sigma^2(A_d) = 500, \quad \sigma(A_s, z) = \sigma(A_d, A_s) = -39.5$$

giving responses of

$$R_d = \frac{500}{\sqrt{1150}}\,\bar{\imath} = 14.744\,\bar{\imath}, \quad \text{and} \quad R_s = \frac{-39.5}{\sqrt{1150}}\,\bar{\imath} = -1.165\,\bar{\imath}$$

While the total response in this case was positive, the large direct response (14.74) was significantly offset by a decrease in the mean social environment ($5 \cdot [-1.16] = -5.83$), giving the total response as (14.74 - 5.82) $\bar{\imath}$ = 8.92$\bar{\imath}$. The lack of relatedness implies no direct selection involving $\sigma^2(A_s)$, and hence the social breeding values only change through their correlation with the direct values, which in this example was negative.

Maternal effects

$$z_i = P_{d_i} + P_{m,j} \tag{20.16a}$$

In the absence of inbreeding, $r = 1/2$ for this group (mother-offspring) with $n = 2$. From Equation 20.12c, the covariance between phenotype and total breeding value ($A_T = A_d + A_m$),

$$\sigma(z, A_T) = \sigma^2(A_d) + (3/2)\sigma(A_d, A_m) + (1/2)\sigma^2(A_m), \tag{20.16b}$$

while Equation 20.13a gives the phenotypic variance as

$$\sigma^2(z) = \sigma^2(A_d) + \sigma(A_d, A_s) + \sigma^2(A_s) + \sigma_e^2 \tag{20.16c}$$

giving the resulting response to selection as

total $$R = \frac{\sigma(z, A_T)}{\sigma_z}\bar{i} = \frac{\sigma^2(A_d) + (3/2)\sigma(A_d, A_m) + (1/2)\sigma^2(A_m)}{\sqrt{\sigma^2(A_d) + \sigma(A_d, A_s) + \sigma^2(A_s) + \sigma_e^2}}\bar{i} \tag{20.16d}$$

$$R_d = \frac{\sigma(A_d, z)}{\sigma_z}\bar{i} = \frac{\sigma^2(A_d) + (1/2)\sigma(A_d, A_m)}{\sigma_z}\bar{i}$$ Direct response

$$R_m = \frac{\sigma(A_m, z)}{\sigma_z}\bar{i} = \frac{\sigma(A_d, A_m) + (1/2)\sigma^2_{A_m}}{\sigma_z}\bar{i}$$ Maternal response

Group selection

Unit of selection u = group mean

$$\sigma\left(A_{T_i}, \sum_{j=1}^{n} z_j\right) = \sigma\left(A_{T_i}, \sum_{j=1}^{n} (A_{T_j} + e_j)\right) = \sum_{j=1}^{n} \sigma\left(A_{T_i}, A_{T_j}\right) = \sigma^2(A_T) \sum_{j=1} r_{ij}$$

$$= \sigma^2(A_T)\left(1 + \sum_{j \neq i} r_{ij}\right) \tag{20.19a}$$

If group members are unrelated, then

$$\sigma\left(A_{T_i}, \sum_{j=1}^{n} z_j\right) = \sigma^2(A_T) \tag{20.19b}$$

which implies $\sigma(A_{T_i}, \overline{z}) = \sigma^2(A_T)/n$. Hence, group selection acts on the total breeding value of an individual, rather than on only part of this as is the case with individual selection (e.g., Equation 20.12e). The associative effects contribution to the total breeding value does not influence the phenotype of the focal individual, but does influence the phenotype of other group members. Group selection directly targets these effects. If all members have the same degree of relationship r,

$$\sigma\left(A_{T_i}, \sum_{j=1}^{n} z_j\right) = \sigma^2(A_T)\left[1 + (n-1)r\right] \tag{20.19c}$$

Key: group mean always correlated with A_T

Group selection -- role of relatives

$$\sigma\left(A_{T_i}, \overline{z}\right) = \frac{1}{n}\sigma^2(A_T)\left[1 + (n-1)r\right] = \sigma^2(A_T)\left(r + \frac{1-r}{n}\right)$$

Group of size n, with r = average relatedness among group members

Note that zbar directly correlated with A_T. Correlation increases if members are related (r > 0)

Response under group selection

$$R = \frac{\sigma(A_T, \overline{z})}{\sigma^2(\overline{z})} S = \frac{\sigma^2(A_T) r_n}{\sigma^2(A_T) r_n + \sigma_e^2 \rho_n} S$$

$$= \frac{\sigma(A_T, \overline{z})}{\sigma(\overline{z})} \overline{i} = \frac{\sigma^2(A_T) r_n}{\sqrt{\sigma^2(A_T) r_n + \sigma_e^2 \rho_n}} \overline{i}$$

$$r_n = r + \frac{1 - r}{n} \quad \textbf{and} \quad \rho_n = \rho + \frac{1 - \rho}{n}$$

r = genetic correlation

ρ = environmental correlation among group members

Example 20.7. Consider group selection using Muir's quail data from Example 20.4. Here $\sigma^2(A_d) = 33.7, \sigma^2(A_s) = 2.87, \sigma(A_d, A_s) = -5.5, n = 16$. Muir estimated the residual variance as $\sigma_e^2 = 69.0$, while Muir's model assumed $\rho = 0$, giving $\rho_n = 1/n$ and hence $\sigma_e^2 \rho_n = 69.0/16 = 4.32$. Applying Equation 20.4 gives the total additive variance as

$$\begin{aligned}\sigma^2(A_T) &= \sigma^2(A_d) + 2(n-1)\sigma(A_d, A_s) + (n-1)^2\sigma^2(A_s) \\ &= 33.7 + 30 \cdot (-5.5) + 30^2 \cdot 2.87 = 2451.7,\end{aligned}$$

while Equation 20.26b gives the response as

$$R = \frac{\sigma^2(A_T) r_n}{\sqrt{\sigma^2(A_T) r_n + \sigma_e^2 \rho_n}} \bar{\imath} = \frac{2451.7 \cdot r_n}{\sqrt{2451.7 \cdot r_n + 4.32}} \bar{\imath}$$

For groups of unrelated individuals, $r = 0$ and $r_n = 0.0625$, and the response becomes R= $12.2\,\bar{\imath}$. For half- and full-sibs, r_n = 0.297 and 0.531, with responses of $26.9\bar{\imath}$ and $36.0\,\bar{\imath}$, a two- and three-hold increase relative to a group of unrelated individuals.

Group + kin selection

Unit of selection
u = I is an index

$$I_i = z_i + g \sum_{j \neq i} z_j$$

This index can also be written as

$$I_i = (1-g)z_i + g \sum_{j=1}^{n} z_j = (1-g)z_i + g\, n\, \overline{z},$$

$$R = \frac{\sigma(I, A_T)}{\sigma(I)}\, \overline{\imath}_I$$

g = group selection
r = kin selection

$$\sigma(A_T, I) = [g + r + (n-2)gr]\, \sigma^2(A_T) + (1-g)(1-r)\left[\sigma^2(A_d) + (n-1)\sigma(A_s, A_d)\right]$$

g & r have symmetric roles

Key: Use group + relatives to maximize Cov(u, A_T)

Consequences: Evolution of fitness

Examining the expected change in mean fitness is straightfoward. Using previous results, we simply take the trait being followed as individual fitness ($z = W$). From Equation 20.1c, the fitness of individual i becomes

$$W_i = \mu + A_{d_i} + \sum_{j \neq i} A_{s_j} + e_i \tag{20.47a}$$

A_d is the direct breeding value of fitness, while A_s is the social breeding value (how a focal individual influences the fitness of others in its group). As above, A_{s_i} does not contribute to W_i, while A_{s_j} for $j \neq i$ does. Likewise, as before the total breeding value for fitness of an individual is simply

$$AT_i = A_{d_i} + (n-1)A_{s_i} \tag{20.47b}$$

with variance

$$\sigma^2(A_T) = \sigma^2(A_d) + 2(n-1)\sigma(A_d, A_s) + (n-1)^2\sigma^2(A_s) \tag{20.47c}$$

The first term is the classical additive genetic variance in fitness in the absence of associative effects. When interactions are present, there is the potential for substantially more heritable variation in fitness. Indeed, the total genetic variance in fitness has the potential to exceed the actual variance in individual fitness ($\sigma^2(A_T) > \sigma^2_W$), as much of the variation is hidden in interactions with others, which do not appear in one's individual fitness.

Mean fitness can decrease when associative effects are strong

Applying Equation 20.12c gives the response in terms of the variance components as

$$R_W = \frac{1}{\overline{W}}\left[\sigma^2(A_d) + (n-1)(1+r)\sigma(A_d, A_s) + r(n-1)^2\sigma^2(A_s)\right] \qquad (20.48c)$$

Just as we have seen for other traits, when $r = 0$, the possibility of a reversed response occurs if the breeding value for direct and social effects on fitness are sufficiently negatively correlated. Hence, under rather realistic conditions, individual selection can result in a *decrease* (and a potentially rather significant one at that) in mean fitness.

If the BVs of direct and associative effects on fitness are sufficiently negatively-correlated, can get a reversed response -- fitness goes down

Ironically, even though a negative response can occur in the presence of associative effects, there is actually more total variance potentially available when they are present, as $\sigma^2(A_T) \geq \sigma^2(A_d)$. However, only a fraction of this may be accessible to *individual* selection, and this fraction (being a covariance rather than a variance) can be negative. The key for exploiting the available variance is either selection among groups and/or the presence of relatives in one's group of interacting individuals.

To see this, note from Equation 20.12e that we can express Equation 20.48c as

$$R_W = \frac{1}{\overline{W}}\left(r\sigma^2(A_T) + (1-r)\left[\sigma^2(A_d) + (n-1)\sigma(A_d, A_s)\right]\right) \tag{20.48d}$$

The term in square brackets represents the response in a group of non-relatives. When interactions occur among kin ($r > 0$), then for sufficiently close relatives, the response becomes positive (mean fitness increases) even if it is negative when $r = 0$. At the extreme, when $r = 1$ (all interactions are among clones), the response in mean fitness is simply $\sigma^2(A_T)/\overline{W}$ and all of the heritable variance in fitness is utilized. Conversely, when interactions occur among unrelated individuals, only a fraction of this genetic potential is exploited. This observation lead Bijma (2010a) to suggest that when heritable fitness interactions are present, the key to evolutionary success is interacting with relatives. The reason for this is clear from our previous discussions. With interactions among unrelated individuals, one's phenotype (here fitness) provides very little information about their social breeding value. With interacting kin, the breeding values of the kin's social effects influences your fitness, and these are positively correlated (via kinship) with your own breeding value for social effects.

Direct and social effects responses

Finally, we can decompose the total response in fitness into response from changes in the mean of the direct effects and response from changes in the mean of the social effects. Equation 20.15a gives

$$R_W = R_{W,d} + (n-1)R_{W,s} \tag{20.49a}$$

Recalling Equation 20.48a, Equations 20.15b,c give these response components as

$$R_{W,d} = \frac{\sigma^2(A_d) + r(n-1)\sigma(A_d, A_s)}{\overline{W}} \tag{20.49b}$$

and

$$R_{W,s} = \frac{\sigma(A_d, A_s) + r(n-1)\sigma^2(A_s)}{\overline{W}} \tag{20.49c}$$

Altruistic traits: An example of a reversed response

Example 20.16. Haldane (1932) coined the term **altruistic trait** to denote a behavior (or trait) that harms an individual, but benefits others. The classic example are alarm calls — others in a group are warned (increasingly their fitness), but at some expense to the individual making the call (a direct effect decreasing fitness). Note that the increase in an altrustic trait is an example of a reversed response, as the trait lowers the fitness of the individual that bears it. What are the conditions for such traits to spread? In terms of our fitness model with associative effects (Equation 20.47a), we can rephrase this as the conditions for the mean value of A_s to increase, which are given by Equation 20.49c. From the definition of altruism, $\sigma(A_d, A_s) < 0$, as performing an altruistic act decreases your direct fitness while increasing the fitness of those in your group. Equation 20.49c shows that a necessary (but not sufficient!) condition for altruism to evolve under individual selection is $r > 0$, i.e., individuals interact in groups of relatives.

As pointed out by Bijma and Wade (2008), we can view $\sigma(A_d, A_s)$ as the **cost** $(-c)$ for an altruistic act towards others in your group. Conversely, the altruistic contribution to you from others in your group is $(n-1)\sigma^2(A_s) \geq 0$, which we denote as the **benefit** b. With these definitions, from Equation 20.49c the condition for altruism to evolve under individual selection is just

$$-c + rb > 0, \quad \text{or} \quad r > b/c$$

This is the classic **Hamilton's rule** (Hamilton 1963; 1964a,b).

Inclusive Fitness

As Equation 20.47a illustrates, when heritable interactions are present, the fitness of an individual depends on both their own genes as well as the genes in others. Hamilton (1964a,b) suggested that the focus should shift from individual fitness to what he called **inclusive fitness** — that component of fitness influenced only by the alleles carried by the focal individual. Hamilton argued that individuals strive to increase their inclusive, as opposed to individual, fitness (also see Michod and Abugov 1980, Grafen 2006). Formally, the inclusive fitness of an individual is context-specific, and is defined as individual fitness minus any contribution to that fitness from the group environment plus the effect of that individual on the fitness of others, weighted by relatedness. While sounding rather abstract, when placed in an associative effect framework, this definition is quite clear.

From Equation 20.47a, for individual i, A_{d_i} is the heritable component of individual fitness W_i remaining when the social contributions from others have been removed. The focal individual's social breeding value A_{s_i} does not influence their own fitness, but the social effects of other group members do, with the (heritable) contribution to individual i's fitness from individual j being A_{s_j}. The correlation between the breeding value A_{s_i} carried by i and the contribution to i's fitness from j is their relatedness r_{ij}, so that $r_{ij}A_{s_i}$ is the predicted value of A_{s_j} given A_{s_i}. Putting these together gives the heritable component (i.e., breeding value) of i's inclusive fitness as

$$A_{incl,i} = A_{d_i} + A_{s_i}\sum_{j\neq i}^{n} r_{ij} = A_{d_i} + r(n-1)A_{s_i} \tag{20.51a}$$

where the last equality makes our standard assumption that all group members are equally related (which is easily relaxed). The resulting variance in the breeding value for inclusive fitness becomes

$$\sigma^2(A_{incl}) = \sigma^2(A_d) + 2r(n-1)\sigma(A_d, A_d) + r^2(n-1)^2\sigma^2(A_s) \tag{20.51b}$$

In the absence of heritable associative effects ($\sigma^2(A_s) = 0$) this simply reduces to the additive variance in direct fitness. Importantly, note that the heritable component of inclusive fitness is not the same as the total breeding value A_T for fitness, as

$$A_{T_i} = A_{incl,i} + (1 - r)(n - 1)A_{s_i} \tag{20.51c}$$

Just as Equation 20.49 decomposed the total response into components from direct and associative effects, we can similarly decompose the change in mean individual fitness into change in mean inclusive fitness plus the residual response. From Equation 20.51c,

$$R_W = R_{W,incl} + (1 - r)(n - 1)R_{W,s} \tag{20.52a}$$

so that total response in fitness is the change in inclusive fitness plus any response in the residual of the mean social value (after the effects of group relatives are absorbed into inclusive fitness). From Equation 20.48b, the response in the mean inclusive fitness is given by

$$R_{W,incl} = \frac{1}{\overline{W}}\,\sigma(W, A_{incl}) \tag{20.52b}$$

where

$$\begin{aligned}\sigma(W, A_{incl}) &= \sigma\left(\mu + A_{d_i} + \sum_{j \neq i} A_{s_j} + e_i, A_{d_i} + r(n-1)A_{s_i}\right) \\ &= \sigma^2(A_d) + 2r(n-1)\sigma(A_d, A_d) + r^2(n-1)^2\sigma^2(A_s)\end{aligned} \tag{20.52c}$$

The last line follows by evaluating the covariance in a similar fashion as done throughout this chapter. Note by comparison with Equation 20.51b, that this is simply $\sigma^2(A_{incl})$, yielding

$$R_{W,incl} = \frac{\sigma^2(A_{incl})}{\overline{W}} \tag{20.52d}$$

Hence (under our simple model), the response in mean inclusive fitness is proportional to the additive variance in inclusive fitness, so that mean inclusive fitness is non-decreasing. Why, then, can the mean of *individual* fitness decline despite the continual increase in mean inclusive fitness? The reason is an even faster decline in the mean (residual) social value. Recalling Equation 20.49c, Equation 20.51a becomes

$$R_W - R_{W,incl} = \frac{(1-r)(n-1)}{\overline{W}}\Big(\sigma(A_d, A_s) + r(n-1)\sigma^2(A_s)\Big) \tag{20.52e}$$

Hence, if the covariance between direct and associative effects is sufficiently negative, any increase in inclusive fitness is more than countered by the decline in the mean social environment. Note that increasingly the relatedness of group members decreases the residual response between mean individual and inclusive fitness, which in turn increases the chances that individual mean fitness increases.

Key: mean inclusive fitness (unlike individual fitness) is non-decreasing