# 16

# Environmental Effects and the Response in Natural Populations

*Associations between phenotype and fitness, however appealing, will give a misleading impression of the potential for evolution in a trait if the true target of selection is unmeasured or immeasurable.* — Kruuk et al. 2002

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Under artificial selection (animal/plant breeding and laboratory selection experiments), the machinery developed in Chapters 10-15 for the prediction and analysis of response generally works well. However there is some angst as to whether this is also true for natural populations (e.g., Merilä et al. 2001c). There are two principal reasons for this. First, in natural populations, one typically *infers* the target of selection by looking for changes in the mean and/or variance of certain candidate traits. The problem is that the moments of an unselected trait can change if it is correlated with another trait under selection. A within-generation change occurs if an unselected trait is phenotypically correlated to a selected one, while a between-generation change occurs if they are genetically correlated. Chapters 30-33 examine these issue in detail.

The focus of this chapter is the second complication: lack of control over the environment. With artificial selection, there is typically considerable control over the environment, in part due to husbandry/cultivation methods designed to standardize environmental effects. This is certainly not the case when attempting to track selection in natural populations. Indeed, we largely ignorant of which environmental factors may be important, much less being able to control them. Further, artificial selection experiments generally have considerable control over the biotic environment in which an organism finds itself. In natural populations, the biotic environment is both absolutely critical and largely uncontrollable. One problem with lack of environment control is fitness-trait correlations (and hence a selection differential) caused by unmeasured environmental factors. Changes in the environment can mask underlying genetic changes and can also result in significant changes in the nature of selection from one generation to the next. Finally, changes in the environment can change the heritability of our focal trait by changing genetic and/or environmental variances.

Building around this central theme of environmental effects on selection response, our discussion starts with a brief introduction of the complications created by correlated traits as a prelude to a much more detailed discussion later (Chapters 30-33). We then examine the complications that environmental correlations between fitness and a focal trait introduce in predicting change in that focal trait. This leads to a classic analysis by Price et al. using such an environmental correlation to account for apparent lack of response to selection for early breeding date in birds. We follow by extending the (single-trait) breeders' equation to cover some of the environmental issues that arise in natural populations. We then examine approaches for detecting if selection is occurring on the breeding, as opposed to the environmental, value of our focal trait. A major recent growth industry in ecological genetics has been the use of the animal model (Chapter 15) to estimate the breeding values of individuals, although this is limited by requirement of the pedigree of the wild individuals in the

study. We conclude by examining a number of studies that attempt to ascertain the causative factor(s) for apparent failures of response on a heritable trait under (apparent) selection in natural populations. Again, our focus is on the change in the mean of a single trait. Many of these problems with selection in natural populations arise from selection on a vector of traits, so our analysis here is a step towards the much more complete treatment (under a multivariate framework) in later chapters.

## RESPONSE IN NATURAL POPULATIONS: WHAT IS THE TRAGET OF SELECTION?

Obviously, the breeder's equation can give misleading results if we incorrectly assign the target of selection. We can see this with a simple application of the multivariate breeders' equation.

### Direct and Correlated Responses

Recall that we briefly introduced the multivariate version of the breeders' equation in Chapter 10, with the vector $\mathbf{R}$ of responses (changes in means) being a function of the genetic (breeding value) $\mathbf{G}$ and phenotypic $\mathbf{P}$ covariance matrices for the traits of interest and the vector $\mathbf{S}$ of selection differentials. From Equations 10.22b and 10.23b,

$$\mathbf{R} = \mathbf{G}\mathbf{P}^{-1}\mathbf{S} = \mathbf{G}\,\boldsymbol{\beta}$$

where the selection gradient $\boldsymbol{\beta} = \mathbf{P}^{-1}\mathbf{S}$ controls for any phenotypic correlations among the measured traits, returning the amount of direct selection acting on each particular character (LW Chapter 8; Chapter 30). While we will examine these equations in considerable detail later (Chapters 31 - 37), the two-trait version provides insight into some of the complications than can arise by incorrectly identifying the target traits(s) under selection. Suppose we are following a trait $z_1$, influenced by a second (and unmeasured) trait $z_2$. Noting that $\mathbf{S} = \mathbf{P}\boldsymbol{\beta}$, the selection differential on trait 1 becomes

$$S_1 = \sigma^2(z_1)\beta_1 + \sigma(z_1, z_2)\beta_2 \tag{16.1a}$$

Thus, a within-generation change can occur in trait one because (i) direct selection on trait 1 ($\beta_1 \neq 0$) and/or (ii) indirect selection on a *phenotypically*-correlated trait under directional selection ($\beta_2 \neq 0$ *and* $\sigma(z_1, z_2) \neq 0$). Likewise, the expected response on trait 1 is

$$\Delta\mu_1 = \sigma^2(A_1)\beta_1 + \sigma(A_1, A_2)\beta_2 \tag{16.1b}$$

Again, trait 1 can change from direct selection on trait 1 (given it has heritable variation) and/or a correlated response from direct selection on a trait that is *genetically* correlated to trait 1 (the breeding values of the two traits are correlated within individuals).

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**Example 16.1.** Alatalo et al. (1989) examined tarsus length in the collared flycatcher (*Ficedula albicollis*) in a population using nest boxes residing in the southern part of the island of Gotland in the Baltic Sea. Measurements of lifetime fitness on this isolated population are possible since most surviving offspring (which are tagged before fledging) return to breed in the area they were reared as offspring. In addition to tarsus length, fledging weight was also measured and a Pearson-Lande-Arnold regression (Chapter 31; LW Chapter 8) preformed to compute the amounts of direct selection (the estimated selection gradients $\beta$) on both characters.

| Year | Observed $S$ on tarsus length | Estimated selection gradients, $\beta$ tarsus length | fledging weight |
|---|---|---|---|
| 1981 | 0.19** | 0.01 | 0.25* |
| 1983 | 0.08 | -0.01 | 0.21* |
| 1984 | 0.20** | 0.12 | 0.33*** |
| 1985 | 0.02 | -0.06 | 0.27*** |
| pooled | 0.12** | 0.03 | 0.27*** |
| | $* = p < 0.05$, | $** = p < 0.01$, | $*** = p < 0.001$ |

Although there is a significant selection differential on tarsus length in two of the years (and in the pooled data), there is no significant direct selection on tarsus length itself ($\beta$ not significantly different from zero). Rather, direct selection is on fledging weight. While there is a significant phenotypic correlation between tarsus length and fledging weight ($r = 0.32$, $p < 0.001$), it appears to be entirely due to within-individual correlations of environmental effects as there is no correlation between offspring weight and parental tarsus length ($r = -0.01$, $p > 0.1$). Hence, the observed selection on tarsus length is a consequence of selection on fledging weight, which has no genetic correlation with tarsus length and hence no response in tarsus length is expected.

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As the last example highlights, some of the best studies of the response to selection in natural populations come from birds. In certain setting (such as isolated islands), the entire population can be banded and all nests located, allowing for an accurate measurement of individual fitness (Chapter 29). We examine a number of such classic studies throughout this chapter.

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**Example 16.2.** As reviewed in Grant and Grant (1995, and references therein), one of the best documented cases of natural selection is on body size and bill morphology in Darwin's finches (*Geospiza fortis*) on the Galápagos island of Daphne Major. Two strong episodes of selection were observed during their long-term study, due to serious droughts in 1976 –1977 (where the population crashed from 634 birds down to 95, a 15% survival rate), and in 1984 – 1986 (556 birds down to 180 birds, a 32% survival rate).

Six morphological traits were followed through both episodes, and (following rescaling of all traits to give them unit variances), the following selection differentials $S$ and gradients $\beta$ over the two episodes were as follows:

| | 1976 - 1977 | | 1984 - 1986 | |
|---|---|---|---|---|
| Trait | $S$ | $\beta$ | $S$ | $\beta$ |
| Weight | $0.74^*$ | $0.477^*$ | $-0.11$ | $-0.040$ |
| Wing lenght | $0.72^*$ | $0.436^*$ | $-0.08$ | $-0.015$ |
| Tarsus lenght | $0.43^*$ | 0.005 | $-0.09$ | $-0.047$ |
| Bill lenght | $0.54^*$ | $-0.144$ | $-0.03$ | $0.245^*$ |
| Bill depth | $0.63^*$ | $0.528^*$ | $-0.16^*$ | $-0.135$ |
| Bill width | $0.53^*$ | $-0.450^*$ | $-0.17^*$ | $-0.152$ |

where $* = p < 0.05$

Two striking features are apparent from this data. First, the observed (within-generation) change in mean ($S$) is not a good predictor of the actual amount of direct selection ($\beta$) on a trait, and can even be of the wrong sign (e.g., tarsus length)! Second, the nature of selection changed over the two drought periods. During the 1976 – 1977 drought, larger individuals

were favored, and there was selection on bill shape (increased bill depth while decreasing bill width). A change in the dominant food supply during a subsequent drought from 1984 – 1986 drought resulted for selection favoring smaller birds. Hence, the two episodes of selection were in opposite directions (at least in terms of body size).

Grant and Grant used the multivariate breeders equation to examine how well responses were predicted. As shown below, response was well predicted in 1976, but over-predicted in three of the six traits in the 1984 episode. Grant and Grant suggest that the main reason for these discrepancies was an environmental change. Higher population densities for offspring in 1984 retarded growth, resulting in an over-prediction of response. We will return often to this notion of an environmental change across generations comprising prediction of response in natural populations.

| | 1976-1977 | | 1984-1986 | |
|---|---|---|---|---|
| Character | Predicted | Observed | Predicted | Observed |
| Weight | $17.39 \pm 0.22$ | $17.52 \pm 0.25$ | $16.82 \pm 0.13$ | $15.48 \pm 0.08^{*}$ |
| Wing length | $69.98 \pm 0.39$ | $69.65 \pm 0.35$ | $67.93 \pm 0.17$ | $67.21 \pm 0.11^{***}$ |
| Tarsus length | $19.45 \pm 0.09$ | $19.32 \pm 0.14$ | $19.02 \pm 0.04$ | $19.02 \pm 0.04$ |
| Bill length | $11.14 \pm 0.10$ | $11.06 \pm 0.11$ | $10.86 \pm 0.05$ | $10.96 \pm 0.03$ |
| Bill depth | $9.83 \pm 0.12$ | $9.94 \pm 0.09$ | $9.51 \pm 0.06$ | $9.32 \pm 0.03^{**}$ |
| Bill width | $8.96 \pm 0.08$ | $8.97 \pm 0.08$ | $8.77 \pm 0.04$ | $8.70 \pm 0.03$ |

where $* = p < 0.05$, $** = p < 0.01$, $*** = p < 0.001$

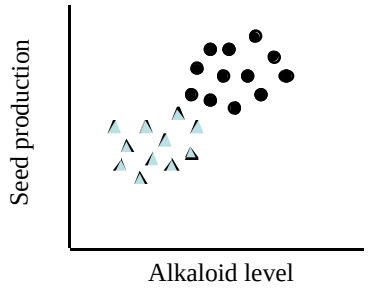


**Figure 16.1**. An environmental variable (soil nitrate) influences both fitness and trait value (alkaloid levels), creating a covariance between the trait and fitness, when in fact the trait value is not causal to fitness. In low nitrate soils (triangles), plants have low fitness and low levels of alkaloids. In high nitrate soils (circles), plants have high fitness and high levels of alkaloids. Within each of the two environments, there is no association between trait and fitness. If one ignores the environmental effects and simply lumps all individuals together, there is a strong association between fitness and the trait value. Figure based on Mauricio and Mojonnier (1997) and Rausher (1992).

### Environmental Correlations Between Fitness and Traits

In natural populations, an environmental factor (or factors) can influence both an individual's trait value and their fitness. This generates a correlation between the trait and fitness, and hence a selection differential on a trait, even if there is no direct selection on the trait itself. Consider the following example, suggested by Rauscher (1992). Suppose soil nitrate concentration influences both fitness (seed production) and the amount of alkaloids (secondary plant chemicals) in the foliage of a plant. As Figure 16.1 shows, if we are able to

partition individuals from a population into high and low nitrate environments, within each group we would find no association between alkaloid concentration and fitness. However, if we ignore this partition and simply consider all individuals as a single group, there is a positive covariance between alkaloid concentration and fitness. An investigator unaware of this difference in soil nitrates might conclude a fitness effect from the presence of alkaloids (for example, as an insect deterrent), when in fact the correlation between trait and fitness simply arises solely because both are influenced by a third, and unmeasured, variable.

Rausher's example introduces the concept of selection on a **non-heritable environmental component** of a trait. Here, soil nitrate is the primary determinant of plant fitness. However, this is a non-heritable environmental trait. (In reality, one could imagine that plants have some genetic variance in their ability in recruit into high-nitrate soils, but we will ignore this possibility for our example). Thus plants with high alkaloid levels, which are heritable, have higher fitness. However, alkaloid level does not increase in the next generation, as plants with high *environmental* values for alkaloids were selected, not plants with high *genetic* values, and hence no response. Put another way, there is selection on the *environmental* value of the trait, *not* on its breeding value or is phenotype.

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**Example 16.3**. Price and Liou (1989), considering the evolution of **clutch size** ($z_1$) in birds (number of eggs laid in a particular episode), suggested that fitness is largely determined by the nutritional state $z_2$ of the bird, which in turn influences her clutch size. Further, they assumed that this nutritional state has no heritable component and hence can be treated as a non-heritable environmental factor. Equation 16.1a implies that, even if there is no direct selection on clutch size *per se* ($\beta_1 = 0$), we would still observe a selection differential on clutch size if it is phenotypically-correlated with nutritional state which is itself under selection ($\beta_2 \neq 0$), as

$$S_1 = \beta_2\, \sigma(z_1, z_2) \neq 0$$

However, if there is no additive-genetic variance in the nutritional state, then $\sigma^2(A_2) = 0$ and hence $\sigma(A_1, A_2) = 0$. Thus, in the absence of direct selection on clutch size ($\beta_1 = 0$), any response is clutch size arises from a correlated response on nutritional state, with Equation 16.1b giving the response as

$$\Delta\mu_1 = \beta_2\, \sigma(A_1, A_2)$$

but this contribution is zero because $\sigma(A_1, A_2)$ is zero as $z_2$ has no heritable variance. The result is an apparent directional selection on clutch size ($S_1 \neq 0$), but no response ($R_1 = 0$).

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## THE FISHER-PRICE-KIRKPATRICK-ARNOLD MODEL FOR EVOLUTION OF BREEDING DATE IN BIRDS

The notion (introduced in Example 16.3) of a nonheritable environmental factor being the target of selection dates back to Fisher and Darwin. Fisher (1958), based on observations by Darwin (1871), suggested that the health status of a bird influences both her clutch size and the date at which she breeds, with healthier females breeding earlier and having larger clutch sizes. Fisher assumed that health can be thought of as a nonheritable environmental value due to nutritional state, with females having higher nutritional values breeding earlier and having larger clutches. A potential measure of "nutritional value" is **condition**, body mass corrected for skeletal mass, and we will return to this trait in several of the studies discussed below.

Price, Kirkpatrick and Arnold (1988) used Fisher's idea to account for apparent the lack of selection response for breeding date in many birds in the temperate zone. Birds that reproduce early have higher fitness than those who breed later in the season, and hence $S$ for breeding date is negative (selection to move the breeding date earlier in the season). Further (where examined) breeding date has moderate to high heritability. Since both $h^2$ and $S$ are nonzero, we expect a response to selection resulting in a decrease in breeding date, but this is not seem.

Price et al. (1988) formally developed a quantitative-genetic model to account for this apparent lack of response (Figure 16.2). For brevity, we will often refer to the **Fisher-Price-Kirkpatrick-Arnold model** as simply **Fisher's model** (we will resist the temptation of referring to this as the Fisher-Price toy model). The model is as follows: assume that the breeding date $z$ of an female has three components,

$$z = A - n + e \tag{16.2}$$

Here $A$ is the breeding value for date, $e$ the environmental value, and $n$ the nutritional state of the female. Equation 16.2 shows that females with a higher value of $n$ (higher nutritional status) breed earlier. Price et al. (following Fisher) treat $n$ as a nonheritable component of the environment, but one could also model $n$ as a trait with both heritable and nonheritable components, changing this to a selection on multiple characters problem (Chapters 30 - 33). For now, we assume $n$ is nonheritable, with all three components of Equation 16.2 uncorrelated and normally distributed with variances $\sigma_z^2$, $\sigma_n^2$, and $\sigma_e^2$. Let $\mu$ be the current mean breeding value, while we take the mean of $n$ and $e$ to both be zero.

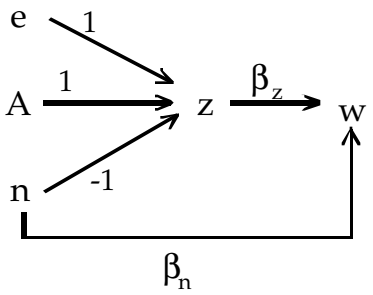


**Figure 16.2**. A path diagram (LW Appendix 1) of the components in the Fisher-Price-Kirkpatrick-Arnold model, showing the connections between breeding data $z$, nutritional state $n$, and fitness $w$. The breeding value $A$, general environmental value $e$, and nutritional state $n$ all influence breeding date $z$, which itself influences fitness (path coefficient $\beta_z$). Likewise, there is a second path to fitness directly from nutritional state ($\beta_n$) which represents the direct contribution to $w$ once the contribution of $n$ through breeding date $z$ is removed. Note that we assume $A$, $e$, and $n$ are all uncorrelated, and hence not connected by any paths. (After Price et al. 1988.)

Price et al. impose selection by considering two separate components. First, they assume there is an optimum breeding date $\theta$ so that $z$ is under stabilizing selection. A standard model for stabilizing selection in natural populations is to assume **normalizing** (or **nor-optimal**) selection (Weldon 1895, Haldane 1954),

$$W(z) = \exp\left( - \frac{(z-\theta)^2}{2\,\omega^2} \right) \tag{16.3a}$$

This fitness function, giving the expected fitness of an individual with phenotypic value $z$, has the same functional form as a normal distribution, with the highest fitness at the optimal phenotypic value $z = \theta$, declining as one moves away from $\theta$. The strength of selection is given by $\omega^2$ (the "variance" of the fitness function). If $\omega^2 >> \sigma_z^2$, fitness falls off slowly as we depart from $\theta$ and selection is weak, while if $\omega^2 << \sigma_z^2$ selection is strong. One advantage of assuming a fitness function of the form given by Equation 16.3a is that if $z$ is normally distributed before selection, it remains normally distributed following selection, and expressions for the new mean and variance are easily obtained (Chapters 41–43). Second, Price et al. assumed that fitness increases with the nutritional value $n$. One way to express this is to assume that the fitness given $n$ is

$$W(n) = \exp(\alpha n) \tag{16.3b}$$

Note that if $|\,\alpha\, n\,| << 1$, then $W(n)$ is approximately $1 + \alpha n$. As with the nor-optimal fitness function, the exponential fitness function also allows traits normally-distributed before selection to remain normal following selection. The resulting joint fitness given both breeding date $z$ and nutritional value $n$ is

$$W(n, z) = W(z) \cdot W(n) = \exp\left(\alpha n - \frac{(z-\theta)^2}{2\,\omega^2}\right) \tag{16.4a}$$

Expressed in terms of the components of our model,

$$W(n, e, A) = \exp\left(\alpha n - \frac{(A - n + e - \theta)^2}{2\,\omega^2}\right) \tag{16.4b}$$

Assuming $A$ is normally distributed with mean $\mu$, while $n$ and $e$ are normally distributed with mean zero, then the change in mean (the selection response) is given by (Heywood 2005),

$$R = \sigma_A^2\, \frac{\theta - \mu + \alpha\sigma_n^2}{\omega^2 + \sigma_z^2} \tag{16.5}$$

At equilibrium ($R = 0$), the mean breeding date is

$$\widehat{\mu} = \theta + \alpha\sigma_n^2 \tag{16.6}$$

which is later than the optimal breeding date $\theta$. Price et al. comment that this displacement beyond the optimal breeding date occurs because females in good nutritional condition are constrained (Equation 16.2) to breed earlier than the mean breeding value at equilibrium. Since $\widehat{\mu} > \theta$, females in good nutritional condition ($n < 0$) have a mean breeding date ($A + e - n$) closer to the optimal breeding date $\theta$. Price et al. note that this model may also apply to clutch size in birds (Price and Liou 1989) and might also be a reasonable model for seed germination time, especially if there is a significant nonheritable nutritional contribution from the maternal endosperm.

Additional insight into Fisher's model was offered by Heywood (2005), who used his decomposition of the Price Equation (Chapter 12) to partition the response into a linear and a spurious response term (Equation 12.25),

$$R = \beta_{z',z}\, S + \sigma(w, z' \,|\, z) + E[\,\Delta z\,]$$

Since the basic Fisher model assumes random mating, no epistasis nor cross-generational environmental effects, the offspring mean in the absence of selection is the same as the

current parental means and hence $E[\,\Delta z\,] = 0$. Heywood shows that the linear response to selection is given by

$$\beta_{z',z} S = \sigma_A^2 \, \frac{\theta - \mu - \omega^2 \alpha \sigma_n^2 / \sigma_z^2}{\omega^2 + \sigma_z^2} \tag{16.7a}$$

(Recall, from Chapter 12, that $z'$ is the offspring value, so that $\beta_{z',z}$ is the slope of the parent-offspring regression.) The linear response component is zero when

$$\widehat{\mu_L} = \theta - \omega^2 \alpha \sigma_n^2 / \sigma_z^2 \tag{16.7b}$$

This is the value for which fitness is optimized with respect to breeding date. Likewise, the correlation between fitness ($w$) and offspring value ($z'$) when the effect of parent breeding date is removed is

$$\sigma(w, z' \mid z) = \alpha \sigma_n^2 \sigma_A^2 / \sigma_z^2 \tag{16.8}$$

As discussed in Chapter 12, this covariance term can be thought of as the spurious response to selection because it is independent of $z$, the trait under selection. At equilibrium (Equation 16.6) $\widehat{\mu} - \theta = \alpha \sigma_n^2$, and the linear response to selection (Equation 16.7a) reduces to $-\alpha \sigma_n^2 \sigma_A^2 / \sigma_z^2$, which is exactly canceled by the spurious response, given a net total response of zero. The reason for a non-zero covariance between $w$ and offspring value $z'$ (conditional on parental value $z$) is that fitness is proportional to nutritional status and (due to the exponential nature of the fitness function), residuals of the (linear) fitness regression of $w$ on $z$ are non-random, increasing with $z$. Likewise, when offspring mean $z'$ is regressed on $z$, residuals are proportion to $n$ (since $z' = z + n - e$). As a result, the residuals of both the regressions of $z'$ on $z$ and $w$ on $z$ covary with $n$, generating a conditional covariance (Chapter 12). Thus, the spurious response arises because both regressions are influenced by a common variable, the nutritional status $n$.

## MODIFYING THE BREEDERS' EQUATION FOR NATURAL POPULATIONS

As the above model shows, one of the most serious limitations to applying the breeders' equation to natural populations is that selection can occur on (unmeasured) characters that are correlated with the particular trait under consideration. Further, genotype-environment *correlations* can be a concern, as (for example) larger individuals may be able to obtain the best environments.

Just how these complications bias the breeders' equation was examined by van Tienderen and de Jong (1994). They assume complete additivity (no dominance or epistasis), multivariate normality, and linear parent-offspring regressions. As discussed in Chapter 12, under these conditions a more general expression for the response to selection is given by Robertson's Secondary Theorem of Natural Selection, which states that response equals the covariance between relative fitness $w$ and trait breeding value $A$, $R = \sigma(w, A)$. As shown in Figure 16.3, van Tienderen and de Jong use a path analysis argument (LW Appendix 2) to explore the relationship between response $R$ and the selection differential $S$ when complications such as selection on correlated characters and genotype-environment correlations exist.

To present their analysis, decompose the phenotype $z$ as

$$z = A + E + E_s$$

where $A$ is the additive genetic value, $E$ the general environmental value (for example, the average value for a particular macrohabitat) and $E_s$ the special environmental value unique

to each individual (LW Chapter 6). By construction, $E_s$ is independent of the other variables (so that the total environmental variance is $\sigma^2_E + \sigma^2_{E_s}$), but $A$ and $E$ may be correlated. Consider Figure 16.3, which shows possible paths of how the environmental value $E$, the genotypic value $A$, and the phenotypic value $z$ can influence fitness. The standard breeders' equation assumes $E$ and $A$ influence fitness only through phenotypic value $z$. van Tienderen and de Jong examine the more general situation where $E$ and $A$ can influence fitness independent of (or in addition to) their effects on $z$, as can occur if the character is phenotypically and/or genetically correlated with other characters under selection. If fitness is entirely determined by the phenotypic value of the character of interest, there should be no expected differences in fitness of individuals with the same phenotypic value $z$ but different underlying genetic ($A$) or environmental values ($E$). However, if other correlated characters are under selection, then individuals with the same $z$ value can have different fitnesses as correlations between their $A$ and/or $E$ values with the genetic and/or environmental values at other traits that influence fitness (Chapters 29 - 31).

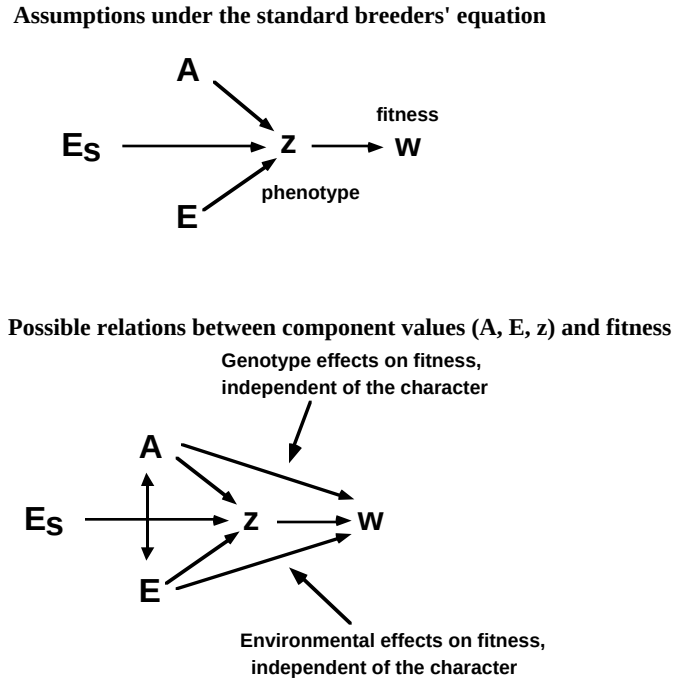


**Figure 16.3**. The pathways by which the components of a character (phenotype $z$, additive genetic value $A$, common environmental effect $E$, and special environmental effect $E_s$) influence fitness $w$. **Top**. The breeders' equation assumes that only the phenotype ($z$) of a character influences fitness. This is not an unreasonable starting assumption for artifical selection, wherein the breeder directly chooses individuals on the basis of phenotypes and randomizes environments with respect to phenotypes. **Bottom**. Other pathways by which the components of a character can influence fitness. Either (or both) of the additive genetic and/or environmental value can influence fitness independent of their influence on phenotype. For example, an environmental value can influence both the character of interest and independently influence fitness. Likewise, the genotypic ($A$) and environmental ($E$) values can be correlated, which is indicated by the double-headed arrows connecting these.

To quantify the effects from these different paths influencing fitness, van Tienderen and

de Jong consider the multiple regression of relative fitness $w$ as a function of $z$, $A$, and $E$, viz.,

$$w = \alpha + \beta_z\, z + \beta_A\, A + \beta_E\, E + \epsilon \tag{16.10}$$

The partial regression coefficients $\beta$ represent the expected change in fitness holding the other variables constant (LW Chapter 8). For example, $\beta_z$ is the effect of phenotype $z$ on fitness, holding the other variables ($A$ and $E$) constant. From multiple regression theory (LW Chapter 8), the partial regression coefficients satisfy

$$\begin{pmatrix} \sigma(w,z) \\ \sigma(w,A) \\ \sigma(w,E) \end{pmatrix} = \begin{pmatrix} S \\ R \\ \sigma(w,E) \end{pmatrix} = \begin{pmatrix} \sigma_z^2 & \sigma(z,A) & \sigma(z,E) \\ \sigma(z,A) & \sigma_A^2 & \sigma(A,E) \\ \sigma(z,E) & \sigma(A,E) & \sigma_E^2 \end{pmatrix} \begin{pmatrix} \beta_z \\ \beta_A \\ \beta_E \end{pmatrix} \tag{16.11}$$

Recall that $S = \sigma(w,z)$ follows from the Robertson-Price identity (Chapter 10) and $R = \sigma(w,A)$ from Robertson's Secondary Theorem. The first vector contains the covariances between relative fitness $w$ and the predictor variables ($z$, $A$, and $E$), while the matrix is the variance-covariance matrix for these predictor variables. Note that

$$\sigma(z,A) = \sigma(A + E + E_s, A) = \sigma_A^2 + \sigma(E,A)$$

Likewise, $\sigma(z,E) = \sigma_E^2 + \sigma(E,A)$. Using these identities and considering the first two rows of Equation 16.11 gives the within-generation change as

$$\begin{aligned} S &= \sigma_z^2\, \beta_z + \left[\, \sigma_A^2 + \sigma(E,A) \,\right] \beta_A + \left[\, \sigma_E^2 + \sigma(E,A) \,\right] \beta_E \\ &= \sigma_z^2\, \beta_z + \sigma_A^2\, \beta_A + \sigma_E^2\, \beta_E + \sigma(E,A)\ (\beta_A + \beta_E) \end{aligned} \tag{16.12a}$$

and the response as

$$\begin{aligned} R &= \left[\, \sigma_A^2 + \sigma(E,A) \,\right] \beta_z + \sigma_A^2\, \beta_A + \sigma(E,A)\, \beta_E \\ &= \sigma_A^2\, \beta_z + \sigma_A^2\, \beta_A + \sigma(E,A)\ (\, \beta_z + \beta_E) \end{aligned} \tag{16.12b}$$

If there are no genotype-environment correlations [$\sigma(E,A) = 0$],

$$R = \sigma_A^2\ (\beta_z + \beta_A) \tag{16.13a}$$

and

$$S = \sigma_z^2\, \beta_z + \sigma_A^2\, \beta_A + \sigma_E^2\, \beta_E \tag{16.13b}$$

Multiplying both sides of Equation 16.13b by $h^2$ and rearranging gives

$$\sigma_A^2\, \beta_z = h^2\, \sigma_z^2\, \beta_z = h^2 \left( S - \left[\, \sigma_A^2\, \beta_A + \sigma_E^2\, \beta_E \,\right] \right)$$

Substituting into Equation 16.13a gives

$$R = h^2\, S + \sigma_A^2 (1 - h^2)\, \beta_A - h^2\, \sigma_E^2\, \beta_E \tag{16.13c}$$

Hence, (positive) selection entirely on additive genetic values ($\beta_A > 0$) inflates response over the breeders' equation, while selection on entirely environmental values ($\beta_E > 0$) decreases response relate to the breeders' equation. Following this same approach gives the general response [when $\sigma(E,A) \neq 0$] as

$$R = h^2\, S + \sigma_A^2 (1 - h^2)\, \beta_A - h^2\, \sigma_E^2\, \beta_E + \sigma(E,A)\ \left( \beta_z - h^2\, \beta_A + (1 - h^2)\, \beta_E \right) \tag{16.14}$$

If selection acts only on the phenotype of the character being considered, then $\beta_A = \beta_E = 0$ and Equation 16.12a reduces to $S = \sigma_z^2 \beta_z$, implying $\beta_z = S/\sigma_z^2$. Substituting into Equation 16.12b gives the response as

$$R = \beta_z \left[\sigma_z^2 + \sigma(E,A)\right] = \left(h^2 + \frac{\sigma(E,A)}{\sigma_z^2}\right) S \tag{16.15a}$$

which (as expected) reduces to the breeders' equation when there is no genotype-environment correlation. Under artifical selection, it is generally assumed (to a first approximation) that individual fitness is entirely based on the phenotype of the character of interest, specifically those phenotypes chosen by the breeder. In this case, the partial regression coefficients of fitness on genotype and environmental values are zero (again, as first approximation), as phenotype entirely determines fitness. In natural populations, we do not have this luxury and another possibility is that there is no natural selection on the character of interest (its phenotype, by itself, has no effect on fitness so that $\beta_z = 0$), but rather selection occurs on characters correlated with the one we are following. If these characters under selection are only connected to the character we are following through genetic value (i.e., no environmental correlation between characters), then $\beta_A \neq 0$ while $\beta_z = \beta_E = 0$. In this case, using Equation 16.13a to express $\beta_A$ in terms of $S$ gives the response as

$$R = \beta_A \sigma_A^2 = S \frac{\sigma_A^2}{\sigma_A^2 + \sigma(E,A)} \tag{16.15b}$$

which reduces to $R = S$ in the absence of genotype-environment correlations. The reason for this strong response is that all of the selection is on the breeding value. With selection on a phenotype, only $h^2$ of this translates into the breeding values.

A final possibility is that the only correlation between characters under selection and our character is through shared environmental effects, giving $\beta_E \neq 0$ while $\beta_A = \beta_z = 0$, in which case the response (again using Equation 16.12a) becomes

$$R = \beta_E \sigma(E,A) = S \frac{\sigma(E,A)}{\sigma_E^2 + \sigma(E,A)} \tag{16.15c}$$

which equals zero unless a genotype-environment correlation exists.

## SELECTION ON ENVIRONMENTAL VERSUS GENOTYPIC VALUES

As we have seen, an observed selection differential can be generated by direct selection on a trait, direct selection on phenotypically correlated traits, an environmental covariance between our focal trait and fitness, or a combination of all of these. Methods for treating phenotypically-correlated traits are discussed in Chapter 30, so our focus here is how can we distinguish between selection acting on the actual phenotypic value versus acting on an environmental component. Ideally, we could separate the phenotypic values of the individuals in our sample into their genotypic and environmental values, and then examine the covariance of each with fitness. An obvious problem with this approach is the estimation of genotypic or breeding values. Two approaches have been suggested to do this.

The first is to replicate genotypes (when clones are available) or sibs (half, full, or selfed) over environments, estimating the genotypic value of a clone by its average over the sampled environments and likewise assigning all sibs the same breeding value, namely their family mean. This approach was initially suggested by Rausher and Simms (1989) and Rausher (1992). One of the interpretation issues with this approach is that individuals are

randomized over the specified environments by the investigator, but hidden (yet critical) environmental factors may be missed.

A second approach is to use BLUP/REML machinery (Chapter 15) to directly estimate individual breeding values. While potentially much more powerful (as we treat individual, rather than group, breeding values), this approach requires the relationship matrix **A** for all the individuals in the sample, which requires obtaining their pedigree. We examine each of these approaches in turn. As we will see, both have biological and statistical limitations.

**Rausher's Method**

A formal test of selection on breeding (or genotypic) values using sibs was suggested by Rausher and Simms (1989) and Rausher (1992), and is obtained as follows. First, consider the decomposition the phenotypic selection gradient $\beta_z$ into gradients on the breeding value $A$ and environmental deviation $\epsilon = z - A$. Recall (Chapter 10) that the directional selection gradient is given by

$$\beta_z = \frac{\sigma(z,w)}{\sigma_z^2}$$

where $\beta_z$ is the slope of the best linear regression predicting relative fitness $w$ as a function of $z$, e.g., $w = 1 + \beta_z(z - \mu_z) + e$. Since $\sigma(w,z) = \sigma(w, A+\epsilon) = \sigma(w,A) + \sigma(w,\epsilon)$, and noting that $\sigma_A^2/h^2 = \sigma_z^2$ and $\sigma^2(\epsilon)/(1-h^2) = \sigma_z^2$, we have

$$\begin{aligned}\beta_z = \frac{\sigma(w,A) + \sigma(w,\epsilon)}{\sigma_z^2} &= \frac{\sigma(w,A)}{\sigma_A^2}h^2 + \frac{\sigma(w,\epsilon)}{\sigma_\epsilon^2}(1-h^2) \\ &= \beta_A h^2 + \beta_\epsilon(1-h^2) \end{aligned} \tag{16.16}$$

Rausher (1992) shows that, in the absence of environmentally-induced correlations between a trait and fitness, $\beta_z = \beta_A$. In this case, Equation 16.16 implies that $\beta_z = \beta_\epsilon$. This observation suggests a test for an environmentally-induced fitness-trait correlation. Following Rausher and Simms (1989) and Rausher (1992), and as corrected by Stinchcombe et al. (2002), compute the multiple regression of fitness on both the estimated breeding value $A$ and the environmental deviation $\epsilon$,

$$w = 1 + \beta_1 A + \beta_2 \epsilon + e \tag{16.17}$$

In the absence of environmental correlations between both the trait and fitness, $\beta_1 = \beta_2$, which can be tested in a straightforward fashion using standard results from regression theory (LW Chapter 8). Here, sib (or clone) means replicated over environments are used for the breeding/genotypic values, so that there are different sample sizes associated with $\beta_1$ (number of families) and $\beta_2$ (number of individuals). Stinchcombe et al. (2002) discuss how to deal with this issue. Also note that another test is whether $\beta_1$ is significantly different from zero, as this indicates that at least some of the selection is translated into selection on breeding value. The test of $\beta_1 = \beta_2$ is stricter, in that it assumes equal selection on both breeding value and environmental deviation. In particular, selection is strictly a function of phenotypic value, no matter how that phenotype is obtained (e.g., individuals with high breeding value vs. high environmental deviation with the same phenotype experience the same amount selection). For example, suppose individuals one and two both have phenotypic value $z = 100$, but individual one has $A = 80, E = 20$, while individual two arrives at this phenotypic value by $A = 10, E = 90$. If selection is entirely on phenotype, both individuals have the same expected fitness, but their expected fitnesses may differ if there is also selection on $A$ and/or $E$ in addition to any selection on $z$. Note that selection on a genetically correlated trait will also impose selection on $A$ in addition to any selection imposed on the focal trait.

Notice that this regression approach *critically* depends upon randomization of genotypes over environments. The estimated breeding value assigned to all members of a sibship

is their family effect, which is a function of the mean breeding value of their parents but also of maternal effects and common family environmental values. In the extreme case, if environments are not randomized, a common family environment could influence both the trait and fitness, and this would appear in the family effect. In this case, $\beta_1$ could be significantly different from zero, but this is a reflection of selection on common family environmental values (including maternal effects), not on breeding values.

Using this sib regression approach, Stinchcombe et al. (2002) and Scheiner et al. (2002) compared genotypic and phenotypic estimates of selection in six plant species grown on experimental plots (and hence stricter environmental control than expected in populations fully in nature). Even in these settings, these authors found that a significant fraction (around 25%) of the traits appeared to show an environmentally-induced bias ($\beta_1$ significantly different from $\beta_2$). While the bias rarely resulted in a change in sign, it often significantly impacted the magnitude of the estimated selection.

**BLUP/REML Estimates of Breeding Values**

Mixed-model approaches (BLUP/REML) provide a very powerful way to account for fixed effects and predict the vector of breeding values for individuals in our sample. They also are easily extented to account for additional fixed effects such as common family effects or the perminant environmental effects when repeated measures of traits occurs. As detailed in Chapter 15, use of the animal model

$$\mathbf{y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}\mathbf{a} + \mathbf{e} \tag{16.18a}$$

allows estimated breeding values (EBVs) to be assigned to each measured individual. Recall that $\boldsymbol{\beta}$ is the vector of fixed effects, $\mathbf{a}$ the vector of breeding values, and $\mathbf{e}$ the vector of residuals. $\mathbf{X}$ is the design matrix for the fixed effects, while the incidence matrix $\mathbf{Z}$ accounts for missing and/or duplicate records of measured individuals. $\mathbf{y}$ is an observed vector, while the matrices $\mathbf{X}$ and $\mathbf{Z}$ are known. The unknowns are the vector $\boldsymbol{\beta}$ of fixed effects while $\mathbf{a}$ and $\mathbf{e}$ are vectors of random effects. In order to predict these vectors of random effects, we first need to specify their covariance structure. The relationship matrix $\mathbf{A}$ accounts for the known pedigree and is the key to estimating breeding values (Chapter 15). Likewise, the standard assumption is made about the covariance structure of the residuals, $\sigma_e^2\,\mathbf{I}$. Thus, asuming multivariate normality, gives

$$\mathbf{a} \sim \text{MVN}(\mathbf{0}, \sigma_A^2\mathbf{A}), \qquad \mathbf{e} \sim \text{MVN}(\mathbf{0}, \sigma_e^2\,\mathbf{I}) \tag{16.18b}$$

as the final specification for the mixed model given by Equation 16.18a. While the matrix $\mathbf{A}$ (for now) is assumed known (given from the known pedigree of measured individuals), the variances $\sigma_A^2$ and $\sigma_e^2$ must be estimated, for which we use REML (LW Chapter 27).

Use of BLUP is a significant improvement over simply assigning all members of a family the same EBV, as different members of a sibship are assigned individual EBVs, as opposed to simply using a single value (the family mean) for all. For those individuals unlinked to any others in our pedigree, their EBVs are simply the estimated heritability times their phenotypic values, adjusted for fixed effects. For individuals who are linked (via pedigree information) to other individuals in our sample, BLUP uses this covariance information, in addition to their own phenotypic value, to obtain an improved estimate of their EBV. Although widely used in animal breeding since the 1970's, applications of the animal model in natural populations starts with Konigsberg and Chevered (1992) and Cheverud and Dittus (1992) who applied it to free-living primate populations. These papers went somewhat unnoticed, and a second wave of applications to ungulate mammals and nesting birds started in 1999 (Réale et al. 1999) and has been a rapid growth industry ever since. An interesting observation is that,

to date, most estimates of heritability based on mixed-model analysis of wild populations are lower than more traditional estimated based on parent-offspring regressions (Kruuk 2004). An interesting general feature is that, to date, most estimates of heritability based on mixed-model analysis of wild populations are lower than more traditional estimated based on parent-offspring regressions (Kruuk 2004) Part of this may arise from lack of control of maternal effects, as most parent-offspring regressions in the wild are mother-offspring, compounding direct and material effects. A second feature is that heritability is a mixed-model is the ratio of the additive variance to the sum of all the estimated variance components (Chapter 15). This later sum is not necessarily the phenotypic variance, as the variation introduced by fixed effects are removed by the mixed-model analysis (Wilson 2008). Thus parent-offspring vs. mixed-model $h^2$ estimates are looking at slightly different quantities. The main obstacle to implementing an animal model analysis of selection response is obtaining (or estimating) the relationship matrix $\mathbf{A}$ in a free-ranging organism.

With EBVs in hand, one can examine whether there is evidence of selection on breeding values, phenotypes, or both. In particular, suppose for individual $i$, one has their phenotypic value $z_i$, estimated breeding value $\widehat{A}_i$, and fitness $W_i$. From the Robertson-Price identity, selection on the phenotypes is measured by $S_z = \sigma(z_i, W_i)$, the covariance between trait value and fitness. Likewise, an estimate of selection on breeding values is given by $S_A = \sigma(\widehat{A}_i, W_i)$, the covariance between fitness and estimated breeding values. Most (of the few) studies using EBVs to look at the target of selection have simply asked whether $S_A$ is significantly different from zero, namely is there selection directly on breeding values. As detailed below, Postma (2006) developed a more formal test ($S_A = h^2 S_z$) to access whether the data are consistent with selection *only* on phenotypic value.

While mixed-model approaches are extremely powerful, they have the strong requirement of a known pedigree (and hence a known $\mathbf{A}$). For most natural populations, this is not possible. At present, use of the animal model is largely restricted to well-studied vertebrate populations in rather confined areas, allowing pedigree links to be ascertained. As introduced in Example 16.1, one such popualtion is the collared flycatcher birds (*Ficedula albicollis*) on the island of Gotland in Sweden (Pärt and Gustafsson 1989, Sheldon et al. 2003, and other references below). Since 1980, through the use of an extensive collection of nest-boxes, essentially all individuals have been marked and an extensive pedigree developed. Brood parasitism is unknown for this species, so maternity is determined with essentially no error. This population has around 15% extra-pair copulations (Sheldon and Ellegren 1999), creating some erroneous paternities (assigned to the male who visits the nest), but this is a very manageable number.

A final important point about applying mixed-models in natural populations is that common family and/or maternal effects must not be overlooked, as they are often important. It is important to have a sufficient number of *both* maternal and paternal connections in the pedigree to separate direct from maternal effects. Some species also show paternal care, such as the father helping with feeding at the nest. In such cases, a parental effect might also be considered. MacColl and Hatchwell (2003) used an animal model to show that paternal feeding rates in the long-tailed tit (*Aegithalos caudatus*) have a significant genetic component.

**Obtaining the Relationship Matrix**

The central difficulty in applying the animal model to free-living populations is obtaining the relationship matrix $\mathbf{A}$ for the measured sample of individuals. In natural populations, such pedigrees can be extremely difficult to access. There are two sources of information one can use. The first are **social pedigrees** based on field observations. If we observe a mother nursing an offspring, we have fairly high certainty that the offspring is from that mother. For example, to a first approximation, one can treat all of the eggs in a nest as the offspring

of the nesting female. Accessing paternity is more difficult. Again, field observations may be useful, for example which male visits the nest in pair-bonded birds, or which appears to be the dominant male in other social settings. Of course, none of these observations is foolproof. **Intraspecific brood parasitism** can occur where a female lays an egg in the nest of another female. Likewise, even with (apparently) pair-bonded birds, **extra-pair paternities** can occur. In some species, this can be upwards of 50%, so that the simple observation of a male helping at the nest does not imply they are the father. Similarly, determining dominant male in a harem is no guarantee that he is the father of all offspring in that harem. Because of this intrinsic bias towards determining the mother in some (but by no means all) wild populations, the pedigrees from such species often show an excess of **maternal linkages**. This has implications if maternal effects are suggested, as the pedigree must also have a sufficient number of **paternal linkages** to disentangle direct versus maternal effects (Clément et al. 2001, Kruuk 2004). A further complication is that pedigree errors can be high even in systems with apparently strong control over matings. For example, Visscher at al. (2002) estimated a sire error rate of around 10% for UK dairy cattle, despite very widespread use of artificial insemination. Recording errors and the biological vagaries of organisms should never be underestimated!

The second source of information is the use of molecular markers to assign relatedness. We can group these methods into two categories: those which are hypothesis-driven (e.g., paternity tests for candidate males or that two individuals are full sibs) and those that make no a prior assumptions about relatedness. Put another way, a focus on **relationships** (discrete classes such as parent-offspring) versus **relatedness** (estimates of the coefficient of coancestry). A number methods to estimate the pairwise relatedness have been proposed (reviewed by Ritland 2000, van de Casteele et al. 2001, Blouin 2003, Garant and Kruuk 2005, Tomas 2005, Csilléry et al. 2006, Frentiu et al. 2008, Pemberton 2008), and at first blush, one might think to simply use one of these to estimate the pairwise relatedness between all sets of pairs in our sample and use these as the elements of $\mathbf{A}$. There are numerous problems with this approach. First, there are high sampling variances with these estimates (see the above reviews). Second, such a procedure typically does not result in the matrix $\mathbf{R}$ used to estimate $\mathbf{A}$ being positive-definite (Frentiu et al. 2008), and hence is not a covariance matrix. There is also the issue of negative estimates of relatedness for unrelated individuals, which commonly occur. These are typically set to zero, but this introduces a bias which has yet to be formalized (akin to the bias introduced by setting negative variance estimates to zero).

A better approach seems to ignore more distant relationships that must be inferred solely by molecular markers and instead use molecular markers to confirm (or find) sets of close relatives, such as assigning parentage (Blouin 2003, Jones and Ardren 2003) or assigning individuals into sibships (Thomas and Hill 2000). Much of the power for obtaining EBVs come from data on close relatives, such as parent-offspring or sibs. Thus, much of the focus should be on detecting, and confirming, such close linkages.

One such critical link is in assigning paternities. As mentioned, there is a natural bias towards maternal connections, and without sufficient paternal connections direct and maternal effects cannot be disentangled. The consequences of miss-assigned paternities are a function of the pedigree structure and heritability. In animal breeding where there is a great excess of mothers (dams) over fathers (sires), the effects can substantial. In beef cattle, Lee and Pollak (1997) observed a significant reduction in the estimated heritability (0.1 versus the true value of 0.3) when 20% of the sires were miss-identified. Their pedigree structure had roughly 2% sires, 22% dames, and the rest were nonparents. In contrast, simulation studies by Charmantier and Réale (2005) with roughly equal percentage of sires and dams found that if the rate of extra-pair paternity was under 20%, then the biases in $h^2$ were modest (less than 15%). The bias introduced by miss-assigned paternities increases with $h^2$. Interestingly, Charmantier and Réale found that miss-assigned paternity does not always underestimate

$h^2$, but it can inflate it as well. Milner at al. (2000) working with Soay sheep (*Ovic aries*) found that variance estimates decreased between a pedigree with 95% confidence on a paternity versus one with 80% confidence. Finally, Keller et al. (2001), working with morphological traits in Darwin's finches (*Geospiza fortis*) on Isla Daphne Major in the Galápagos, found that not accounting for maternal effects introduced a much greater bias than did extra-pair matings. While incorrect pedigree links can certainly bias estimates of $h^2$, they have an even greater impact on estimates of individuals EBVs, which are key in accessing direct selection on breeding values. Pedigree errors typically result in heritability being underestimated, which in turn results in a shrinkage back towards the mean of the estimated breeding values (Geldermann et al. 1986, Israel and Weller 2000) so that (true) extreme breeding values are underestimated and low breeding values overestimated.

Henderson (1988) has suggested if a father is not known with certainty, it may be more efficient to include all possible sires (weighted by their probability) than to simply not include any sire-offspring linkages in the pedigree. He introduced the idea of an average numerator relationship matrix to accomplish this. Konigsberg and Cheverud (1992) applied this approach to estimate craniometric traits on a macaque colony on Cayo Santiagos. Here, mothers were known with certainty, but sires are unknown. However, field and social data can be used to exclude many males as possible sires, leading to a subset for each offspring. If there were $k$ possible sires, following exclusion, Konigsberg and Cheverud weighted them with equal probability $(1/k)$ and applied Henderson's method.

So, how does an investigator deal with all of this uncertainty? A nice approach was suggested by Morrissey et al. (2007), wherein the investigator first assumes a rough pedigree framework and then incorporates in the types of pedigree errors suspected given the biological system in question. Simulation studies can then be used to examine power (the ability to detect parameters) and sensitivity (how robust these estimates are in the face of errors). Morrissey et al. using a framework pedigree for Soay sheep (*Ovis aries*) found that the simple animal model (breeding values are the only random effect) was relatively robust to pedigree errors, but that when maternal effects were also included (as is typically for many studies), the results were much less robust. This is perhaps not surprising as separating maternal and direct effects is fairly sensitive to the types of links in the pedigree (Chapter 15). Quinn et al. (2006), using the pedigrees for two bird species, suggested a rough rule of thumb that at least three generations (years) and 100 individuals/year is required to estimate heritability with confidence.

In summary, although there appeals to be wealth of tools for using molecular markers to assign relationships, using them as the sole means to reconstruct $\mathbf{A}$ is suspect at best. Rather, a combination of using field observations to first suggest potential linkages and then the use of molecular markers to confirm these relationships should provided fairly reliable (albeit culled for more distant relatives) $\mathbf{A}$ matrices. If one is following a multigenerational study, these methods can provide links across generations and connecting these links over several generations can largely fill out the important entries in $\mathbf{A}$. A further caveat is that, as mentioned in Chapter 15, BLUP and REML methods can be compromised by previous selection, which is exactly what is expected in natural populations. Finally, again as mentioned in Chapter 15, Bayesian approaches should be used to fully account for all sources of variation. This has yet to be done, especially in cases when the elements of $\mathbf{A}$ are themselves estimated. Despite all of these issues, if one is trying to estimate a population feature, such as heritability, or a group measure, such as the average breeding value for a given generation, then the uncertainty in these approaches is somewhat moderated (although still needs to be accounted for). However, if the goal is to estimate *individual* breeding values for EBV-fitness correlations, then extreme care must be taken.

**Accuracy, Reliability, and Caveats with Using EBVs**

Postma (2006) discusses several caveats with using EBVs for tests of selection on breeding vs. environmental values. These issues revolve around the obvious fact that we are using an estimated value $\widehat{A}$ in place of the true breeding value $A$. As a starting point, we first need to consider several related measures of the uncertainty of the predicted breeding value, namely the accuracy, reliability, and prediction error variance. The **accuracy** $\rho$ of a EBV was introduced in Chapter 10 and is simply the correlation between the predicted $(\widehat{A})$ and actual $(A)$ breeding value. Recall (from Chapter 10) that the accuracy in using an invidual's phenotype to predict their breeding value is simply $h$, the square root of the heritability. Likewise, the **reliability** $\rho^2$ is the fraction of variation in breeding value accounted for by the EBVs. Thus, the reliability of just using an individual's phenotype to predict their breeding value is the heritability $h^2$. For an EBV from an animal model, the difference between the heritability and the reliability is a measure of how much additional insight any pedigree information supplies. Finally, recall that the **prediction error variance**, or **PEV**, (Chapter 15) is just $\sigma^2(\widehat{A} - A)$. Since breeding values are random effects, the variance in a EBV is the variance of the predicted value $\widehat{A}$ around the true value $A$. The smaller this variance, the better the prediction. These measures of uncertainty in EBVs are connected as followed. First, since $\sigma(\widehat{A}, A) = \sigma^2(\widehat{A})$, the PEV can be written as

$$\sigma^2(\widehat{A} - A) = \sigma^2(\widehat{A}) - 2\sigma(\widehat{A}, A) + \sigma^2(A) = \sigma^2(A) - \sigma^2(\widehat{A}) \tag{16.19a}$$

Second, recalling the definition of a correlation gives the accuracy as

$$\rho = \frac{\sigma(\widehat{A}, A)}{\sqrt{\sigma^2(\widehat{A})\sigma^2(A)}} = \sqrt{\frac{\sigma^2(\widehat{A})}{\sigma^2(A)}} \tag{16.19b}$$

Hence,

$$\sigma^2(\widehat{A}) = \rho^2\sigma^2(A) \tag{16.19c}$$

from which it immediately follows that $\sigma^2(\widehat{A}) \leq \sigma^2(A)$. Thus, the variance of the EBVs is less than the actual additive variance. Finally, from Equations 16.19a and 19c, the PEV for an EBV can be written as

$$\sigma^2(\widehat{A} - A) = \left(1 - \rho^2\right)\sigma^2(A) \tag{16.19d}$$

How do we extract the accuracy, reliability, and PEV from a specific EBV from an animal model? Recall that Equations 15.4d and 15.4e give the covariance matrix of the prediction error variances (PEVs) for each of the estimated breeding values. In particular, $PEV_{ii}$, the $i$-th diagonal element from the PEV matrix gives the prediction error variance for the EBV for individual $i$, which is just

$$PEV_{ii} = (1 - \rho_i^2)\sigma_A^2$$

assuming that $\sigma_A^2$ is known without error. Bayesian approaches (Chapter 15) can be used to account for this additional sort of error. Rearranging, we can find the predicted reliability for any particular EBV by

$$\rho_i^2 = 1 - PEV_{ii}/\sigma_A^2$$

The amount by which this exceed $h^2$ is a measure of the amount of information from the pedigree (beyond an individual's phenotype). Thus, individuals with a large number of relatives in the pedigree have higher reliabilities than individuals with no, or few, measured relatives. This is an important point, as in Chapter 15 our focus was using the *mean* EBV for all individuals within a generation to measure response, which tends to smooth out individuals differences. However, in direct tests of association between EBV and fitness,

individuals values are directly used, some of which contain more information than others. Further, values for relatives at typically correlated as well.

Two final identities concering EBVs are needed to complete our discussion. Postma (2006) shows that the prediction error variance is the covariance between the EBV and the environmental deviation $e$,

$$\text{PEV} = \sigma(\widehat{A}, e) \tag{16.20b}$$

Postma also shows that

$$\sigma(\widehat{A}, z) = \sigma(A, z) \tag{16.20c}$$

Due to their lower variance, EBVs resemble the phenotype more than do true breeding values. To see this consider the correlation between the true breeding value and phenotype versus that between the EBV and phenotype,

$$|\,\rho(\widehat{A}, z)\,| = \frac{|\,\sigma(\widehat{A}, z)\,|}{\sqrt{\sigma^2(\widehat{A})\,\sigma_z^2}} = \frac{|\,\sigma(A, z)\,|}{\sqrt{\sigma^2(\widehat{A})\,\sigma_z^2}} = \frac{|\,\sigma(A, z)\,|}{\rho\sqrt{\sigma^2(A)\,\sigma_z^2}} = \frac{|\,\rho(A, z)\,|}{\rho} \geq |\,\rho(A, z)\,|$$

Put another way, EBVs contain an element of the environmental deviation, the fraction given by $1-\rho^2$. Thus, when the reliability is low (close to $h^2$), the predicted breeding value is largely determined from an individual's phenotypic value and is thus potentially heavily influenced by the environmental deviation. Note that this is exactly what occurs when an individual is unlinked to any others in the pedigree and hence its phenotypic value is the sole predictor of its breeding value. As more links (and closer relatives) are added to the pedigree, $\rho^2$ increases, and the prediction error variance (and hence covariance between predicted breeding value and environmental value) decreases.

This has subtle implications for populations under selection. If an individual is lost before it leaves offspring, (i.e., its fitness is zero), it will have fewer links in the pedigree than individuals who survive to leave offspring. Thus individuals of low fitness have lower reliability than individuals with higher fitness, and the EBV for such low-fitness individuals are more influenced by environmental deviations than are higher-fitness individuals. Obviously, this can bias estimates of the amount of selection on breeding value.

Applying the above ideas, Postma points out a significant problem with the regression test suggested by Equation 16.17 when using estimated breeding values (EBVs) in place of true breeding values. In particular, the selection gradients are different for true and predicted breeding values. First, for true breeding values,

$$\beta_A = \frac{\sigma(W, A)}{\sigma^2(A)} = \frac{h^2\sigma(w, z)}{h^2\sigma^2(z)} = \beta_z \tag{16.21a}$$

so that the gradient using true breeding values equals the gradient using phenotypic value, as noted by Rausher (1992). However, when using predicted breeding values,

$$\beta_{\widehat{A}} = \frac{\sigma(W, \widehat{A})}{\sigma^2(\widehat{A})} = \frac{h^2\sigma(w, z)}{\rho^2 h^2\sigma^2(z)} = \frac{\beta_z}{\rho^2} \tag{16.21b}$$

If $\rho^2 \simeq 1$, gradients using phenotypic and EBVs should be very similar (in the absence of environmental factors influencing both the trait and fitness). However, if $\rho^2 < 1$, $\beta_A < \beta_{\widehat{A}}$ and gradients based on predicted breeding values overestimate the gradient expected for true breeding values, which in turn compromises Rausher's $\beta_1 = \beta_2$ test (Equation 16.7).

To circumvent this problem, Postma suggests comparing covariances (selection differentials) rather than gradients. If fitness is entirely determined by the trait value, then the

selection differential on the phenotype has a simple relationship with the selection differential on breeding value,

$$S_A = h^2 S_z \tag{16.22}$$

Noting that we can express the breeding value of an individual with phenotypic value $z$ as $A = h^2(z - \mu) + e$, where $e$ is the deviation about this expected value, Equation 16.20 follows follows since $S_A = \sigma(A, w) = \sigma(h^2(z - \mu) + e, w) = h^2\sigma(z, w) = h^2 S_z$. Likewise, if selection is entirely on phenotypic value, then $\sigma(A, w) = \sigma(\widehat{A}, w)$. Thus, **Postma's test** is to compare the ratios of the selection differentials using breeding versus phenotypic values, which should equal the heritability in the absence of environmentally-induced covariances between the trait value and fitness.

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**Example 16.4**. Gienapp et al. (2006) examined breeding times in a Dutch population of great tits (*Parus major*) followed over a thirty year period (1973 - 2003). In six of the 31 years of the study, there was phenotypic selection for later breeding, so they focused on the 25 year subset of the data which showed phenotypic selection for earlier breeding. Estimated breeding values (EBVs) for breeding date were obtained using the animal model and the pedigree of this population. The covariance between EBVs and fitness was $S_A = -0.24 \pm 0.08$, while the estimate of the heritability times the phenotypic selection differential was $S_z \cdot h^2 = -.029 \pm 0.05$. Thus, breeding date in this study satisfied Postma's criteria (Equation 16.22) for selection on the phenotype, as a opposed to an environmental, value. The annual mean date of breeding advanced over this thirty year period by 5.4 days, which was not significant. The predicted evolutionary response was for a decrease of 1.5 days over this thirty year period, a value too small to be detected. Hence, they conclude that the apparent stasis was not due to an environmental factor influencing both breeding date and fitness, but rather to statistical power, with the sample sizes not being large enough to detect such a small difference given the noisy sampling.

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**Standarized vs. Unstandarized Gradients**

One final comment is in order. When one wishes to compare selection on different characters, we typically first standardize those traits to give them mean zero and unit variance, and then compare the resulting standardized selection gradients, $\beta'$. We standardized (the variance) by using the transformation $x' = x/\sigma_x$. For a single trait,

$$\beta'_x = \beta_{x'} = \frac{\sigma(x', w)}{\sigma^2(x')} = \frac{\sigma(x/\sigma_x, w)}{1} = \frac{\sigma(x, w)}{\sigma_x} = \frac{S_x}{\sigma_x} = \sigma_x \beta_x$$

Using unstandarized variables, $\beta_z = \beta_A$ if there is direct selection on the phenotype. However, as noted by Stinchcombe et al. (2002), if one first standardizes the variables, this is no longer true. Here $\beta'_z = \sigma_z \beta_z$, while

$$\beta'_A = \sigma_A \beta_A = \sigma_A \beta_z = \frac{\sigma_A}{\sigma_z}\, \beta'_z = h\, \beta'_z \tag{16.23a}$$

Likewise, the standardized gradient using EBVs is (Postma 2006)

$$\beta'_{\widehat{A}} = \sigma_{\widehat{A}} \beta_{\widehat{A}} = \frac{h}{\rho}\, \beta'_z \tag{16.23b}$$

Thus, one must apply these corrections if we wish to used standardized comparisons across different traits.

## APPARENT FAILURES OF RESPONSE IN NATURAL POPULATIONS

Merilä et al. (2001c), in a detailed review of the long-studied populations of mammals and birds, noted a number of cases where (i) there was a consistent selection differential on a particular trait, (ii) that trait was heritable, and yet (iii) no response (or worse, response in the opposite direction) was observed over a lengthy period (many generations) of study (Table 16.1). While there are several classic examples of natural populations responding to either imposed artificial selection (Example 16.1, Semlitsch and Wilbur 1989), a new environmental challenge (such as a habit shift, major weather event, or introduction of a novel selective agent, e.g., Losos et al. 1997; Reznick et al. 1997), or even the introduction of a new competitor species (Grant and Grant 2006), Merilä et al. lamented the apparent lack of response outside of these situations. Indeed, they raise the central question as to the basis for this apparent **statsis** in response in the face of apparent selection.

**Table 16.1**. Examples of natural populations of mammals and birds in which apparent strong directional selection on a heritable trait fails to show response. Length is the length of the study (in years). After Merilä et al. (2001c)

| Species/Trait | $h^2$ | $\lvert\bar{i}\rvert$ | Observed Response | Lenght | Ref |
|---|---|---|---|---|---|
| **Mammals** | | | | | |
| *Cervus elaphus* (Red deeer) | | | | | |
| Antler mass | 0.36 | 0.46 | Opposite response | 29 | Kruuk et al. (2000, 2002) |
| Birth mass (Male) | 0.11 | 0.40 | No change | | |
| Birth mass (Female) | 0.11 | 0.40 | No change | | |
| *Ovis aries* (Soay sheep) | | | | | |
| Body mass (Male) | 0.12 | 0.11 | No change | 12 | Milner et al. (1999, 2000) |
| Body mass (Female) | 0.24 | 0.07 | No change | | |
| **Birds** | | | | | |
| *Branta leucopsis* (Barnacle Goose) | | | | | |
| Tarsus lenght (M) | 0.53 | 0.03 | Opposite response | 13 | Larsson et al. (1998) |
| Tarsus lenght (F) | | 0.09 | Opposite response | | |
| *Anser caerulescens* (Snow Goose) | | | | | |
| Clutch size | 0.20 | 0.22 | Opposite response | 20 | Cooke et al. (1990) |
| *Ficedula albicollis* (Collared Flycatcher) | | | | | |
| Relative mass | 0.30 | 0.23 | Opposite response | 17 | Merilä et al. (2001a, b) |
| Tarsus lenght | 0.52 | 0.12 | No change | 4 | Alatalo et al. (1990) |
| Tarsus lenght | 0.35 | 0.18 | No change | 17 | Kruuk et al. (2001) |
| Breeding time | 0.19 | 0.31 | No change | 19 | Sheldon et al. (2003) |
| *Parus major* (Great Tit) | | | | | |
| Breeding time | 0.50 | 0.46 | No change | 23 | Boyce and Perrins (1987) |
| Breeding time | 0.17 | 0.21 | No change | 30 | Perrins and Jones (1974) |

The explanation for apparent statis in some cases may as trival as lack of sufficient power to detect the change (Example 16.4). Fisher's model (an unmeasured environmental factor correlated with fitness and trait value) provides one explanation for stasis. Another is a lack of a consistent selection differential. For example, the selection differential may changing sign over generations (e.g., Example 16.2; Grant and Grant 2002), resulting in a net long-term

selection differential of close to zero. Immigration and differential dispersal can also mask a local selection response, or it can enhance it (Garant et al. 2005).

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**Example 16.5**. One popular suggestion for stasis is the presence of within-generation **trade-offs**, wherein a trait favored during one part of the life cycle (say viability) is selected for, while it is selected against in another (say fecundity), resulting in no significant net selection (Chapter 30). Sheldon et al. (2003) present an interesting example from our old friend, the collared flycatcher (*Ficedula albicollis*). EBVs for breeding date were obtained and their correlation with two components of fitness examined. There was a significant negative covariance between EBVs for breeding data and fecundity selection. However, when fitness was measured by viability (adult female survival), the covariance fluctuated in sign between years. Thus, while there was direct selection on the breeding value for breeding date, it was partial constrained by these life-history tradeoffs. The authors predict the year-to-year change in EBVs is given by

$$\Delta EBV = \beta_f \pi/2 + \beta_v(1-\pi)$$

where $\beta_f$ and $\beta_v$ are the gradients with respect to fecundity and viability selection and $\pi$ is the fraction of yearling females in the subsequent year. The correlation between predicted and observed changes in EBVs from year to year was significant ($r = 0.66, p = 0.002$) but the direction of select constantly changed, resulting in very little *net* change in EBVs.

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While any of the above suggestions (or perhaps a combination of them) might indeed account for stasis in a given trait, analysis of some of the better studied cases often suggest more complex and complicated reasons for lack of a response.

### Cryptic Evolution: Genetic Change Masked by Environmental Change

One explanation for stasis is that change in the environment can dilute, and indeed even swamp, underlying genetic change. In the extreme, one can have **cryptic evolution** – significant genetic change that does not show up as phenotypic change because it is countered by environmental change. Conover and Schultz (1995) coin the phrase **counter-gradient variation** for situations in which the environmental trend is opposite to the direction of selection. Interestingly, such situations may actually increase the strength of a selection on a trait, as the population struggles to keep pace with the declining environment. In the extreme, a population faces the risk of extinction if the environment is deteriorating at a faster rate than compensating traits can evolve.

One striking example of cryptic evolution is the study by Merilä et al. (2001b) in the Gotland population of collared flycatchers. These authors examined an index of body condition (a measure of relative body weight) at **fledging** (time of leaving the nest). Formally, the condition of a bird is often taken as the nonskeletal component of body weight, and this is often assumed (as did Fisher) to be largely controlled by environmental factors. Merilä et al. defined their index of condition based on the residuals of the regression of body mass on tarsus (leg) length, the later being a surrogate measure of the skeletal component. Merilä et al. (2001a) showed that, despite the widespread view to the contrary, that condition does indeed have substantial heritable genetic variation (estimated $h^2$ = 0.30). Despite the heritable nature of this trait coupled with constant positive selection (an average selection intensity of 0.23 on this index, so that survivors are, on average, 0.23 standard deviations above the mean before selection) condition has *declined* over time in this population (Figure 16.4). The

regression of mean condition from 1981 to 1999 had a significant negative slope of $b = -0.036$ /year.

As mentioned above, there is extensive pedigree data on this population and breeding values for individuals have been estimated using BLUP/REML methodology. With estimated breeding values (EBVs) in hand, Merilä et al. were able to show that selection for condition occurs directly on the breeding values, so that the covariance between breeding value for condition and fitness is non-zero. As shown in Figure 16.4, the regression of breeding values on time does indeed show a significant positive slope ($b$ = 0.0022) so that the population is indeed showing genetic improvement, despite the mean phenotype declining over time. Thus, the environmental component of condition has been declining over time, and at a rate faster than the genetic improvement, resulting in a net phenotypic *decline* over time. Merilä et al. (2001b) suggest that this decline is likely attributable to reductions in the caterpillar food supply due to large-scale climatic trends.

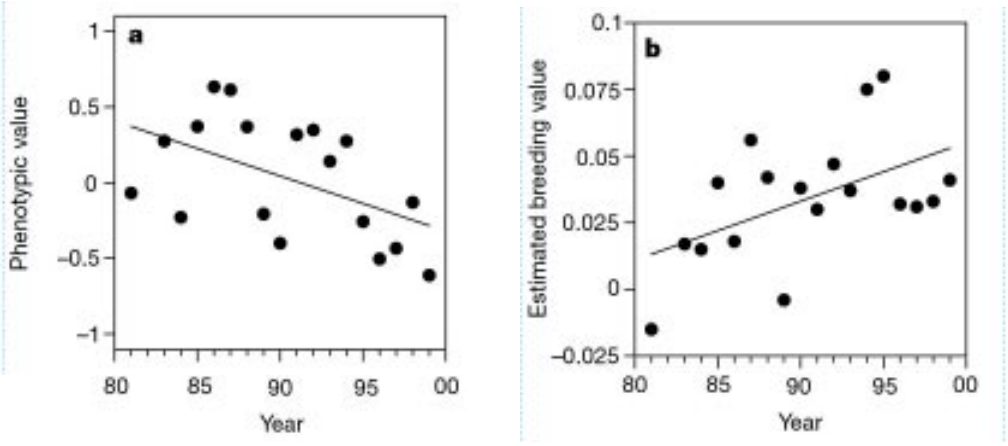


**Figure 16.4**. Body condition at fledgling for a Gotland population of collared flycatchers between 1980 and 1999. **Left:** Mean phenotype. **Right:** Mean breeding value (the average of the EBVs). After Merilä et al. 2001b.

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**Example 16.6**. A second example of a negative environmental trend was offered by Larsson et al. (1998), who examined body size in the Barnacle Goose (*Branta leucopsis*). The natural colonization of the Baltic area of Sweden of this normally Arctic species started with a single breeding pair in 1971, followed by subsequent rapid increase in the population size. Larsson et al. studied the two largest Baltic colonies from 1984 to 1996. Head size and tarsus length were measured to extract a structural body size index, and larger females were found to have larger, and earlier, clutches (with larger eggs, resulting in more and heavier young than smaller females). Further, juvenile body mass was significantly correlated with postfledging survival. Both size measures are highly heritable, but average body size *declined* over the 13 year study period (by 0.7 and 0.5 standard deviations for head and tarsus length, respectively). The authors conclude (from a variety of evidence) that the environment had declined due to the growth of the colony. For example, birds from the smaller (and younger) colony studied were initially around a standard deviation larger than birds from the larger (and older) colony. Thus in this case the declining environment seems to be density-dependent effects on individual growth brought on by the overall success (population growth) of the colony itself.

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As Example 16.6 highlights, as organisms evolve they necessarily change at least part of their environment. Indeed, van Valen's (1973) **Red Queen hypothesis** states that organisms have to evolve just to stay where they are relative to the evolving biosphere around them. (The Red Queen, introduced in Lewis Carroll 's *Through the Looking Glass*, had to run just to stay in place.) Cooke et al. (1990) suggest that the lack of response to selection on clutch size in birds may have a red-queen style of explanation. Motivated by Fisher's arguments for lack of response on egg laying date, Price and Liou (1989) suggested that selection for clutch size was largely on nutritional state, which they presumed was largely environment with no significant heritable component. Cooke et al. agree that a non-heritable environmental component may be present, but further suggest that birds with the better quality territories have larger clutches and higher fitness, so that a component of selection for clutch size is selection to compete for territories. Although competitive ability may increase over time, average territory quality, and hence average clutch size, remains relatively constant, and hence no response is seen. Under their model, if one could hypothetically compete ancestral and current populations for territories, current individuals, possessing higher breeding values for territorial competitiveness, would win.

**Tarsus Size in Collared Flycatchers: Selection on E or EBVs?**

The saga of lack of response in tarsus size in flycatchers, started in Example 16.1, offers an interesting cautionary tale. Recall that Alatalo et al. (1990) puzzled over why the highly heritable tarsus length ($h^2 = 0.52$), which showed an average selection intensity (using juvenile survival) of 0.12 over a four year study period from 1981 to 1985, did not show a response to selection. They reasoned that this lack of response was due to selection on a non-heritable environmental component, likely nutritional state. Individuals with a higher nutritional state grow faster (and hence have larger tarsus lengths) as well as higher fitness due to increased nutritional value (i.e., better condition). To test this hypothesis, Alatalo et al. considered fledging weight (13 day weight) as a more sensitive measure of condition than tarsus length. Further, since parent-offspring regressions of fledging weight gave very low estimates of heritability that were not significantly different from zero ($h^2 = 0.072 \pm 0.082$), they assumed that this trait was largely environmental and hence a good indicator of the nonheritable nutritional state. As discussed in Example 16.1, when selection on fledging weight was jointly analyzed with tarsus length, the authors found direct selection on weight, but no evidence selection on tarsus length. Since both traits a phenotypically correlated, direct selection on weight induces a correlated selection different on tarsus length. Alatalo et al. took this as evidence in favor of selection on a nonheritable environmental trait, weight, that was correlated with both the focal trait (tarsus length) and fitness. One problem with this analysis is that Merilä et al. (2001a) later showed that condition does indeed have a significant heritable component, and hence it is not clear if selection is acting only on the environmental component of condition.

Kruuk et al. (2001), using a more extensive data set on the same population, came to very different conclusions from Alatalo et al. Using pedigree data on over 20,000 measured individuals over an 18 year study, they used BLUP/REML to obtain both estimates of heritability for fledgling tarsus length as well as estimates for the breeding values of tarsus length. They found tarsus length to be significantly heritable (although their estimated heritability was $h^2 = 0.35$, lower than the parent-offspring estimates of Alatalo) and also under selection, with a selection intensity of 0.18. However, their EBVs allowed then to look for selection directly on the breeding values of tarsus length, which would generate a non-zero covariance between the EBVs of tarsus length and fitness. The selection differentials on breeding values were indeed positive and significant, although not as large as the selection differentials based on phenotypic value. Thus, both breeding value and environmental value are under selection, and the suggestion that tarsus length does not response because of a lack of selection

on its heritable component is not substantiated by this study. Kruuk et al. were perplexed as to possible causes for lack of response. Their experimental design had the power to detect the predicted change. Further, they did not find evidence of either fluctuating selection differentials (the vast majority were positive over the study period) nor did they find apparent fitness tradeoffs.

**Antler Size in Red Deer**

Free-living red deer (*Cervus elaphus*) on the Isle of Rum in Scotland are another well-studied natural population with a largely complete pedigree spanning several decades. Males fight to compete for mates, suggesting antler size as a potential trait under selection. Males shed antlers in the early spring, and given that antler shape is very individual-specific, cast antlers found in the field can easily be assigned back to a specific stag. Growth of new antler continues until late summer. Kruuk et al. (2002) examined antler size, finding that males with larger antlers had increased lifetime breeding success (total number of offspring), with a selection differential of $S = 0.445 \pm 0.094$ (scaled in phenotypic standard deviations). While body size (measured by leg length) also had an effect on lifetime breeding success, antler size still had a significant effect on fitness even after accounting for body size, with a standardized selection gradient of $\beta' = 0.44 \pm 0.18$. Antler size (measured as the mass of the annually-shed antlers) was heritable ($h^2 = 0.329 \pm 0.12$), and the breeders' equation would suggest a response of $R = h^2 S = 0.329 \cdot 0.445 = 0.146$ standard deviations per generation. Given a generation time of roughly 8 years and a standard deviation of 163 grams for antler mass, this suggests an expected change of roughly 2.3 grams/year. However, the average mass of antlers *declined* by 6.7 grams per year. One apparent reason for the decline was an environmental change due to increased population density over the study period, with antler size decreasing with increasing density.

Was this also a case of genetic change being masked by this environmental change? Apparently not. These authors could find no correlation between a males predicted breeding value (EBV) for antler mass and his lifetime breeding success. However, the environmental component of phenotype (phenotype - EBV) showed a strong positive covariance with lifetime breeding success. Thus, this appears to be an example of selection on an environmental component. The authors suggest that male fighting ability is, at least in part, a function of the nutritional condition of a male, and males with better nutritional value may be both better fighters and also grow larger antlers. Being better fighters, they have a greater lifetime reproductive success as well as having higher antler mass. As a final test, the authors examined the genetic correlation between lifetime breeding success and antler size (the correlation between the EBV for both traits in an individual) and found it to be non-significant, $-0.254 \pm 0.289$

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**Example 16.7**. An interesting counterexample to lack of response in antler size in red deer is a study by Coltman et al. (2003) on the impact of trophy hunting on horn size in bighorn rams (*Ovis canadensis*). Trophy hunters prefer large males with large horns, with the estimated heritability for horn size being $0.69 \pm 0.10$. A 30 year study at the aptly-named Ram Mountain (Alberta, Canada) had 57 rams shot since 1975, about 40% of those legally available for harvest each year. Partial pedigree data is available for this population, allowing BLUP to provide EBVs for horn size. The authors found that hunters selectively harvested rams with high EBVs for horn length. Much of horn growth occurs between ages two and four, while much of the effect of horn size on mating success occurs after age six. The net result is that rams with high breeding value for horn size are harvested before they can contribute significant offspring, with the authors finding a negative covariance between EBV for horn length and lifetime mating success in the selected population. As a result,

mean horn length significantly decreased over the course of the study, around 0.15 standard deviations per generation. Likewise, EBVs for horn length also significantly deceased over this period. Thus, horn size showed a genetic response to selection. The difference between this example and red deer antler size, both of which are heritable, is that the selective agent (hunting for horn size) is clear for rams, while the selective agent acting on antler size in red deer is less clear. For this comparison, it is not free-living organisms per se that cause issues with the breeders' equation, but rather lack of a clear understanding of the target(s) of selection (no pun intended).

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**Changes in Heritability/Selection Over Environments**

A major theme throughout this chapter is that, in natural populations, the lack of control over environmental factors can severely comprise the breeders' equation. We have already seen that selection on an environmental value influencing both our focal trait and fitness compromises the breeders' equation, and likewise how an environmental trend can mask a genetic trend. Moreover, changes in the environment can do more than simply mask a genetic trend. The nature of selection can easily change as the environment (biotic and abiotic) changes. For example, if the environment randomly switches we might have a situation where a trait is favored to increase roughly half the time and decrease roughly the other half. The resulting cumulative result for (potentially) strong selection each generation is no net selection differential. A more subtle implication of environmental change arises when significant genotype-environment interaction is present. As the environment changes, the heritabilities can change, either due to changes in the environmental and/or the genetic variances (Hoffmann and Parsons 1997a,b; Hoffmann and Merilä 1999).

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**Example 16.8**. Charmantier et al. (2004) examined chick tarsus length and body mass at fledging in a population of blue tits (*Parus caeruleus*) in three French populations, two on the island of Corsica and a third on the mainland. Their study followed roughly 8000 banded chicks from roughly 1200 individual broods representing three different habitats, which the authors were able to rank in quality. They found that poorer habitats showed weak selection to increase tarsus length and strong selection to increase body mass, while in good habitats there was no significant selection on either trait. Interestingly, heritability for body mass increased with habitat quality, with the lowest heritability occurring in the habitats experiencing the strongest selection for increased body weight. Thus in such low quality environments strong selection would be at least partly countered by lower heritabilities, leading to a weaker response.

A similar situation was observed by Wilson et al. (2006) for birthweight Soay Sheep (*Ovis aries*). These authors used a random regression animal model (Chapter 38), which allowed the estimation of maternal performance over a continuous environmental variable (here the population-wide neonatal mortality for a given year). Harsh environmental conditions generated strong selection for higher birthweights but also result in low genetic variance in this trait. More benign environments resulted in weaker selection but higher brithweight heritability.

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A final interest example is the study on breeding time in the mute swan (*Cygnus olor*) by Charmantier et al. (2006). In a 25-year study of a swan colony in Abbotsbury on the south coast of England, the authors showed that selection does indeed act on EBVs for clutch size,

and an observed change of around 0.4 standard deviations occurred during the course of the study. While this is a fairly dramatic example of an observed change on a selected trait, the authors note that a supplemental feeding program for the colony started around the time of the experiment, and this may have resulted in an environmental shift (higher nutrition) that allowed for the selection response.

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