15

Short-term Changes in the Mean: 3. Permanent versus Transient Response

The phenotypic gains from selecting for epistatic differences come from distorting the gametic array and soon disappear after selection is relaxed, as the gametic array returns to random. By contrast, the gains from changes in gene frequency are permanent. — Lush (1948)

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While the basic breeder's equation is the foundation for much of the theory of selection response in quantitative traits, its elegant form $R = h^2 S$ arises because of certain simplifying assumptions (Table 13.2). One interesting complication is that the response can have both transient and permanent components, and this chapter examines common situations where this can occur (also see Chapter 23). Selection can change allele and genotype frequencies, generate linkage disequilibrium, and create nonrandom association among environmental values, all of which can contribute to response. Of these, only allele frequency changes are permanent, as any change remains in place after selection stops and random mating occurs (the Hardy-Weinberg theorem). In contrast, random mating eventually ensures Hardy-Weinberg frequencies for genotypes, linkage equilibrium, and random association of environmental effects. The contributions to response due to departures from these equilibrium values are therefore *transient*, decaying to zero following the cessation of selection.

Transient components of response can be both positive and negative, so that response may decay following selection or it can actually increase. In the extreme situation, a **reversed response** occurs, wherein a trait selected to increase actually *decreases* due to a negative transient component overwhelming a positive permanent response. The potential of transient contributions from previous generations of selection are one reason for the caveat of the breeder's equation requiring an unselected base population.

Our treatment starts with a brief discussion of why the breeder's equation focuses on h^2 even though other factors can also contribute to response. We then turn to two genetic situations that give transient components to response: additive epistasis in diploids and dominance in tetraploids. This leads to a discussion of the method of ancestral regressions to determine the conditions under which a component of response is transient. The implications of correlated environment effects between relatives are considered next, examining response when the parent-offspring covariance is entirely due to shared environmental effects. We conclude by considering the complications introduced by the presence of heritable maternal effects, which show some of the properties seen with shared environmental values.

WHY ALL THE FOCUS ON h^2?

As was discussed in LW Chapters 7 and 17, epistasis and/or environmental effects shared

between parents and their offspring can cause the parent-offspring regression slope to deviate significantly from $h^2/2$, altering the response from that predicted by the breeder's equation. With epistasis and correlation between parental and offspring environmental values, the single parent-offspring slope becomes

$$b_{op} = \frac{\sigma(z_p, z_o)}{\sigma_z^2} = \frac{1}{\sigma_z^2}\left(\frac{\sigma_A^2}{2} + \frac{\sigma_{AA}^2}{4} + \frac{\sigma_{AAA}^2}{8} + \frac{\sigma_{AAAA}^2}{16} + \cdots + \sigma(E_p, E_o)\right) \tag{15.1a}$$

Assuming a linear biparental regression with identical slopes for both parents, the response to a single generation of selection becomes

$$R = 2b_{op}S = h^2\, S + \frac{S}{\sigma_z^2}\left(\frac{\sigma_{AA}^2}{2} + \frac{\sigma_{AAA}^2}{4} + \frac{\sigma_{AAAA}^2}{8} + \cdots + \sigma(E_{fa}, E_o) + \sigma(E_{mo}, E_o)\right) \tag{15.1b}$$

which can deviate significantly from $h^2\, S$. Why then is so much attention focused on h^2?

The reason is that we are interested in the **permanent response** to selection. Selection changes allele frequencies, which changes the mean genotypic value. Under the infinitesimal model, very small changes in allele frequencies over a large number of loci can generate a significant change in the mean without much of change in the additive variance (Chapter 24), and these changes are reflected in the component $h^2 S$ of the response. Any additional response from epistasis and/or shared environmental arise because of gametic-phase disequilibrium and/or nonrandom associations of environmental values. Recombination and randomization of environmental effects causes these associations to decay to zero under random mating. Conversely, changes in allele frequencies are permanent, as once selection stops, Hardy-Weinberg implies the new allele frequencies are stable. Hence, as will be shown shortly, the permanent response under the conditions leading to Equation 15.1b is $h^2\, S$. One exception, discussed in Chapter 23, is when significant inbreeding occurs. In this case, σ_{AA}^2 and other non-additive variance components (σ_{DI}^2, σ_{ADI}) introduced in Chapter 12 can contribute to permanent response.

GENETIC SOURCES OF TRANSIENT RESPONSE

Diploid parents undergoing sexual reproduction pass along single alleles at each locus to their offspring, with a parent's breeding value being the sum of allelic effects over all loci. Genotypic values are generally different from breeding values, with deviations representing interaction terms, such as dominance (between alleles at the same loci) or epistasis (between different loci). Such interactions are not typically passed to an offspring from a randomly-mating parent, as their transmission requires that a parent passes along all of the component parts of the interaction (such as both alleles at a locus for dominance). As such, only part of an exceptional genotypic value of a parent is passed onto the offspring. However, there are two common settings where at least some of these interaction terms can be passed to an offspring under random mating — additive epistasis in a diploid and dominance in a tetraploid. When nonrandom mating occurs, such as inbreeding, then the possibility exists that other interactions can be passed onto a offspring (such as dominance in a diploid), see Chapter 23.

Additive Epistasis

Epistasis involves interactions between either single alleles at different loci (additive epistasis, $A \times A$) or entire genotypes at different loci (dominance epistasis, $D \times D$). These

interactions result in a different trait value than predicted from a simple summation of all single-locus effects, and thus can contribute to response. Since a random-mating diploid passes along a single allele at each locus to its offspring, additive ($A \times A$, $A \times A \times A$, etc.) interactions between alleles at different loci can be passed from a parent to its offspring. Under random mating, any epistatic term involving dominance ($A \times D$, $D \times D$, etc.) cannot be passed along, as a parent must pass along *both* alleles at a given locus to its offspring to transmit a D component. Finally, it is important to remind the reader that even when significant epistasis in genotypic values is present, much of this is already loaded into the additive variance (which is the variance accounted for by the best linear fit of a potentially highly nonlinear system) and thus accounted for by the breeder's equation (Crow 2010). Under fairly general conditions, Hill et al. (2008) show that extreme allele frequencies (near zero or one), as would be expected under drift (Chapter 2), result in most of the genetic variance being additive even when the genotypic values are highly nonadditive. Our concern here is how any residual variance (e.g., σ^2_{AA}) not loaded onto the additive variance influences response.

The response when additive $\times$ additive epistatic variance is present was examined by Griffing (1960a,b) for the infinitesimal model, although Lush (1948) and Kempthorne (1957) clearly grasped the key idea of response from additive epistasis being transient. Under the assumptions that phenotypes are normally distributed, effects at any particular locus are very small relative to the total phenotypic variation, and no third (or higher) order additive epistasis is present, the response to one generation of selection is

$$R = S\left(h^2 + \frac{\sigma^2_{AA}}{2\sigma^2_z}\right) \tag{15.2}$$

One might expect that $R(t)$, the cumulative response after t generations of selection, is simply t times the result given by Equation 15.2. However, any increased response due to epistasis is only temporary, reflecting gametic-phase disequilibrium generated by selection. As disequilibrium decays under recombination, so does the component of response due to epistasis. This occurs because the $A \times A$ contribution is due to favorable *combinations* of alleles at different loci, above and beyond their individual contributions (which are accounted for by changes in the mean breeding value of the selected parents, $h^2 S$). Recombination breaks down these combinations, removing the epistatic contribution. Griffing showed that for two linked loci separated by recombination fraction c, the response when a generation of selection is followed by τ generations of random mating is

$$S\left(h^2 + (1-c)^\tau \frac{\sigma^2_{AA}}{2\sigma^2_z}\right) \tag{15.3}$$

which converges to $h^2\, S$. Equation 15.3 follows by noting that the probability a gamete containing specific alleles from both loci remains intact following one generation of recombination is $1 - c$. Thus, after τ generations only $(1-c)^\tau$ of the favorable two-locus combinations selected at $\tau = 0$ remain unaltered by recombination.

Summing Equation 15.3 over t gives the cumulative response after t generations with constant selection differential S as

$$R(t) = t\, h^2\, S + R_{AA}(t) \tag{15.4a}$$

where

$$R_{AA}(t) = S\,\frac{\sigma^2_{AA}}{2\sigma^2_z}\left(\sum_{i=1}^{t}(1-c)^{i-1}\right) = S\left(\frac{1-(1-c)^t}{c}\right)\left(\frac{\sigma^2_{AA}}{2\,\sigma^2_z}\right) \tag{15.4b}$$

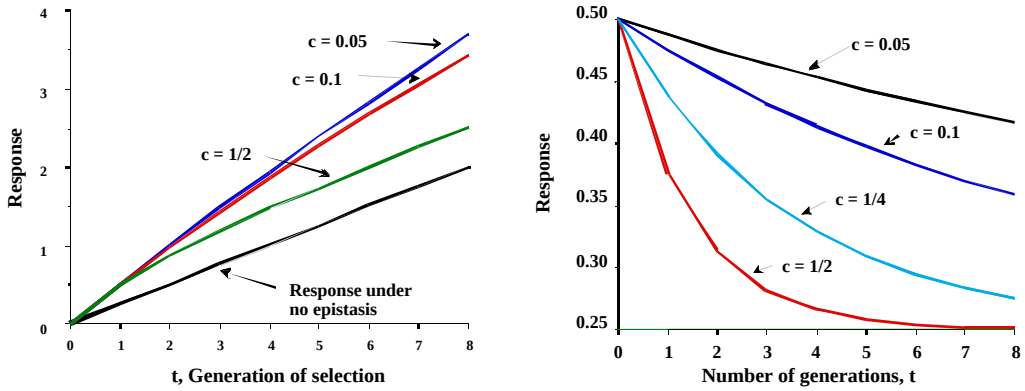

Figure 15.1. The permanent and transient response to selection (scaled in units of S) assuming pairwise epistasis in a diploid, with $h^2 = 1/4$ and $\sigma^2_{AA}/\sigma^2_z = 1/2$. **Left**: The cumulative response assuming a constant amount of selection for various values of c. Note that even with this large amount of epistasis (σ^2_{AA} accounting for half the total variance), it is difficult to distinguish the curvilinear response with epistasis from a linear response. **Right**: The decay of the response to a single generation of selection due to the decay of the contribution from epistasis. Provided $c > 0$, the cumulative response eventually decays to $h^2 S = S/4$, the expectation under no epistasis.

denotes the cumulative additive $\times$ additive epistatic contribution. The last equality follows using the partial sum of a geometric series,

$$\sum_{i=0}^{n} x^i = \frac{1 - x^{n+1}}{1 - x} \tag{15.5a}$$

We will also shortly make use of the related identity,

$$\sum_{i=1}^{n} x^i = \frac{1 - x^{n+1}}{1 - x} - 1 = \frac{x - x^{n+1}}{1 - x} \tag{15.5b}$$

If loci are unlinked ($c = 1/2$), $R_{AA}(t)$ rapidly converges to $S\,\sigma^2_{AA}/\sigma^2_z$, while if loci are completely linked ($c = 0$), $R_{AA}(t) = t\,S\,\sigma^2_{AA}/(2\sigma^2_z)$. Provided $c > 0$, the total epistatic contribution approaches a limiting value of

$$\widetilde{R}_{AA} = \lim_{t\to\infty} R_{AA}(t) = \frac{1}{c}\left(S\,\frac{\sigma^2_{AA}}{2\,\sigma^2_z}\right) \tag{15.6a}$$

$\widetilde{R}_{AA}$ represents the balance between selection generating disequilibrium and recombination removing it. Equation 15.6a shows that the limiting contribution is $1/c$ times the epistatic response in the first generation. For unlinked loci, this is just twice the initial response. With tight linkage, the total response can be significantly larger. Solving for t in $1-(1-c)^t = 1/2$ gives

$$t_{1/2} = \frac{-\ln(2)}{\ln(1-c)} \tag{15.6b}$$

as the time (in generations) for half the total response to occur. For small c, this is approximately $\simeq 0.68/c$. As linkage becomes tighter, the total cumulative epistatic response increases, as does the time for half of this total response to occur (Figure 15.1).

Once selection stops, the epistatic contribution decays to zero. With t generations of selection followed by τ generations of random mating, the cumulative response is

$$t\,h^2\,S + (1-c)^\tau\,R_{AA}(t) \tag{15.7}$$

which (for large τ) converges to $R = t\,h^2\,S$, the value predicted from the breeder's equation. The half-life for decay of R_{AA} is also given by Equation 15.6b.

The presence of epistasis can result in a curvilinear selection response if σ^2_{AA}/σ^2_z is sufficiently large. However, as Figure 15.1 shows, any such curvilinearity is usually difficult to distinguish from a linear response. Unless there is tight linkage, most of the nonlinearity occurs in the first few generations (Equation 15.6b), after which the response is linear, further reducing any signal. With a constant selection differential, the additional increment to response from epistasis decreases each generation as R_{AA} approaches its limiting value $\widetilde{R}_{AA}$, at which point the per-generation response is just h^2S and linear over future generations.

Once selection is relaxed, the total response decays back to that predicted from the breeder's equation. Interestingly, this situation mimics the effects of natural selection countering artificial selection, which also results in a decay of the cumulative response once artificial selection stops. Thus, in order to predict the *permanent* response correctly we must know h^2. If only the parent-offspring slope is estimated, this can overestimate the final amount of response due to the inclusion of σ^2_{AA} and higher-order (additive) epistatic variances, although the bias is generally likely to be small.

Griffing's analysis is restricted to two loci, and hence limited to only pair-wise (additive $\times$ additive) epistasis. Equation 15.1 gives the single-generation response for arbitrary levels of additive epistasis, provided the biparental offspring regression is linear. Again assuming the infinitesimal model (and unlinked loci), Bulmer (1980) found the response due to a single generation of selection decays after one generation to

$$R = S\left(h^2 + \frac{1}{4}\,\frac{\sigma^2_{AA}}{\sigma^2_z} + \frac{1}{16}\,\frac{\sigma^2_{AAA}}{\sigma^2_z} + \frac{1}{64}\,\frac{\sigma^2_{AAAA}}{\sigma^2_z} + \cdots\right) \tag{15.8}$$

which again rapidly converges to $R = h^2\,S$ after several generations of random mating. For n-locus additive epistasis (e.g., $\sigma^2_{A\cdots A}$, where there are n A's), the per-generation decay rate for unlinked loci is $(1/2)^{n-1}$, the probability that a parental gamete containing specific alleles at n unlinked loci is passed on to an offspring. The probability that such a gamete remains unchanged after t generations is $2^{-t(n-1)}$, which rapidly converges to zero. Example 15.2 (below) uses the method of ancestral regressions to more fully examine the response under n-locus additive epistasis. A final caveat is that these results apply to infinite populations. As shown in Chapter 12, with finite populations some of the additive epistatic contribution can be permanent due to some of σ^2_{AA} being transformed into simple additive variation by drift.

Dominance in Autotetraploids

Polyploidy is very common in plants and can introduce complications in predicting the response to selection (Gallais 2003). For example, the dynamics of selection response for autotetraploids with dominance is very similar to diploids with epistasis. From LW Equation 7.22 and LW Table 7.5, the autotetraploid parent-offspring covariance when dominance (but no epistasis) is present is

$$\sigma(z_p, z_o) = \frac{\sigma^2_A}{2} + \frac{\sigma^2_D}{6}$$

The inflation of the parent-offspring covariance over that for a diploid is due to dominance interactions between the two alleles per locus that each autotetraploid parent passes on to its offspring. Thus, like additive epistasis in diploids, favorable *combinations* of alleles can be passed down from parent to offspring in polyploids. With equal amounts of selection on both sexes (e.g., selection occurs before pollination), the resulting response (assuming linearity of the parent-offspring regression) is

$$R = S\left(h^2 + \frac{\sigma_D^2}{3\sigma_z^2}\right) \tag{15.9}$$

If selection occurs after pollination, S is replaced by $S/2$. Gallais (1975) extended Griffing's (1960a) method (and hence assumed phenotypes are normally distributed with each gene having a very small effect on the character) to obtain the response after t generations of selection with constant differential S as

$$R(t) = th^2S + R_D(t) \tag{15.10a}$$

where

$$R_D(t) = S\,\frac{3}{2}\left[1 - \left(\frac{1}{3}\right)^t\right]\,\frac{\sigma_D^2}{3\sigma_z^2} \tag{15.10b}$$

which converges to a limiting value of

$$\widetilde{R}_D = S\,(\sigma_D^2/2\sigma_z^2) \tag{15.10c}$$

This is just a 50% increase over the first-generation response. As with the contribution from epistasis, the total contribution from dominance approaches a limiting value representing the balance between selection favoring specific combinations of alleles and reproduction re-shuffling those combinations. In particular, segregation reduces the departure from tetraploid Hardy-Weinberg proportions (LW Chapter 4) generated by the selection of favorable combinations of allelic pairs, reducing their contribution to response. The response for t generations of selection followed by τ generations of random mating is

$$t\,h^2\,S + (1/3)^\tau\,R_D(t) \tag{15.10d}$$

which again rapidly converges to $t\,h^2\,S$. In LW Chapter 4, it was shown that in an autotetraploid the difference in the frequency of pairs of alleles from Hardy-Weinberg expectation decays by $1/3$ each generation in the absence of double reduction ($c = 0$), as would occur for a locus completely linked to the centromere. More generally, if c is the per-generation probability of a double reduction, the decay rate of $(1/3)^t$ is replaced in the above equations by $(1-c)^t/3^t$. Swanson et al. (1974) found that if some double reductions occur ($c > 0$), the additive variance is slightly inflated over the value expected with no double reductions ($c = 0$), permanently increasing selection response. This results from the slight excess of homozygotes at equilibrium over the Hardy-Weinberg expectation (see LW Chapter 4). Wricke and Weber (1986) discuss additional topics on autotetraploid selection, while single-locus models have been examined by R. Hill (1971), Hagg and Hill (1974), Hill and Hagg (1974), and Rowe and Hill (1984). By far the most complete treatment of selection with autopolypoids is the outstanding text by Gallais (2003).

ANCESTRAL REGRESSIONS

A general approach for examining which components of response are transient is to consider the expected value of an offspring as a function of all its direct relatives that have been under selection (Pearson 1898; Bulmer 1971, 1980). If this **ancestral regression** is linear (as would occur if the joint distribution of the phenotypic values of all relatives is multivariate normal), response can be described by specifying the regression coefficients by an obvious extension of the biparental regression to now include all selected relatives back to the original unselected base population. For example, if selection starts in generation 0, the response in the first generation is $R(1) = 2\,\beta_{1,0}\,S_0$, where $\beta_{1,0}$ is the regression of offspring at generation one on a parent from generation zero (this assumes both parents have the same regression coefficients and selection differentials, an assumption that will be relaxed shortly). Likewise, the total response after two generations, $R(2) = 4\,\beta_{2,0}\,S_0 + 2\,\beta_{2,1}\,S_1$, depends on the nature of selection on the four grandparents (S_0) and both parents (S_1) as well as the transmission from grandparent to grandchild ($\beta_{2,0}$) and parent to offspring ($\beta_{2,1}$). Note that this formulation allows the parent-offspring regression to change through time (e.g., $\beta_{2,1}$ need not equal $\beta_{1,0}$), as can happen with inbreeding (Chapter 23). Similarly, the response following three generations of selection depends upon the nature of selection on that individual's eight great-grandparents, four grandparents, and two parents,

$$R(3) = 8\,\beta_{3,0}\,S_0 + 4\,\beta_{3,1}\,S_1 + 2\,\beta_{3,2}\,S_2$$

Proceeding in this fashion gives the total response by generation T as

$$R(T) = \sum_{t=0}^{T-1} 2^{T-t}\,\beta_{T,t}\,S_t \tag{15.11a}$$

where $\beta_{T,t}$ is the partial regression coefficient for the phenotype of an individual in generation T on one (out of 2^{T-t}) of its relatives in generation $t < T$. With pure selfing each individual has only a single relative, giving the ancestral regression as

$$R(T) = \sum_{t=0}^{T-1} \beta_{T,t}\,S_t \tag{15.11b}$$

Recall from standard regression theory (LW Chapter 8) that the partial regression coefficients are are function of the covariances between relatives, with the vector of regression coefficients given by $\boldsymbol{\beta} = \mathbf{V}^{-1}\boldsymbol{\sigma}$, where $\boldsymbol{\sigma}_t = \sigma(z_T, z_t)$ and $\mathbf{V}_{t,\tau} = \sigma(z_t, z_\tau)$. If we have independence so that the partial regression coefficients reduce to univariate regression coefficients (i.e., $\beta_i = \sigma(y, x_i)/\sigma^2_{x_i}$), then

$$R(T) = \sum_{t=0}^{T-1} 2^{T-t}\,\frac{\sigma_G(T,t)}{\sigma^2(z_t)}\,S_t \tag{15.11c}$$

where $\sigma_G(T,t) = \sigma(z_T, z_t)$ is the **cross-generation covariance**, the phenotypic covariance between an individual in generation t and its descendent in generation $T > t$. With selection under pure selfing, each individual has a single ancestor and the 2^{T-t} term in Equation 15.11c is absent. As we will see in Chapter 23, ancestral regression offers a very powerful approach for the analysis of selection under inbreeding, where the cross-generation covariances are

not simple functions of $T - t$. For example, the parent-offspring covariance changes over generations as inbreeding proceeds.

If different relatives in the same generation experience different amounts of selection, with $S_{k,i}$ being the selection differential on relative i in generation k, then

$$R(T) = \sum_{t=0}^{T-1} \left[\beta_{T,t} \left(\sum_{i=1}^{n(t,T)} S_{t,i} \right) \right] \tag{15.12}$$

where $n(t, T)$ is the number of relatives in generation t that contribute to response in generation T. Note for the case of pure selfing $n(t, T) = 1$. Finally, we can also allow for different regression coefficients on each relative to completely generalize this approach,

$$R(T) = \sum_{t=0}^{T-1} \left(\sum_{i=1}^{n(t,T)} \beta_{T,t,i} \, S_{t,i} \right) \tag{15.13}$$

where $\beta_{T,t,i}$ is the partial regression coefficient of the phenotype of an individual in generation T on its i-th relative in generation t.

To apply ancestral regression for predicting response, we require that the regression remains linear and that *selection-induced* changes in the variances and covariances are negligible. Thus, while we allow changes in $\beta_{T,t}$ due to the particular genetic system being considered (e.g., selfing wherein the additive genetic variance decreases by a predictable amount each generation in the absence of selection), we assume that selection does not confound these changes. Bulmer (1980) shows that the joint distribution of an offspring and all its direct ancestors is multivariate normal, and hence the ancestral regression is linear (LW Chapter 8), under the infinitesimal model. Since selection does not change allele frequencies under the infinitesimal model, this might suggest that the regression coefficients $\beta_{T,t}$ are unaffected by selection. The problem, however, is that selection generates gametic-phase disequilibrium which can significantly alter the genotypic moments (Chapters 16, 24). For now, we assume that these changes (over short time scales) are small enough to be neglected. In Chapter 19 we show that BLUP estimates of breeding values are a type of ancestral regression, with the relationship matrix $\mathbf{A}$ accounting for drift and disequilibrium.

Example 15.1: As an application of ancestral regressions, consider additive-by-additive epistasis. In this case, Cockerham (1984) found (under the infinitesimal model) that for two linked loci, the cross-generation covariance is

$$\sigma_G(\tau + t, \tau) = \frac{\sigma_A^2(\tau)}{2^t} + \frac{\sigma_{AA}^2(\tau)}{2} \left(\frac{1 - c}{2} \right)^t,$$

giving

$$2^t \, \sigma_G(\tau + t, \tau) = \sigma_A^2(\tau) + (1 - c)^t \, \frac{\sigma_{AA}^2(\tau)}{2}.$$

Provided the genetic variances remain constant, applying Equation 15.11a we recover Equation 15.3.

The behavior of the regression coefficients over time informs us as to the permanency of response. Equation 15.11a shows that unless $2^t \beta_{\tau+t,\tau}$ remains constant as t increases, the contribution to cumulative response from selection on adults in generation τ changes over time. For strictly additive loci, $\sigma_G(\tau+t,\tau) = 2^{-t}\sigma_A^2(\tau)$ and thus $2^t \beta_{\tau+t,\tau} = h_\tau^2$, the standard result from the breeder's equation. Conversely, any term of $\sigma_G(\tau+t,\tau)$ that decreases by more than $1/2$ each generation contributes only to the transient response, as $2^t \sigma_G(\tau+t,\tau) \to 0$ as $t \to 0$. An exception is with pure selfing, as an individual has only a single ancestor t generations ago (as opposed to 2^t ancestors under random mating). As a result, the total contribution in generation $t+\tau$ from an ancestor in generation τ is $\sigma_G(\tau+t,\tau)$, so that any components that decline as τ increases will contribute to the transient response.

Example 15.2: Consider the expected response under arbitary levels of additive epistasis (under the infinitesimal model with unlinked loci). LW Equation 7.12 gives the genetic covariance between relatives x and y as

$$\begin{aligned}\sigma_G^2(x,y) &= (2\Theta_{x,y})\,\sigma_A^2 + (2\Theta_{x,y})^2\,\sigma_{AA}^2 + (2\Theta_{x,y})^3\,\sigma_{AAA}^2 + \cdots + (2\Theta_{x,y})^i\,\sigma_{A^i}^2 \\ &= \sum_{i=1}^{n} (2\Theta_{x,y})^i\,\sigma_{A^i}^2\end{aligned}$$

The coefficient of coancestry $\Theta_{t,t+\tau}$ between a parent in generation t and its direct great-offspring in generation $t+\tau$ under random mating is

$$\Theta_{t,t+\tau} = \left(\frac{1}{2}\right)^{\tau+1}.$$

Now let's compute the contribution $\sigma_{G,i}(t+\tau,t)$ to the total genetic covariance due to i-th order additive epistasis (A^i). Substituting the above results gives

$$\frac{\sigma_{G,i}(t+\tau,t)}{\sigma_{A^i}^2} = (2\Theta_{t+\tau,t})^i = 2^i\left(\frac{1}{2}\right)^{(\tau+1)i} = \left(\frac{1}{2}\right)^{\tau i},$$

with the ancestral regression term becoming

$$2^\tau \frac{\sigma_{G,i}(t+\tau,t)}{\sigma_{A^i}^2} = 2^\tau 2^{-\tau i} = \left(\frac{1}{2}\right)^{\tau(i-1)} = \left(\frac{1}{2^{i-1}}\right)^\tau.$$

With constant selection S, the contribution to total response from i-th order additive epistasis follows from Equation 15.11c as

$$R_{A^i}(t) = S\frac{\sigma^2(A^i)}{\sigma^2(z)}\sum_{\tau=1}^{t}\left(\frac{1}{2^{i-1}}\right)^\tau.$$

Recalling Equation 15.5b,

$$\sum_{\tau=1}^{t}\left(\frac{1}{2^{i-1}}\right)^\tau = \frac{x-x^{t+1}}{1-x} \quad \text{where} \quad x = (1/2)^{i-1}$$

The limit of this sum (as $t \to \infty$) is

$$\frac{x}{1-x} = \frac{(1/2)^{i-1}}{1-(1/2)^{i-1}} = \frac{1}{2^{i-1}-1}$$

Since the initial contribution ($t = 1$) is $(1/2)^{i-1}$, the extra increment to response beyond that seen in the first generation is

$$\widetilde{R}_{A^i} - R_{A^i}(1) = \frac{1}{2^{i-1}-1} - \frac{1}{2^{i-1}} = \frac{1}{(2^{i-1}-1)2^{i-1}}$$

The response in generation one $R(1)$ and at the limit (both in units of of $S\sigma^2(A^i)/\sigma_z^2$), and the fraction of total response occuring in the first generation are as follows:

	AA	AAA	AAAA	AAAAA
$R(1)$	0.500	0.250	0.125	0.063
Limit	1.000	0.333	0.143	0.067
$R(1)$/Limit	0.500	0.750	0.875	0.938

For higher-order additive epistasis among unlinked loci, the limiting contribution is small and essentially all of the response occurs in the first generation.

RESPONSE DUE TO ENVIRONMENTAL CORRELATION

Imagine a situation where a bird is large by chance, and that this large size makes them able to defend a larger and more productive breeding location. As a result, their offspring are better provisioned and are larger themselves, above and beyond any genetic effects on size. This is an example of a **shared environmental effect** and is a special case of the more general setting where at least part of the environment experienced by an individual is a function of the phenotypes of other individuals with which they interact (Chapter 22). Rossiter (1996) and Bonduriansky and Day (2009) review a number of such shared environmental effects.

As Equation 15.1 indicates, shared parent-offspring environmental effects ($\sigma(E_p, E_o) \neq 0$) can influence response. This contribution is also transient. Consider a character whose variation is entirely environmental, in which case the phenotypic value can be decomposed as

$$z = \mu + E = \mu + e_{fa} + e_{mo} + e$$

where μ is the mean value of the character when environmental effects are randomly distributed, and the environmental value E has been decomposed into the maternal and paternal contributions to the offspring due to shared environmental effects (e_{mo} and e_{fa}) and a residual due to special environmental effects (e). In order to predict the shared environmental contribution from a parent, we assume the simplest model, that a fraction b of the total environmental value of a parent is passed on to its offspring. This model serves as a useful introduction to some of the dynamics that can occur for certain models of maternal effects (examined in the next section). The expected contribution from a father to his offspring is $e_{fa} = b\,E_{fa} = b\,(z_{fa} - \mu)$, where E_{fa} is the father's total environmental value. To simplify matters further, assume that this regression coefficient is independent of the sexes of the parent and offspring, although this can easily be relaxed. Further assuming that parents

and offspring have the same phenotypic variance, then $b = \rho/2$, where ρ is the slope of the midparent-offspring regression, which can be negative. For example, suppose parents and offspring compete for a limited amount of common resource. Larger parents may gather a disproportionate share of resources, resulting in smaller offspring.

Provided E_{fa} and E_{mo} are uncorrelated, the expected value of an offspring from parents with phenotypic values z_{fa} and z_{mo} is

$$E(\, z_o \,|\, z_{mo}, z_{fa}\,) = \mu + \frac{\rho}{2}\,(\, z_{fa} - \mu\,) + \frac{\rho}{2}\,(\, z_{mo} - \mu\,) \tag{15.14a}$$

Denoting the mean of adults selected in generation t by μ_t^*, the mean at generation $t+1$ is given by

$$\mu_{t+1} = \mu + \rho\,(\,\mu_t^* - \mu\,) \tag{15.14b}$$

where $(\,\mu_t^* - \mu\,)$ is the environmental deviation in selected parents at generation t, ρ of which is passed on to their offspring. If shared environmental effects are only passed on through one parent, $\rho/2$ replaces ρ in Equation 15.14b. Rewriting the mean after selection as $\mu_t^* = \mu_t + S_t$,

$$\mu_{t+1} = \mu + \rho\,(\,\mu_t + S_t - \mu\,) \tag{15.15}$$

The change in mean in generation t, $\Delta\mu_t = \mu_{t+1} - \mu_t$, is

$$\begin{aligned}\Delta\mu_t &= [\,\mu + \rho\,(\,\mu_t + S_t - \mu)\,] - [\,\mu + \rho\,(\mu_{t-1} + S_{t-1} - \mu\,)\,] \\ &= \rho\,[\,(\,\mu_t - \mu_{t-1}) + (S_t - S_{t-1}\,)\,] \\ &= \rho\,[\,\Delta\mu_{t-1} + (S_t - S_{t-1})\,]\end{aligned} \tag{15.16a}$$

Suppose that constant selection (with differential S) is applied starting at generation 1. Here, $\Delta\mu_0 = 0$, $S_0 = 0$, and $S_t = S$ for $t \geq 1$. Equation 15.16a gives $\Delta\mu_1 = \rho\,S$. Further iterations yield

$$\Delta\mu_t = \rho^t\,S, \tag{15.16b}$$

which decreases each generation, approaching zero for large t. Hence, even under continued selection, the total response to selection eventually reaches a limit. Note that if $\rho < 0$, the sign of the response switches each generation (Figure 15.2), although the cumulative response has a constant sign (Equation 15.17a).

The reason for this decline in the per-generation rate of response can be seen from Equation 15.16b. Change in the character mean due to previous selection decays, countering gain from selection in the current generation. Only a fraction ρ of the change from generation $t-1$ is passed on, and, in general, only ρ^k of the response from generation $t-k$ persists by generation t. Summing over Equation 15.16b, the total response to selection after t generations is

$$R(t) = \mu_{t+1} - \mu_0 = \sum_{i=1}^{t} \Delta\mu_i = S\sum_{i=1}^{t} \rho^i \tag{15.16c}$$

Recalling the partial sum of a geometric series (Equation 15.5b), this reduces to

$$R(t) = S\,\frac{\rho}{1-\rho}\,(\,1 - \rho^t\,). \tag{15.17a}$$

As was the case for epistasis, the total cumulative response reaches an equilibrium value representing the balance between selection generating correlations and reproduction removing them, with

$$\widetilde{R} = \lim_{t\to\infty} R(t) = S\,\frac{\rho}{1-\rho}. \tag{15.17b}$$

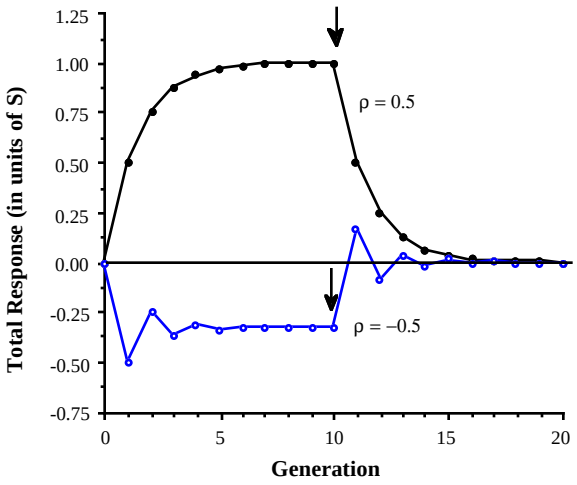

Figure 15.2. Response when resemblance between relatives is due entirely to correlation between environmental values in parents and offspring. Selection with constant differential S starts at $t = 0$ and continues until generation 10 (indicated by the arrow), at which point selection is stopped. Note the interesting dynamics that occur if environmental values are negatively correlated. The response to selection is reversed with respect to the selection differential. In this case, selection for *increased* character value results in a *decreased* mean value, with the total response eventually converging to $-S/3$ (for $\rho = -0.5$). Once selection is relaxed there is an initial positive response (generation 11), although response quickly decays to zero.

Thus, no matter how long selection is applied, the mean can never change by more than $S\rho/(1-\rho)$. Further, none of this response is permanent. Suppose selection is stopped after t generations, giving $S_t = S$, $S_{t+\tau} = 0$ for $\tau \geq 1$. Substituting into Equation 15.16a and using Equation 15.16b, the expected change in generation $t + \tau$ is

$$\Delta\mu_{t+\tau} = \rho^\tau \, (\, \Delta\mu_t - S \,) = -S \, \rho^\tau \, (\, 1 - \rho^t \,). \tag{15.18}$$

By generation $t + \tau$ the cumulative response is

$$R(t+\tau) = R(t) + \sum_{i=1}^{\tau} \Delta\mu_{t+i} = R(t) - S \, (\, 1 - \rho^t \,) \sum_{i=1}^{\tau} \rho^i = \rho^\tau \, R(t) \tag{15.19}$$

which converges to zero, with the rate of decay being set by ρ (Figure 15.2). Hence, while there can be some selection response when the resemblance between relatives is entirely environmental, any response is transient, decaying away once selection stops. Further, no matter how long selection proceeds, the response reachs a limit beyond which no further response is possible (Equation 15.17b).

SELECTION IN THE PRESENCE OF HERITABLE MATERNAL EFFECTS

A mother can influence the phenotype of her offspring in two ways. The assumption up to now is simply through the genes (including those on organelle genomes) that she transmits. Together with the paternal genes, these create the genotype of the offspring, and the

expression of this genotype, together with the environment it experiences, determine its phenotype. The second route is through maternal effects, traits or genes *expressed in the mother* that influence the phenotypes of her offspring (Wolf and Wade 2009). These can be thought of as influencing the *environment* that her offspring's genotype experiences, which can significantly influence its phenotype. Examples include **maternal performance characters** such as body size, amount of care invested in offspring, milk yield, and endosperm production that potentially influence a variety of traits in her progeny. These maternal performance traits can themselves have a genetic basis and also respond to selection, creating potentially very complex dynamics even in the simplest of settings.

Example 15.3: Maternal effects include genes expressed in the mother than influence the very early development of her offspring, with the maternal phenotype of these genes being extremely difficult to measure in the mother. For many metazoans, much of the gene expression during the first few rounds of cell division in a newly fertilized zygote is due to the translation of maternal mRNAs deposited in the egg. The resulting gene products provide information for early key development steps in the nascent embryo, with their phenotypic expression seen in trait values of her offspring, rather than the mother. The classic example is the work by Sturtevant (1923) on right- versus left-handed shell coiling of the snail *Limnaea*, where the genotype of the mother, not the offspring, determines offspring phenotype. Offspring from a mother homozygous for the *sinistral* allele are all left-handed, independent of their genotypes, while the presence of at least one *dextral* allele in the mother yields all right-handed offspring. While the maternal trait is easy to assay given her offspring, it cannot be assayed using just the mother, as the gene products are still unknown, although Kuroda et al. (2009) have shown the direction of coiling is determined at the eight-cell stage of the embryo.

Paternal effects are also possible, especially in situations where the father plays a role in caring for the offspring. While not considered here, they can be treated in exactly the same fashion as maternal effects. There is an extensive literature on the maternal effects and their evolutionary implications (reviewed by Roach and Wulff 1987, Bernardo 1996, Rossiter 1996, Mousseau and Fox 1998, Reinhold 2002, Räsänen and Kruuk 2007, Mousseauet al. 2009). Here we introduce some of the basic ideas, with additional material developed in Chapter 23. The bulk of our discussion will be in Volume 3, within the framework of multivariate selection response.

Decomposing Maternal Effects

Assuming a maternal effect, the environmental value E of an individual can be decomposed as $E = M + e$, a **maternal performance** component M plus an environmental deviation e , giving

$$z = G + E = G + M + e$$

The genotypic value G of the offspring is often referred to as the **direct** (or **intrinsic**) **effect** on the trait, reflecting the intrinsic genetics of the individual. If M is entirely environment, the results of the previous section apply, and any contribution from maternal effects is transient. However, there is often a *genetic* component to M, so that selection can change the population means of *both* the direct and maternal values (Dickerson 1947; Willham 1963, 1972; Cheverud 1984; Riska et al. 1985; Kirkpatrick and Lande 1989). In this case,

$M = G_m + E_m$, where G_m is the contribution to z resulting from the mother's genotypic value for the maternal performance character, while E_m is the contribution resulting from the environmental value of the maternal performance character (reviewed in LW Chapter 23). Although M is treated as an environmental effect from the offspring's standpoint, it can have both a genetic and environmental basis in the mother.

Letting G_d denote the genotypic value of the direct effect (hitherto G), the phenotypic value of an individual can be written in terms of direct and maternal contributions,

$$z = G + M + e = G_d + G_m + E_m + e.$$

This significantly complicates selection response, as selection to increase a trait might result in an improved direct value ($\Delta\mu_{G_d} > 0$) but a decreased maternal value ($\Delta\mu_{G_m} < 0$) if the correlation between G_d and G_m is negative. In the extreme, this can result in a reversed response, with the decline in maternal environment overwhelming any improvement in direct effects. Since trait value z is a function of two potentially correlated genetic components, G_d and G_m, this is formally a multiple-trait problem and can be attacked with the machinery of multivariate selection (Cheverud 1984; Kirkpatrick and Lande 1989, 1992; Lande and Kirpatrick 1990). Here we first consider the simplest case, where M is a function of the value of the focal trait in the mother, which collapses the model to a single trait problem, but with much more complicated dynamics than given by the simple breeder's equation. We then very briefly discuss a two-trait model, while Volume 3 examines maternal effects in the full multivariate framework.

An important point to note is that maternal effects models are a special case of **associative effects** (or **social effects**, or **indirect genetic effects**) models, where the phenotype of an individual within a group is a function of both its direct value plus the associative values from members of its group. Both kin and group selection are also special cases of this more general formulation, which is examined in detail in Chapter 22.

Response Under Falconer's Dilution Model

The simplest model of maternal effects (motivated by the inheritance of litter size in mice) is that of Falconer (1965): the value of the focal trait in the mother determines M (reviewed in LW Chapter 23). Falconer reasoned that offspring from large litters get less maternal resources that those in smaller litters, and that this can have carry-over effects when they become mothers themselves. Assume that the maternal contribution is a linear function of the maternal phenotype z_{mo}, so that $M = mz_{mo}$ and the phenotypic decomposition becomes

$$z = G + m\, z_{mo} + e \tag{15.20}$$

Conceivably, M could be a nonlinear function of z_{mo}, but linearity is assumed for tractability. Equation 15.20 is called the **dilution model**, since the effect of the maternal phenotype becomes diluted over successive generations (for $|m| < 1$). The parameter m can be regarded as the partial regression coefficient (holding genotypic value constant) of offspring phenotype on maternal phenotype and can be estimated as the difference between the maternal- and paternal-offspring regression slopes (LW Equation 23.13).

Negative estimates of m have been reported: values of -0.15 for litter size in mice (Falconer 1965), -0.58 and -0.40 for age of maturity in two replicate lines of the springtail insect *Orchesella cincta* (Janssen et al. 1988), -0.25 for clutch size in the collared flycatcher *Ficedula albicollis* (Schluter and Gustafsson 1993), and -0.29 for juvenile growth rate in the red squirrel *Tamiasciurus hudsonicus* (McAdam and Boutin 2003)

Assume that the joint distribution of phenotypes and breeding values in parents and offspring is multivariate normal. Further assuming no (additive) epistasis, the expected

phenotypic value of an offspring whose mother has phenotypic value z_{mo} is

$$E(z_o \mid A_{mo}, A_{fa}, z_{mo}) = \frac{A_{mo}}{2} + \frac{A_{fa}}{2} + m\, z_{mo} \tag{15.21a}$$

where A_{mo} and A_{fa} are the maternal and paternal breeding values (see Example 7 in LW Chapter 8). Averaging over the selected parents, the mean in generation $t+1$ becomes

$$\mu_z(t+1) = \frac{A^*_{fa}(t) + A^*_{mo}(t)}{2} + m\, \mu^*_{mo}(t) \tag{15.21b}$$

where $A^*_{fa}(t)$ and $A^*_{mo}(t)$ are the mean breeding values of the selected parents and $\mu^*_{mo}(t)$ the mean phenotypic value of selected mothers in generation t. Using the regression of breeding value on phenotype

$$A = \mu_A + b_{Az}\,(\, z - \mu_z\,) + e$$

allows us to predict the breeding value A of an individual from its phenotypic value z. In particular, we can rewrite $A^*_{mo}(t)$ as

$$\begin{aligned} A^*_{mo}(t) &= E_s\left(\, \mu_A(t) + b_{Az}\,[\, z_{mo} - \mu_z(t)\,] + e \,\right) \\ &= \mu_A(t) + b_{Az} S_{mo}(t) \end{aligned} \tag{15.22}$$

where $E_s(\ )$ denotes the expected value over the selected parents. A similar expression holds for $A^*_{fa}(t)$. In the absence of maternal effects, $b_{Az} = h^2$. However, the dilution model generates a covariance between M and A, specifically $\sigma_{A,M} = m\,\sigma^2_A\,/\,(\,2-m\,)$, which in turn alters the covariance between z and A (Falconer 1965, Kirkpatrick and Lande 1989; see LW Equation 23.12a). The resulting regression slope (at equilibrium) is

$$b_{Az} = h^2 \frac{2}{2-m} \tag{15.23}$$

If there is a negative maternal effect ($m < 0$), $b_{Az} < h^2$, reducing the correlation between breeding value and phenotype. Conversely, $m > 0$ increases the correlation between breeding value and phenotype above h^2. Applying Equations 15.21–15.23 and using $\mu^*_{mo}(t) = \mu_z(t) + S_{mo}(t)$, gives

$$\mu_z(t+1) = \mu_A(t) + \frac{h^2}{2-m}\Big(S_{mo}(t) + S_{fa}(t)\Big) + m\Big(\mu_z(t) + S_{mo}(t)\Big) \tag{15.24}$$

The change in population mean over one generation, $\Delta\mu_z(t)$, is thus

$$\begin{aligned} \Delta\mu_z(t) = \mu_z(t+1) - \mu_z(t) =& \left[\, \mu_A(t) + \frac{h^2}{2-m}\Big(S_{mo}(t) + S_{fa}(t)\Big) + m\Big(\mu_z(t) + S_{mo}(t)\Big)\right] \\ &- \left[\, \mu_A(t-1) + \frac{h^2}{2-m}\Big(S_{mo}(t-1) + S_{fa}(t-1)\Big) + m\Big(\mu_z(t-1) + S_{mo}(t-1)\Big)\right] \\ \\ =& \frac{h^2}{2-m}\Big(S_{mo}(t) + S_{fa}(t)\Big) + m\, S_{mo}(t) + m\Big(\Delta\mu_z(t-1) - S_{mo}(t-1)\Big) \end{aligned} \tag{15.25}$$

The last simplification follows from the regression of breeding value on phenotype, with

$$\mu_A(t) = \mu_A(t-1) + \frac{h^2}{2-m}\Big(S_{mo}(t-1) + S_{fa}(t-1)\Big).$$

Equation 15.25 can be interpreted as follows: the first two terms are the change in character value resulting from selection in generation t due to genetic ($h^2 / [\, 2 - m \,]$) and maternal (m) contributions. The final term, which can also be expressed as $m\,[\,\mu_z(t) - \mu_z^*(t-1)\,]$, represents the decay in the maternal contribution from the previous generation.

Starting with an unselected base population, the response to a single generation of selection is

$$\Delta\mu_z(1) = \frac{h^2}{2-m}\Big(S_{mo}(1) + S_{fa}(1)\Big) + mS_{mo}(1) \tag{15.26}$$

An interesting consequence of Equation 15.26 is that if $m < 0$, there is some possibility of a reversed response, where $\Delta\mu_z$ has opposite sign of S. If $S_{fa} = S_{mo} = S$, a reversed response is expected if

$$m < 1 - \sqrt{1 + 2h^2} \tag{15.27a}$$

If selection is only occuring on females, this condition is

$$m < 1 - \sqrt{1 + h^2} \tag{15.27b}$$

An example of an apparent maternally-induced reversed response was seen by Falconer (1960, 1965) in his selection experiments on litter size in mice. This character shows a negative maternal effect, with m and h^2 estimated to be -0.13 and 0.11, respectively. Since selection for litter size occurs only in females, Equation 15.27b implies that a reversed response in the first generation is expected (as $1 - \sqrt{1 + 0.11} \simeq -0.05 > m$). As Figure 15.3 shows, a reversed response was indeed observed.

An observed reversed response is misleading because the *permanent* response is expected to have the same sign as S, while the initial observed response also includes a transient component that (in this case) is of opposite sign and of larger magnitude than the permanent response component. It may take several generations for this transient component to decay and reveal the actual genetic changes (Figure 15.3), which is (for a single generation of selection) $2S\,h^2/\,[\,(2-m)(1-m)\,]$, as shown by Equation 15.33 (below).

The possibility of reversed response hints at some of the complicated dynamics that can appear when maternal effects are present. To examine these dynamics in more detail, consider the dilution model with constant directional selection occuring equally on both sexes. i.e., $S_{fa}(t) = S_{mo}(t) = S$ for $t \geq 1$. Iteration of Equation 15.25 gives

$$\Delta\mu_z(t) = S\left[\frac{2h^2}{(1-m)(2-m)}\Big(1 - m^t\Big) + m^t\right] \tag{15.28a}$$

which converges (for $|m| < 1$) to

$$\Delta\mu_z = S\,\frac{2h^2}{(1-m)(2-m)} \tag{15.28b}$$

After a sufficient number of generations, the per generation change is constant. If $|m|$ is near zero, the per generation response rapidly converges to this asymptotic value, while if $|m|$ is near one, the rate of convergence is considerably slower. Summing over the single-generation changes (Equation 15.28a) and recalling Equation 15.5b, the cumulative response to t generations of selection is

$$R(t) = \sum_{i=1}^{t} \Delta\mu_z(i) = \frac{S}{1-m}\left[\,t\,\frac{2h^2}{2-m} + m\,(1-m^t)\left(1 - \frac{2h^2}{(1-m)(2-m)}\right)\right], \tag{15.29a}$$

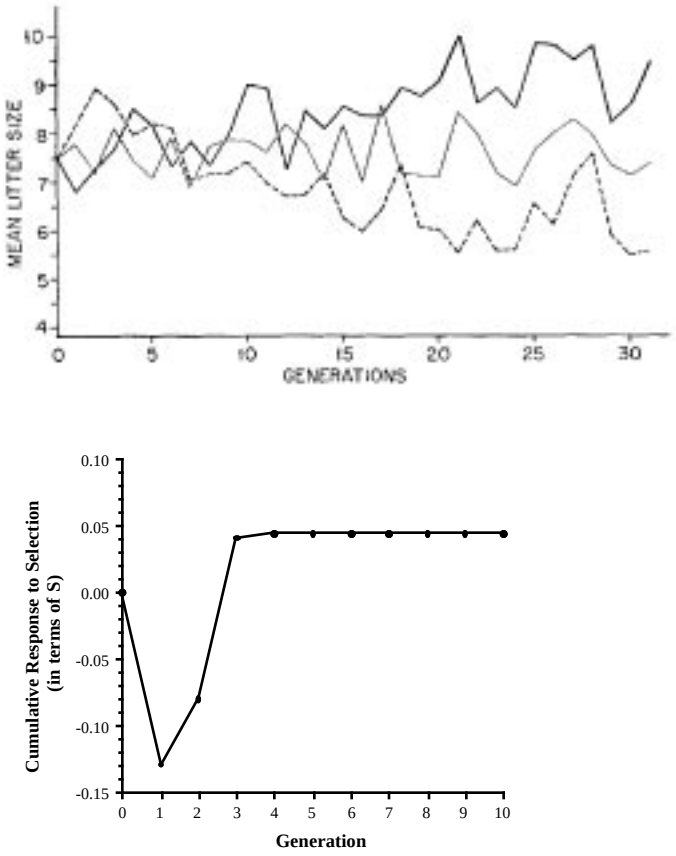

Figure 15.3. **Top:** Falconer's experiments on selection response for litter size in mice. The dashed line is the response to selection for small litters, the thick line selection for large litters, and the thin line the control. Note the reversed response in the first generation in both the up- and down-selected lines. After Falconer (1960). **Bottom:** Predictions from the model, using Falconer's estimated values of $h^2 = 0.11$ and $m = -0.13$. The predicted change in population mean following a single generation of selection on females with $S_{mo} > 0$ is plotted. There is a reversed response in the first generation, even though the net genetic change is to increase the character. By generation 3, the nongenetic change in phenotypic mean has largely decayed away, revealing the net genetic change of $S_{mo}\, h^2/[(1-m)(2-m)] = 0.044 \cdot S_{mo}$.

which converges (for $|m| < 1$) to

$$\frac{S}{1-m}\left[\frac{2h^2}{2-m}\left(t - \frac{m}{1-m}\right) + m\right] \tag{15.29b}$$

How much of this response is permanent? Suppose selection ends at generation t, and denote by τ the number of generations since selection was stopped. Iterating Equation 15.25 with $S(t) = S$, $S(t+\tau) = 0$ for $\tau \geq 1$ yields

$$\Delta\mu_z(t+\tau) = m^\tau(\Delta\mu_z(t) - S) \tag{15.30}$$

where $\Delta\mu_z(t)$ is given by Equation 15.28a. Thus, *response continues even after the cessation of selection*, a feature that Kirkpatrick and Lande (1989) call **evolutionary momentum**. Using Equation 15.5b to sum Equation 15.30 over τ yields the cumulative response *following* the last generation of selection,

$$R^*(\tau) = \frac{m(1-m^\tau)}{1-m}\,(\,\Delta\mu_z(t) - S\,) \tag{15.31}$$

which converges as $\tau \to \infty$ to

$$R^* = S\,\frac{m\,(1-m^t)}{1-m}\left[\frac{2h^2}{(1-m)(2-m)} - 1\right] \tag{15.32}$$

Summing Equations 15.29a and 15.32, the *permanent* response to t generations of selection is

$$R(t) + R^* = t\,h^2\,S\,\frac{2}{(1-m)(2-m)}, \tag{15.33}$$

which is just t times the asymptotic response (Equation 15.28b). If R^* is opposite in sign to S, there is some erosion of the cumulative response upon relaxation of selection (we have already seen a special case of this with reversed response). For $|m| < 1$, erosion in response occurs if

$$0 < m < \frac{3 - \sqrt{1+8h^2}}{2} \tag{15.34a}$$

On the other hand, if maternal effects are either negative ($m < 0$) or sufficiently large

$$m > \frac{3 - \sqrt{1+8h^2}}{2} \tag{15.34b}$$

the response continues for a few generations following the relaxation of selection. This occurs by the transient component of response decaying away to reveal the actual permanent response due to changes in breeding values. Figure 15.4 plots some sample trajectories.

In summary, the presence of maternal effects introduces several complications even under this simplest of models (the direct and maternal trait are the same). First, predicting the response to selection in a given generation requires not only of the inheritance parameters (m, h^2) and current selection differential, but also requires knowledge of *previous selection* [$\Delta\mu_z(t-1)$, $S_{mo}(t-1)$]. Second, after selection is stopped, the mean is likely to continue to change due to lag effects (e.g., Figure 15.4). If $m < 0$, the response will continue, while if $m > 0$ the response can either continue or decay. This clearly causes problems if we are trying to estimate the nature of selection acting on a character by comparing changes in means between generations. For example, an observed cross-generation decrease in a character could be due to four very different causes: (i) $S < 0$, (ii) $S > 0$ and a reversed response due to maternal effects, (iii) no selection in the observed generation but a previous history of $S > 0$, with the decrease in mean due to a positive maternal effect (reflecting a decay in response), or (iv) no current selection but a previous history of $S < 0$, with the decrease in mean due to a negative (or sufficiently large positive) maternal effect (reflecting a continuation of response).

Separate Direct and Maternal Traits

Despite its complex dynamics, Falconer's approach is based on the simplest model of maternal effects, where the direct trait has a dual role as the maternal trait. A more realistic

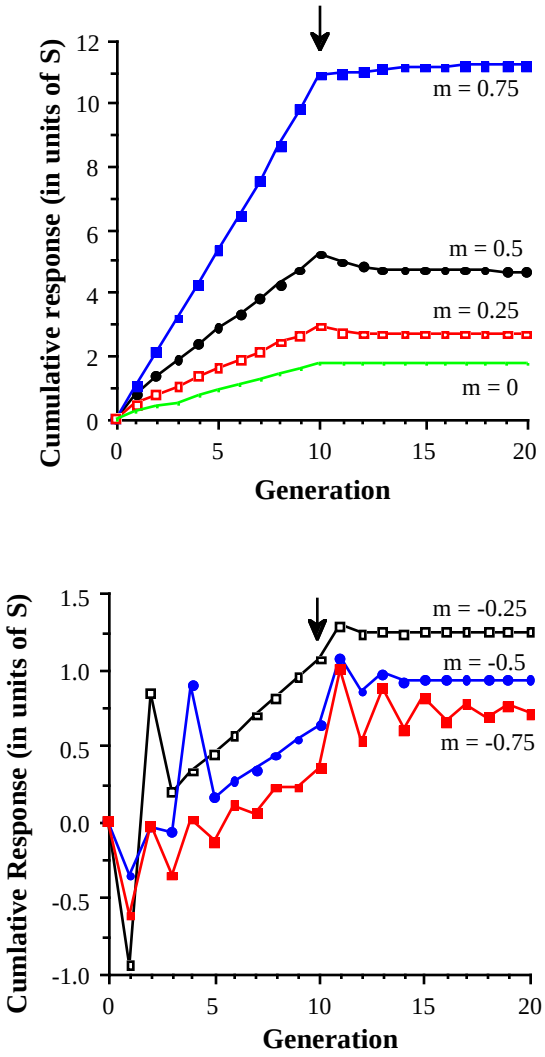

Figure 15.4. Examples of the predicted selection response with maternal effects under Falconer's dilution model. Selection starts at generation zero, with $S_{fa} = S_{mo} = S$ until generation 10 (arrow), at which point selection stops. We assume $h^2 = 0.35$, with the different curves corresponding to different maternal effect values, m. **Top:** Positive maternal effects ($m > 0$). For this value of h^2, Equations 15.34a gives the critical m value as 0.52, so that for $m = 0.75$ response continues (for a few generations) after selection is relaxed, while response decays for $m = 0.5$ and 0.25. **Bottom:** Negative maternal effects ($m < 0$). The dynamics here are considerably more interesting, with additional response following the cessation of selection for all values of $m < 0$.

model is to imagine that the direct and maternal effects are separate traits, for example weight in an offspring and milk production in its mother. In this case, Equation 15.20 now

becomes

$$z_d = G_d + m\, z_{m,mo} + e \tag{15.35}$$

where z_d is the value of the direct trait in an offspring (e.g., its weight) and $z_{m,mo}$ the value of the maternal trait in its mother (e.g., her milk production). Clearly, this generalizes to a vector of direct traits depending on a vector of maternal traits, as modeled by Kirkpatrick and Lande (1989, 1992) and Lande and Kirkpatrick (1990). We examine their full multivariate treatment in detail Volume 3. Here, we briefly comment on the bivariate case (a single direct z_d and a single, and distinct, maternal trait z_m). In this case, the joint dynamics were obtained by Kirkpatrick and Lande (1989, 1992) as

$$R_m(t) = \left(G_{md} + \frac{m}{2} G_{mm}\right) \beta_d(t) + G_{mm}\beta_m(t) \tag{15.36a}$$

$$\begin{aligned} R_d(t) = &\left(G_{dd} + \frac{m}{2} G_{md}\right) \beta_d(t) + G_{md}\beta_m(t) + mR_m(t-1) \\ &+ m\left[P_{mm}\Delta\beta_m(t-1) + P_{md}\Delta\beta_d(t-1)\right] \end{aligned} \tag{15.36b}$$

where G_{xy} and P_{xy} are, respectively, the additive genetic and phenotypic covariances between traits x and y, while β_d and β_m are the selection gradients on the direct and maternal traits. Note that the expression given for Equation 15.36b is based on the corrected version of Kirkpatrick and Lande (1992). As with the simple Falconer model, there are time lags in the direct response due to dependency of response on selection in the previous, as well as the current, generation. By contrast, no such lags are seen for the maternal response.

Example 15.4. McAdams and Boutin (2004) examined the response in mean juvenile growth rate of red squirrels (*Tamiasciurus hudsonicus*) in a long-term monitored population from southwest Yukon. Significant yearly variation was seen in the selection gradient for this trait, to the point were β took on both positive and negative values. Given that previous work showed significant maternal effects on this trait (McAdams et al. 2002), the authors applied Equation 15.36b to see if it resulted in an improved fit to the observed responses. They chose litter size as the maternal trait influencing growth rate. While there was evidence for stabilizing selection on litter size, there was no evidence for directional selection and β_m was set to zero. Inclusion of this single maternal effect resulted in a significant improvement of the fit compared to a model with no maternal effects. The authors also found, consistent with the above model, that response in a given generation was influenced by the strength of selection in both the current and previous generation.

A few other features of this model are worth noting. In the absence of genetic variance for the direct trait ($G_{dd} = G_{md} = 0$), there can still be a permanent response in the direct trait due to response in the maternal trait (the $mR_m(t-1)$ term in Equation 15.36b). Likewise, in the absence of selection on the maternal effect ($\beta_m = 0$), it can still evolve when the direct and maternal trait are genetically correlated ($G_{md} \neq 0$). As reviewed by Wilson and Réale (2006), this covariance is often both significant, and negative, so that selection to increase the trait value can result in a declining maternal value (Cheverud 1984; Chapter 22).

Example 15.5. Assume constant selection differentials following the start of selection, so that (after the first generation) terms involving $\Delta\beta$ (changes in β) are zero. Equation 15.26

simplifies to

$$R_m(t) = \left(G_{md} + \frac{m}{2}G_{mm}\right)\beta_d + G_{mm}\beta_m \tag{15.37a}$$

$$\begin{aligned} R_d(t) &= \left(G_{dd} + \frac{m}{2}G_{md}\right)\beta_d + G_{md}\beta_m + mR_m(t-1) \\ &= \left(G_{dd} + \frac{m}{2}G_{md}\right)\beta_d + G_{md}\beta_m + m\left[\left(G_{md} + \frac{m}{2}G_{mm}\right)\beta_d + G_{mm}\beta_m\right] \\ &= \left(G_{dd} + \frac{3m}{2}G_{md} + \frac{m^2}{2}G_m\right)\beta_d + \left(G_{md} + mG_{mm}\right)\beta_m \end{aligned} \tag{15.37b}$$

One central question in these (and more general) models is which trait (or traits) in the mother determine the value of the maternal effect in her offspring. Falconer made the simplification that the direct effect in a female is scaled to give the maternal effect in her offspring. The classic counterpart to this one-trait model is to assume that $M = z_m$, a general measure of maternal performance in her offspring (Dickerson 1947; Willham 1963, 1972; Cheverud 1984; Riska et al. 1985; Kirkpatrick and Lande 1989). Under this model, the trait (or more likely some composite index of traits) comprising z_m is not specified (and hence not directly scored in mothers), but BLUP allows us to estimate all its variance components (e.g., G_{mm}, G_{md}) given an appropriate pedigree design (Chapter 22). Assuming no direct selection on z_m, then $\beta_m = 0$, and by the construction of this maternal trait, $m = 1$ (as $M = z_m$), and the above response equations further simplify to

$$R_m = \left(G_{md} + \frac{1}{2}G_{mm}\right)\beta_d \tag{15.38a}$$

$$R_d = \left(G_{dd} + \frac{3}{2}G_{md} + \frac{1}{2}G_{mm}\right)\beta_d \tag{15.38b}$$

The assumption of selection only on the direct trait implies $\beta_d = S_d/P_{dd}$, giving

$$R_d = \left(h_d^2 + \frac{3}{2}\frac{G_{md}}{P_{dd}} + \frac{G_{mm}}{2P_{dd}}\right)S_d, \tag{15.38c}$$

showing that ignoring maternal effects results in potentially biased estimate of response. This is the **Dickerson-Willham model** of response in the presence of heritable maternal effects, which can also be expressed in terms of a **total heritability**

$$h_T^2 = \frac{G_{dd} + 1.5G_{md} + 0.5G_m}{P_{dd}} = \frac{\sigma^2(A_d) + 1.5\,\sigma(A_d, A_M) + 0.5\sigma^2(A_m)}{\sigma^2(z_d)} \tag{15.38d}$$

giving the response as

$$R_d = h_T^2 S_d. \tag{15.38e}$$

If $G_{md} > 0$, $h_T^2 > h_d^2$ and the direct heritability underestimates the total response (e.g., McAdam et al. 2002). Conversely, if G_{md} is sufficiently negative, $h_d^2 > h_T^2$, and the direct heritability overestimates the response (e.g., Wilson et al. 2005).

Maternal Selection vs. Maternal Inheritance

In addition to influencing the *phenotype* of their offspring, parents can potentially influence its *fitness* as well, with the offspring fitness being a function of both its own phenotype and

that of its parents. Indeed, this is simply a special case where the trait with the maternal effect is fitness. Kirkpatrick and Lande (1989) call this **maternal selection**, as opposed to **maternal inheritance** (what we have been calling maternal effects). One can have maternal selection without maternal inheritance and vise-versa.

Example 15.6. An example of maternal selection is offered by Karn and Penrose's (1951) classic work on human infant survival as a function of their birth weight. This trait is under stabilizing selection for an optimal weight, but Karn and Penrose also found that survivability was also a function of their gestation period (a maternal trait). Heavier offspring had higher survivability if they also experienced longer gestations, while lighter offspring had improved survivability if they experienced shorter gestations.

To see how the presence of maternal selection changes the response equations, consider the simplest case (analogous to the Falconer model) where the expected fitness of an individual (indexed by i) is a function of its current phenotype z_i and the phenotype of the same trait in its mother $z_{m,i}$. The multiple regression predicting its relative fitness (e.g., Equation 13.25b) becomes

$$w_i = 1 + \beta_d z_i + \beta_{m^*} z_{m,i} + e \tag{15.38a}$$

where β_{m^*} is expected change in relative fitness from a one unit change in the maternal phenotype while holding the offspring phenotype constant. Using the Robertson-Price identity (Equation 6.10), the selection differential S on this trait becomes

$$\begin{aligned} S &= \sigma\left(z_i, w_i\right) = \sigma\left(z_i, 1 + \beta_d z_i + \beta_{m^*} z_{m,i} + e\right) \\ &= \beta_d \sigma(z_i, z_i) + \beta_{m^*} \sigma(z_i, z_{m,i}) \end{aligned} \tag{15.38b}$$

where $\sigma(z_i, z_{m,i})$ is the phenotypic covariance between the trait in the mother and in her offspring. In the absence of maternal inheritance, $\sigma(z_i, z_{m,i}) = \sigma_A^2/2$, and the response becomes

$$R = h^2 S = h^2 \left(\beta_d \sigma_z^2 + \beta_{m^*} \sigma_A^2/2\right) = \sigma_A^2(\beta_d + \beta_{m^*}/2) \tag{15.38c}$$

The first term is the standard breeder's equation (Equation 13.8c), while the second term is the correlated change from selection on the mother's phenotype. Since one imagines that a significant fraction of the maternal effect on fitness occurs early in life, there is already selection on the direct trait, potentially even before it is expressed in the offspring due to the β_{m^*} (Kirkpatrick and Lande 1989).

If the trait experiences maternal inheritance, under the dilution model $\sigma(z_i, z_{m,i}) = m\sigma_A^2/(2-m)$, giving

$$S(t) = \beta_d(t)\sigma_z^2 + \beta_{m^*}(t) m\sigma_A^2/(2-m) \tag{15.38d}$$

which is used in the response dynamics under the dilution model (Kirkpatrick and Lande 1989). The multivariate extension of maternal selection is given by Kirkpatrick and Lande, and is examined in Volume 3.

One final clarification is in order, namely the important distinction between the gradient β_{m^*} on the maternal value in Equation 15.38a (maternal selection) and the gradient β_m on maternal effects (selection on a maternal inheritance *trait*) in the two-trait model (Equation 15.36). This distinction is most apparent by considering the case where the direct and maternally-selected traits are different, being z and ζ, respectively. For example, fitness

is a function of the height of an individual and the weight of its mother. The regression for relative fitness now becomes

$$w_i = 1 + \beta_d z_i + \beta_m \zeta_i + \beta_{m^*} \zeta_{m,i} + e \tag{15.39}$$

where z_i is the individual's phenotype at a direct trait, ζ_i its phenotype at the maternal trait, and $\zeta_{m,i}$ the phenotype of its mother at the maternal trait. Here, β_m represents the expected change in relative fitness given a one unit increase in an individual's maternal value holding its direct value and the maternal value of its mother constant. If $\beta_{m^*} = 0$, there is no maternal selection, but ζ may still be a trait with a maternal inheritance effect on the direct trait. In this case, a nonzero β_m implies that there is direct selection on this maternal trait, in addition to any selection ($\beta_d \neq 0$) on the direct trait.

This distinction between fitness consequences of the maternal trait value to an individual (β_m) and to her offspring (β_{m^*}) is critical — a maternal effect trait value may be harmful to an individual but helpful to its offspring, generating **antagonistic selection**. For example, having a large litter size is beneficial to a female, but harmful to her offspring, as individuals from smaller litters may have higher survivorship (Wilson et al. 2005). The issue deciding when a fitness component is assigned to an offspring versus a parent can be problematic (Wolf and Wade 2001), a point we consider further in Chapter 29.

Literature Cited

Bernardo, J. 1996. Maternal effects in animal ecology. *Amer. Zoo.* 36: 83–105. [15]

Bonduriansky, R., and T. Day. 2009. Nongenetic inheritance and its evolutionary implications. *Ann. REv. Ecol Evol. Syst.* 40: 103–125. [15]

Bulmer, M. G. 1971. The effect of selection on genetic variability. *Amer. Natl.* 105: 201–211. [15]

Bulmer, M. G. 1980. *The mathematical theory of quantitative genetics.* Oxford Univ. Press, NY. [15]

Cheverud, J. M. 1984. Evolution by kin selection: A quantitative genetic model illustrated by maternal performance in mice. *Evolution* 38: 766–777. [15]

Cockerham, C. C. 1984. Additive by additive variance with inbreeding and linkage. *Genetics* 108: 487–500. [15]

Crow, J. F. 2010. On epistasis: why it is unimportant in polygenic directional selection. *Phil. Trans. R. Soc. B* 365: 1241–1244. [15]

Dickerson, G. E. 1947. Composition of hog carcasses as influenced by heritable differences in rate and economy of gain. *Iowa Agr. Exp. Stat. Res. Bull.* 354: 492–524. [15]

Falconer, D. S. 1960. The genetics of litter size in mice. *J. Cell and Comp. Physiol.* 56 (Suppl 1): 153–167. [15]

Falconer, D. S. 1965. Maternal effects and selection response. *In* S. J. Geerts (ed.) *Genetics today, proceedings of the XI international congress on genetics, Vol 3* pp. 763–774. Pergamon, Oxford. [15]

Gallais, A. 1975. Selection with truncation in autotetraploids: Comparison with diploids. *Theor. Appl. Genet.* 46: 386–394. [15]

Gallais, A. 2003. *Quantitative Genetics and Breeding Methods in Autopolyploid Plants.* INRA, Paris. [15]

Griffing, B. 1960a. Theoretical consequences of truncation selection based on the individual phenotype. *Aust. J. Biol. Sci.* 13: 307–343. [15]

Griffing, B. 1960b. Accommodation of linkage in mass selection theory. *Aust. J. Biol. Sci.* 13: 501–526. [15]

Hagg, W. L., and R. R. Hill, Jr. 1974. Comparison of selection methods for autotetraploids. II. Selection for disease resistance in alfalfa. *Crop Sci.* 14: 591–593. [15]

Hill, R. R. Jr. 1971. Selection in autotetraploids. *Theor. Appl. Genet.* 41: 181–186. [15]

Hill, R. R., Jr., and W. L. Hagg. 1974. Comparison of selection methods for autotetraploids. I. Theoretical. *Crop Sci.* 14: 587–590. [15]

Hill, W. G., M. E. Goodard, and P. M. Vusscher. 2008. Data and theory point to mainly additive genetic variance for complex traits. *PLoS Genetics* 4: e1000008. [15]

Janssen, G. M., D. DeJong, E. N. G. Joose, and W. Scharloo. 1988. A negative maternal effect in springtails. *Evolution* 42: 828–834. [15]

Karn, M. N., and L. S. Penrose. 1951. Birth weight and gestation time in relation to maternal age, parity and infant survival. *Ann. Eugen.* 15: 206–233. [15]

Kempthorne, O. 1957. *An introduction to genetic statistics.* WIley and Sons, New York.

Kirkpatrick, M. and R. Lande. 1989. The evolution of maternal characters. *Evolution* 43: 485–503. [15]

Kirkpatrick, M., and R. Lande. 1992. The evolution of maternal characters: Errata. *Evol.* 46: 284. [15]

Lande, R., and M. Kirkpatrick. 1990. Selection response in traits with maternal inheritance. *Gene. Res.* 55: 189–197. [15]

Kuroda, R., B. Endo, M. Abe, and M. Shimiizu. 2009. Chiral blastomere arrangement dictates zygotic left–right asymmetry pathway in snails. *Nature* 462: 790–794. [15]

Lush, J. L. 1948. *The Genetics of Populations.* Animal Husbandry Department, Iowa State University, Ames. [15]

McAdam, A. G., and S. Boutin. 2003. Variation in viability selection among cohorts of juvenile red squirrels (*Tamiasciurus hudsonicus*). *Evol.* 57: 1689–1697. [15]

McAdam, A. G., S. Boutin, D. Réale, and D. Berteaux. 2002. Maternal effects and the potential for evolution in a natural population of animals. *Evol.* 56: 846–851. [15]

Mousseau, T. A., and C. W. Fox (eds). 1998. *Maternal effects as adaptations.* Oxford University Press, Oxford. [15]

Mousseau, T. A.,T. Uller, E. Wapstra, and A. V. Badyaev. 2009. Evolution of maternal effects: past and present. *Phil. Trans. R. Soc. B* 364: 1035–1038. [15]

Pearson, K. 1898. Mathematical contributions to the theory of evolution. On the law of ancestral heredity. *Proc. Roy. Soc. Lond.* 62: 386–412. [15]

Räsänen, K., and L. E. B. Kruuk. 2007. Maternal effects and evolution at ecological time-scales. *Func. Ecol.* 21: 408–421. [15]

Riska, B., J. J. Rutledge, and W. R. Atchley. 1985. Covariance between direct and maternal genetic effects in mice with a model of persistent environmental influences. *Genet. Res.* 45: 287–297. [15]

Reinhold, K. 2002. Maternal effects and the evolution of behavioral and morphological characters: a literature review indicates the importance of extended maternal care. *J. Hered.* 93: 400–405. [15]

Roach, D. A., and R. D. Wulff. 1987. Maternal effects in plants. *Ann. Rev. Ecol. Syst.* 18: 209–235. [15]

Rossiter, M. C. 1996. Incidence and consequences of inherited environmental effects. *Ann. Rev. Ecol. Syst.* 27: 451–476. [15]

Rowe, D. E., and R. R. Hill, Jr. 1984. Theoretical improvement of autotetraploid crops: Interpopulation and intrapopulation selection. U. S. Dept. of Agriculture, Technical Bulletin No 1689. [15]

Schluter, D., and L. Gustafsson. 1993. Maternal inheritance of condition and clutch size in the collared flycatcher. *Evol.* 47: 658–667. [15]

Swanson, M. R., J. W. Dudley, and S. G. Carmer. 1974. Simulated selection in autotetraploid populations. II: Effects of double reduction, population size, and selection intensity. *Crop. Sci.* 14: 630–636. [15]

Van der Steen, H. A. M. 1985. The implication of maternal effects for genetic improvement of litter size in pigs. *Livestock Prod. Sci.* 13: 159–168. [15]

Willham, R. L. 1963. The covariance between relatives for characters composed of components contributed by related individuals. *Biometrics* 19: 18–27. [15]

Willham, R. L. 1972. The role of maternal effects in animal breeding. II. biometrical aspects of maternal effects in animals. *J. Anim. Sci.* 35: 1288–1293. [15]

Wilson, A. J., D. W. Coltman, J. M. Pemberton, A. D. J. Overall, K. A. Byrne, and L. E. B. Kruuk. 2005. Maternal genetic effects set the potential for evolution in a free-living vertebrate population. *J. Evol. BIol.* 18: 405–414. [15]

Wilson, A. J., J. G. Pilkington, J. M. Pemberton, D. W. Coltman, A. D. J. Overall, K. A. Byrne, and L. E. B. Kruuk. 2005. Selection on mothers and offspring: whose phenotype is it and does it matter? *Evol.* 59: 451–463. [15]

Wilson, A. J., and D. Réale. 2006. Ontogeny of additive and maernal genetic effects: Lessons from domestic mammals. *Amer. Natl.* 167: E23–E38. [15]

Wolf, J. B., and M. J. Wade. 2001. On the assignment of fitness to parents and offspring: whose fitness is it and when does it matter? *J. Evol. BIol.* 14: 347–356. [15]

Wolf, J. B., and M. J. Wade. 2009. What are maternal effects (and what are they not)? *Phil. Trans. R. Soc. B* 364: 1107–1115. [15]

Wricke, G., and W. E. Weber. 1986. *Quantitative genetics and selection in plant breeding*. Walter de Gruyter and Co., New York. [15]