

# 13

## Short-term Changes in the Variance

*In artificial selection experiments it seems likely that the effects of linkage disequilibrium will be more important than the slower and less dramatic effects due to changes in gene frequencies — Michael Bulmer (1976a)*

*Draft version 20 February 2010*

While we have focused on how selection changes the mean of a character, it can also change the variance. In many cases, selection is expected to be acting more on the variance than the mean, for example as occurs with selection either for or against extreme individuals (disruptive and stabilizing selection, respectively). Even if our concern is only on the change in the mean, changes in the variance alter the heritability (modifying the response in the mean) and so must still be considered.

Selection can change the distribution of genotypic (and hence phenotypic) values by two routes: changing allele frequencies and/or generating gametic-phase disequilibrium. Under the infinitesimal model (Chapter 24), the amount of selection acting on any given locus is very small, and hence the expected change in allele frequencies over a few generations is also very small. However, even under the infinitesimal model, selection-induced changes in gametic-phase disequilibrium can cause rapid and significant changes in the additive genetic variance. Directional and stabilizing selection decrease the phenotypic variance, resulting in a reduction in  $\sigma_A^2$  due to the generation of negative disequilibrium (alleles that increase trait values become negatively correlated within a gamete). This reduction in the additive variance is often called the **Bulmer effect**, and we examine its consequences in this chapter. Conversely, disruptive selection can increase the phenotypic variance, and this generates positive disequilibrium, inflating the additive variance. Phenotypic assortative mating also generates disequilibrium, and we will consider all of these changes in variance under the infinitesimal model framework in this chapter. Chapter 24 continues this discussion, relaxing many of the infinitesimal assumptions (such as allowing for linkage and a finite number of loci).

Finally, there is a third route by which the phenotypic variance can change. Allele frequency changes and disequilibrium can modify the genetic variance, but if genotypes differ in their environmental variances then there can also be selection for genotypes with increased or decreased environmental variation. Thus, selection can change *both*  $\sigma_A^2$  and  $\sigma_E^2$ , and we conclude by considering selection on  $\sigma_E^2$ .

### CHANGES IN VARIANCE DUE TO GAMETIC-PHASE DISEQUILIBRIUM

In the absence of epistasis, gametic-phase disequilibrium does not change the population mean (Chapter 11). However, as pointed out by Lush (1945), it affects the response to selection by introducing correlations between alleles at different loci, altering the additive genetic variance  $\sigma_A^2$ . To see this, let  $a_1^{(k)}$  and  $a_2^{(k)}$  be the average effects of the two alleles at locus  $k$  from a random individual, where subscript one denotes the maternally-derived allele and subscript two denotes the paternal allele. We assume (for now) random mating, therefore

there is no covariance between alleles of maternal and paternal origin, so that  $\sigma(a_1^{(k)}, a_2^{(j)}) = 0$  for all  $k$  and  $j$ . However, when gametic-phase disequilibrium is present, there *can* be covariances between alleles at different loci from the same parent, so that  $\sigma(a_1^{(k)}, a_1^{(j)})$  and  $\sigma(a_2^{(k)}, a_2^{(j)})$  can be non-zero. That is, there can be correlations between alleles in any particular gamete. Since  $\sigma_A^2$  is the variance of the sum of average effects over all loci,

$$\sigma^2 \left( \sum_{k=1}^n (a_1^{(k)} + a_2^{(k)}) \right) = 2 \sum_{k=1}^n \sigma^2 (a^{(k)}) + 4 \sum_{k < j}^n \sigma (a^{(j)}, a^{(k)}) \quad (13.1a)$$

$$= 2 \sum_{k=1}^n C_{kk} + 4 \sum_{k < j}^n C_{jk} \quad (13.1b)$$

where  $n$  is the number of loci and  $C_{jk}$  is the covariance between allelic effects at loci  $j$  and  $k$  (when contributed by the same parent and hence on the same gamete).

We can thus decompose the additive variance as

$$\sigma_A^2 = \sigma_a^2 + d \quad (13.2)$$

where  $\sigma_a^2 = 2 \sum C_{kk}$  is the additive variance in the absence of gametic-phase disequilibrium and the disequilibrium contribution  $d = 4 \sum_{j < k} C_{jk}$  is the covariance between allelic effects at different loci (in terms of the notation used in LW Equation 7.14,  $d = \sigma_{A,A}$ ). The component of the additive genetic variance that is unaltered by changes in gametic-phase disequilibrium,  $\sigma_a^2$ , is often referred to as the **additive genic variance** (or simply the **genic variance**) to distinguish it from the additive genetic variance  $\sigma_A^2$ . In the absence of disequilibrium, the genic and genetic variances are equivalent. Negative disequilibrium ( $d < 0$ ) conceals the presence of additional additive variance, with  $\sigma_A^2$  increasing as the disequilibrium decays. If  $d > 0$ , additive variance is inflated relative to a random-mating population (i.e., the genic variance) and  $\sigma_A^2$  decreases as disequilibrium decays. Since  $n(n-1)$  terms contribute to  $d$  compared to  $n$  terms contributing to  $\sigma_a^2$ , gametic-phase disequilibrium can generate large changes in the additive genetic variance even when changes in these individual covariances are all very small.

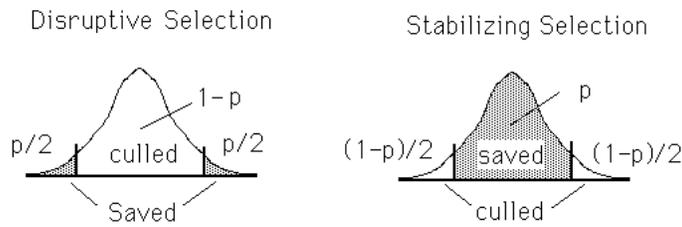
The allelic effects  $a^{(k)}$  (and hence the genic variance  $\sigma_a^2$ ) are altered as allele frequencies change, resulting in a permanent change in  $\sigma_a^2$ . Changes in  $\sigma_a^2$  due to selection strongly depend on the initial distribution of allelic effects and frequencies (Chapters 5, 24), both of which are extremely difficult to estimate. Changes in  $d$ , however, are generally less sensitive to the initial distribution of allelic effects (Sorensen and Hill 1982). Unlike changes in  $\sigma_a^2$ , changes in  $\sigma_A^2$  due to changes in  $d$  are *transient* — in the absence of selection, recombination removes disequilibrium and the additive genetic variance  $\sigma_A^2$  approaches the additive genic variance  $\sigma_a^2$  as  $d$  decays to zero.

Under our assumption that allele frequencies remain constant, changes in  $\sigma_A^2$  are due entirely to changes in  $d$ . Hence, the additive genetic variance at generation  $t$  is given by  $\sigma_A^2(t) = \sigma_a^2 + d(t)$ , with change in (additive) variance  $\Delta\sigma_A^2(t) = \Delta d(t)$ . Under random mating in the absence of selection, the disequilibrium between pairs of unlinked loci is halved each generation (LW Equation 5.12), halving the covariance between allelic effects, so that

$$d(t+1) = \frac{d(t)}{2} \quad (13.3)$$

Thus, with unlinked loci, recombination decreases the disequilibrium each generation by  $d(t)/2$ . Countering this, selection tends to generate gametic-phase disequilibrium. Directional selection to change the mean of a character also reduces its variance, generating

negative  $d$ . Selection can also occur strictly on variances, resulting in a change in the phenotypic variance without a change in the mean (Chapters 28, 29). **Stabilizing selection** (selection against extreme individuals, see Figure 13.1) reduces the phenotypic variance and creates negative  $d$ , while **disruptive selection** (selection for extreme individuals, see Figure 13.1) increases the phenotypic variance, generating positive  $d$ . Stabilizing and disruptive selection are occasionally referred to as **centripetal** and **centrifugal selection**, respectively (Simpson 1944). How these within-generation changes in the variance translate into between-generation changes has been worked out for the infinitesimal model by Bulmer (1971, 1974, 1976a, 1980), whose approach we follow closely. Chapter 24 moves beyond the infinitesimal model by considering some of these issues in the presence of both linkage and a finite number of loci. Estimation of the nature and amount of selection acting on the mean and the variance of a character is covered in Chapters 28 and 29.



**Figure 13.1** Stabilizing and disruptive selection using **double truncation**. In both cases, a fraction  $p$  of the population is allowed to reproduce. In stabilizing selection, the central  $p$  of the distribution is saved, while under disruptive selection the uppermost and lowermost  $p/2$  are saved.

**CHANGES IN VARIANCE UNDER THE INFINITESIMAL MODEL**

Since allele frequencies remain constant under the infinitesimal model, the additive genic variance  $\sigma_a^2$  remains constant and all changes in the additive genetic variance  $\sigma_A^2$  are due to changes in  $d$ . Assuming the population is initially in gametic-phase equilibrium,  $\sigma_A^2(0) = \sigma_a^2$ , giving  $\sigma_A^2(t) = \sigma_a^2 + d(t)$ . Likewise, let  $h^2(t)$  and  $\sigma_z^2(t)$  be the heritability and phenotypic variance before selection in generation  $t$ , and  $h^2$  and  $\sigma_z^2$  be the values of these quantities in the absence of gametic-phase disequilibrium.

Under the infinitesimal model, gametic-phase disequilibrium does not change the dominance variance (Bulmer 1971). To see this, first note from LW Equation 5.16b that the disequilibrium contribution with a finite number of loci ( $n$ ) to dominance variance is of order  $n^2 \bar{D}^2$ , where  $\bar{D}$  is the average pairwise disequilibrium. Under the infinitesimal model, the total disequilibrium (summing over all pairs of loci) remains bounded as the number of loci increases, implying  $\bar{D}$  is of order  $n^{-2}$  since there are  $n(n - 1)/2 \approx n^2/2$  pairs of loci contributing to  $\bar{D}$ . The disequilibrium contribution to dominance variance is thus of order  $n^2(n^{-2})^2 = n^{-2}$ , which converges to zero in the infinitesimal model limit (as the number of loci  $n \rightarrow \infty$ ). With this result in mind, in the absence of any epistatic variance, genotype  $\times$  environment interactions and/or correlations, the phenotypic variance and heritability at generation  $t$  become

$$\sigma_z^2(t) = \sigma_E^2 + \sigma_D^2 + \sigma_A^2(t) = \sigma_z^2 + d(t) \tag{13.4a}$$

$$h^2(t) = \frac{\sigma_A^2(t)}{\sigma_z^2(t)} = \frac{\sigma_a^2 + d(t)}{\sigma_z^2 + d(t)} \tag{13.4b}$$

where  $\sigma_z^2 = \sigma_z^2(0)$  is the phenotypic variance before selection in the initial (unselected) population. Thus, predicting the value of  $d(t)$  is sufficient to predict changes in the variances, and hence the heritability and the response in the mean using  $R(t) = h^2(t)S(t)$ .

Under the infinitesimal model, genotypic values are normally distributed before selection (Bulmer 1971, 1976b). Recalling  $z = G + E$ , we see that if the environmental values  $E$  are also normally distributed, then the joint distribution of phenotypic and genotypic values is multivariate normal. Hence, from standard statistical theory (e.g., LW Chapter 8), the regression of offspring phenotypic value  $z_o$  on parental phenotypes  $z_m$  and  $z_f$  is linear and homoscedastic, with

$$z_o = \mu + \frac{h^2}{2}(z_m - \mu) + \frac{h^2}{2}(z_f - \mu) + e \quad (13.5a)$$

where

$$\sigma_e^2 = \left(1 - \frac{h^4}{2}\right) \sigma_z^2 \quad (13.5b)$$

The derivation of Equation 13.5 follows from standard multiple regression theory and the correlations between relatives (see Example 6 in LW Chapter 8 for details).

Denote the within-generation change in variance by  $\delta(\sigma_z^2) = \sigma_{z^*}^2 - \sigma_z^2$ , where  $z^*$  refers to a phenotypic value from the selected population. Throughout this chapter we use the notation of  $\delta$  to distinguish the *within-generation* change of a variable from its *between-generation* change  $\Delta$ , the latter incorporating the effects of genetic transmission across a generation. Taking variances of both sides of Equation 13.5a and assuming random mating and identical selection on both sexes, the phenotypic variance among the offspring from selected parents becomes

$$\begin{aligned} \sigma^2(z_o) &= \frac{h^4}{4} [\sigma^2(z_m^*) + \sigma^2(z_f^*)] + \sigma_e^2 \\ &= \frac{h^4}{2} [\sigma_z^2 + \delta(\sigma_z^2)] + \left(1 - \frac{h^4}{2}\right) \sigma_z^2 \\ &= \sigma_z^2 + \frac{h^4}{2} \delta(\sigma_z^2) \end{aligned} \quad (13.6)$$

The change in phenotypic variance in the offspring due to selection on their parents generating disequilibrium is thus  $(h^4/2) \delta(\sigma_z^2)$ . Since there is no change in the environmental, dominance, or genic variances, this change is all in the disequilibrium component  $d$  of the additive genetic variance  $\sigma_A^2$ . Combining Equations 13.6 and 13.3 gives the general recursion for changes in the variance under the infinitesimal model with unlinked loci as

$$d(t+1) = \frac{d(t)}{2} + \frac{h^4(t)}{2} \delta(\sigma_{z(t)}^2) \quad (13.7a)$$

This result is due to Bulmer (1971) and implies that the between-generation change in the disequilibrium contribution is

$$\begin{aligned} \Delta d(t) &= \Delta \sigma_{z(t)}^2 = \Delta \sigma_A^2(t) \\ &= -\frac{d(t)}{2} + \frac{h^4(t)}{2} \delta(\sigma_{z(t)}^2) \end{aligned} \quad (13.7b)$$

The first term is the decay due to recombination in the disequilibrium contribution from the previous generation (assuming unlinked loci), while the second term is the amount of new disequilibrium generated by selection that is passed on to the offspring. Note that

if we start from a base population in equilibrium ( $d(0) = 0$ ) that the sign of the within-generation change in the variance equals the sign of  $d$ . Thus, selection that *decreases* the phenotypic variance generate negative  $d$ , while selection that *inflates* the variance generates positive  $d$ . As previously mentioned, this change in the variance (typically a reduction) due to selection generating disequilibrium is often called the Bulmer effect. Equation 13.7 is the variance response analogue of the breeders' equation. Provided the joint distribution of phenotypic and genotypic values remains multivariate normal, under the infinitesimal model the complete dynamics of the phenotypic distribution are jointly described by the change in the variance (Equation 13.7) and the change in the mean [given from the modified breeders' equation,  $R(t) = h^2(t)S(t)$ ]. Equation 13.7 makes the further point that if we wish to use variance components to predict the response to selection, we need to start from an *unselected base population*. If a population has been experiencing previous selection, then  $d \neq 0$  and hence the change in  $\sigma_A^2$  (and, in turn, the response to selection) cannot be predicted without knowing the value of  $d$  in the starting population. Finally, we note that, at equilibrium,

$$\tilde{d} = \tilde{h}^4 \tilde{\delta}(\sigma_z^2) \quad (13.7c)$$

where tilde denotes an equilibrium value.

**Example 13.1.** Data of Rendel (1943) suggests stabilizing selection occurs on egg weight in ducks. Of 960 eggs followed, 64.5% hatched. The change in mean egg weight (in grams) after selection was negligible, but the variance showed a significant decrease. The variance was 52.7 before selection (using all 960 eggs) and 43.9 after selection (in those eggs that hatched), giving  $\delta(\sigma_z^2) = -8.8$ . Assuming that the reduction in variance due to gametic-phase disequilibrium is at equilibrium and taking  $\tilde{h}_z^2 = 0.60$  (the heritability for egg weight in poultry) gives

$$\tilde{d} = \tilde{h}^4 \tilde{\delta}(\sigma_z^2) = (0.6)^2(-8.8) = -3.2$$

and

$$\tilde{\sigma}_A^2 = \tilde{h}^2 \tilde{\sigma}_z^2 = 0.6 \cdot 52.7 = 31.6$$

Assuming the infinitesimal model, if selection is stopped, the additive variance is expected to eventually increase to

$$\sigma_A^2 = \sigma_a^2 = \tilde{\sigma}_A^2 - \tilde{d} = 31.6 + 3.2 = 34.8$$

with half this change being accomplished in one generation (assuming all underlying loci are unlinked). Similarly,  $\sigma_z^2 = 52.7 + 3.2 = 55.9$  and  $h^2 = 34.8/55.9 = 0.62$ . (Example slightly modified from Bulmer 1971.)

### Within- and Between-Family Variance Under the Infinitesimal Model

An alternative, and instructive, approach to the phenotypic regression argument leading to Equation 13.7 is to instead consider the regression of offspring breeding value ( $A_o$ ) on the breeding values of its parents ( $A_f, A_m$ ). Under the infinitesimal model, the joint distribution of parental and offspring breeding values before selection is multivariate normal (Bulmer 1971), and Example 7 in Chapter 8 of LW shows that the distribution of breeding values in the offspring of parents with breeding values  $A_f$  and  $A_m$  is given by the regression

$$A_o = \frac{1}{2}A_m + \frac{1}{2}A_f + e \quad (13.8a)$$

The residual  $e$  is the contribution due to segregation, which is normally distributed with mean zero and variance  $\sigma_a^2/2 = \sigma_A^2(0)/2$ , half the additive genetic variance present in the absence of disequilibrium (Bulmer 1971, Felsenstein 1981, Tallis 1987). Since  $e$  is the residual of a regression, it is uncorrelated with both  $A_f$  and  $A_m$  (LW Chapters 3, 8). Taking variances and assuming random mating (so that  $A_f$  and  $A_m$  are uncorrelated),

$$\begin{aligned}\sigma_{A_o}^2(t+1) &= \sigma_{A_o}^2(t+1) = \sigma^2 \left( \frac{A_m(t)}{2} + \frac{A_f(t)}{2} \right) + \sigma_e^2 \\ &= \frac{1}{4} \left[ \sigma_{A_m}^2(t) + \sigma_{A_f}^2(t) \right] + \frac{1}{2} \sigma_A^2(0) \\ &= \frac{1}{2} \sigma_{A^*}^2(t) + \frac{1}{2} \sigma_a^2\end{aligned}\tag{13.8b}$$

where  $\sigma_{A^*}^2(t)$  is the variance of the breeding values of the selected parents [with assortative mating, Equation 13.8b has an additional term,  $\sigma(A_m^*, A_f^*)/2$ , see Equation 13.26b]. Equation 13.8b shows that additive variance can be decomposed into a *between-family* component [half the additive *genetic* variance,  $\sigma_{A^*}^2(t)/2$ ] that measures the differences between the mean breeding values of families and a *within-family* component [half the additive *genic* variance,  $\sigma_a^2(0)/2 = \sigma_a^2/2$ ] due to segregation that measures the variation within families. Equations 13.8a and b imply that under the infinitesimal model, *the within-family additive variance remains constant*. The change in the population additive genetic variance is entirely due to changes in the variance of the mean values of different families. Positive disequilibrium ( $d > 0$ ) increases the between-family component while negative disequilibrium ( $d < 0$ ) decreases it (Reeve 1953).

The within-family variance,  $\sigma_a^2/2$  deserves additional comment. This is often called the **Mendelian sampling variance**. Notice that this variance (under the infinitesimal model) is *not* affected by selection, as we assume negligible change in allele frequencies. As we will see shortly, it can be decreased by drift and/or inbreeding. Likewise, with a finite number of loci  $\sigma_a^2$  can indeed be affected by selection, but the change per generation is typically very small (Chapter 24) An especially important implication of this constant within-family segregation variance is that it tends to largely restore a normal distribution of breeding values following selection. As Equation 13.8a demonstrates, the distribution of breeding values in the offspring is the sum of two components: the breeding values of the selected parents plus the contribution due to segregation. Even if the distribution of breeding values in the selected parents departs significantly from normality, segregation tends to reduce this departure. Interestingly, Smith and Hammond (1987) found that the short-term deviation from normality is largest when selection is moderate, becoming smaller as selection increases. This can be seen from Equation 13.8a by writing  $A_o = A_{mp} + e$ , where  $A_{mp}$  is the midparental breeding value and  $e$  the contribution due to segregation. Under the assumption that  $e$  is normally distributed, as selection intensity increases, the variance of  $A_{mp}$  decreases, and more and more of the variance of  $A_o$  is accounted for by  $e$ , decreasing the departure from normality.

The derivation of Equations 13.7 and 13.8a requires the assumption that breeding values remain normally distributed. Selection and/or assortative mating, however, cause the population to deviate from normality by creating gametic-phase disequilibrium that generates skew and kurtosis in the distribution of breeding values. If selection changes the distribution of breeding values away from normality, the parent-offspring regression may no longer be linear and homoscedastic (so that the residual variance  $\sigma_e^2$  may now depend on the breeding values). Consequences of departures from linearity were briefly discussed in Chapter 10 and are developed more fully in Chapter 24. Heteroscedasticity of the residuals implies that  $\sigma_e^2$  depends on the actual parental values chosen, which greatly complicates matters. For example, the within-family variance in such cases is no longer a constant independent of the

parental breeding values, potentially reducing the effectiveness of segregation in restoring normality. In our discussions below, we assume that these departures from normality can be ignored. Chapter 24 works at relaxing these assumptions.

### Accounting Inbreeding and Drift

The effects of drift and regular inbreeding are easily accommodated by the infinitesimal model (Verrier et al. 1989). The segregation variation (the variance in the residual  $e$  in Equation 13.8a) is just half the additive genetic variance of the parents. When genetic drift is present, the genic variance in generation  $t$  becomes

$$\sigma_a^2(t) = \sigma_a^2(0) \left(1 - \frac{1}{2N_e}\right)^t \quad (13.9a)$$

while Keightley and Hill (1987) show that drift modifies the disequilibrium by

$$\Delta d(t) = -\frac{d(t)}{2} \left(1 + \frac{1}{N_e}\right) - \frac{1}{2} \left(1 - \frac{1}{N_e}\right) \kappa h^2(t) \sigma_A^2(t) \quad (13.9b)$$

where  $\kappa$  is the fractional reduction in variance following selection (see Equation 13.10). Hence, when the population size is finite, the variance in any particular generation can be computed by jointly iterating Equations 13.9a and b. Using these updated values for the additive variance and heritability, one can then use the breeders' equation to predict the change in mean.

Similarly, when the parents are inbred, the segregation variance is also correspondingly reduced. Assuming no correlation between the parents, the within-family segregation variance is

$$\sigma_a^2(t) = \frac{\sigma_a^2(0)}{2} \left[1 - \frac{f_m(t) + f_f(t)}{2}\right] \quad (13.9c)$$

where  $f_m$  and  $f_f$  denote the average amount of inbreeding in selected male and female parents. Thus the additive variance recursion equation under the infinitesimal model assumptions becomes

$$\sigma_A^2(t+1) = \frac{1}{4} \left[\sigma_{A_m}^2(t) + \sigma_{A_f}^2(t)\right] + \frac{\sigma_a^2(0)}{2} \left[1 - \frac{f_m(t) + f_f(t)}{2}\right] \quad (13.9d)$$

## CHANGES IN VARIANCE UNDER TRUNCATION SELECTION

Provided the normality assumptions of the infinitesimal model hold, the changes in variance under any selection model can be computed by obtaining the within-generation change in the phenotype variance,  $\delta(\sigma_{z(t)}^2)$ , and applying Equation 13.7. In the general case, this requires numerical iteration to obtain the equilibrium heritability and genetic variance. However, in many cases phenotypic variance after selection can be written as

$$\sigma_{z^*}^2 = (1 - \kappa) \sigma_z^2 \quad (13.10a)$$

implying

$$\delta(\sigma_z^2) = \sigma_{z^*}^2 - \sigma_z^2 = -\kappa \sigma_z^2 \quad (13.10b)$$

where  $\kappa$  is a constant independent of the variance. When Equation 13.10 holds (implying a constant proportionate reduction in variance), simple analytic solutions for the equilibrium

variances and heritability can be obtained. Truncation selection — both as we have defined it for directional selection (Chapter 10) and **double truncation** giving disruptive or stabilizing selection (Figure 13.1) — satisfies Equation 13.10. As shown in Table 13.1, for truncation selection on a normally-distributed phenotype,  $\kappa$  is strictly a function of the fraction  $p$  of the population saved and the type of truncation selection used.

**Table 13.1** Changes in the phenotypic variance under the various schemes of single- and double-truncation given in Figure 13.1. Assuming the character is normally distributed before selection, the phenotypic variance after selection is given by  $\sigma_z^{2*} = (1 - \kappa) \sigma_z^2$ , where  $\kappa$  is a function of the fraction  $p$  of individuals saved. Here  $\varphi$  denotes the unit normal density function and  $x_{[p]}$  satisfies  $\Pr(U \leq x_{[p]}) = p$  [so that  $\Pr(U > x_{[1-p]}) = p$ ], where  $U$  is a unit normal random variable. Finally,  $\bar{i}$  is the selection intensity (Equation 10.26b). While first presented in the quantitative genetics literature by Bulmer (1976a), derivations for these expressions can be found in Johnson and Kotz (1970).

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**Directional Truncation Selection:** Uppermost (or lowermost)  $p$  saved

$$\kappa = \frac{\varphi(x_{[1-p]})}{p} \left( \frac{\varphi(x_{[1-p]})}{p} - x_{[1-p]} \right) = \bar{i} (\bar{i} - x_{[1-p]})$$

**Stabilizing Truncation Selection:** Middle fraction  $p$  of the distribution saved

$$\kappa = \frac{2 \varphi(x_{[1/2+p/2]}) x_{[1/2+p/2]}}{p}$$

**Disruptive Truncation Selection:** Uppermost and lowermost  $p/2$  saved

$$\kappa = -\frac{2 \varphi(x_{[1-p/2]}) x_{[1-p/2]}}{p}$$


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Suppose selection is such that Equation 13.10 is satisfied. We allow for differential selection on the sexes by letting the variance after selection in mothers and fathers be  $\sigma^2(z_m^*) = (1 - \kappa_m) \sigma_z^2$  and  $\sigma^2(z_f^*) = (1 - \kappa_f) \sigma_z^2$ , respectively. If parental phenotypes are uncorrelated (i.e., random mating),

$$\sigma_{z^*}^2(t) = \frac{\sigma^2(z_f^*(t))}{2} + \frac{\sigma^2(z_m^*(t))}{2} = (1 - \kappa) \sigma_z^2(t) \quad (13.11a)$$

where

$$\kappa = \frac{\kappa_f + \kappa_m}{2} \quad (13.11b)$$

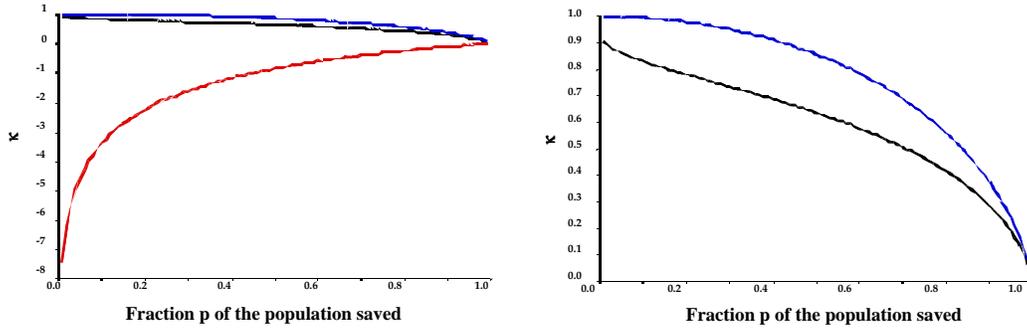
Hence, the within-generation change in the variance due to selection becomes

$$\delta(\sigma_z^2(t)) = -\kappa \frac{\sigma_A^2(t)}{h^2(t)} \quad (13.11c)$$

where we have used the identity  $\sigma_z^2 = \sigma_A^2/h^2$ . Substituting Equation 13.11c into Equation 13.7a recovers the result of Bulmer (1974),

$$\begin{aligned} d(t+1) &= \frac{d(t)}{2} - \frac{\kappa}{2} h^2(t) \sigma_A^2(t) \\ &= \frac{d(t)}{2} - \frac{\kappa}{2} \frac{[\sigma_a^2 + d(t)]^2}{\sigma_z^2 + d(t)} \end{aligned} \quad (13.12)$$

The last step follows from  $h^2 \sigma_A^2 = \sigma_A^4 \sigma_z^2$ .



**Figure 13.2** The fractional reduction  $\kappa$  of phenotypic variance removed by truncation selection (Figure 13.1) as a function of the fraction  $p$  of individuals saved. Following selection, the new variance is  $(1 - \kappa)\sigma_z^2$ . **Left:** The lowest-most curve (values of  $\kappa < 0$ ) corresponds to disruptive selection (and hence an *increase* in the variance), while the upper two curves ( $\kappa > 0$ ) correspond to directional (middle curve) and stabilizing selection (upper curve) and hence a decrease in the variance. **Right:** Closeup for directional (lower curve) and stabilizing selection (upper curve).

At equilibrium,  $\tilde{d} = -\kappa \tilde{h}^2 \tilde{\sigma}_A^2$ . Since  $\tilde{\sigma}_A^2 = \sigma_a^2 + \tilde{d}$  and  $\tilde{h}^2 = (\sigma_a^2 + \tilde{d})/(\sigma_z^2 + \tilde{d})$ , we have

$$\tilde{d} = -\kappa \frac{(\sigma_a^2 + \tilde{d})^2}{\sigma_z^2 + \tilde{d}}$$

This is a quadratic equation in  $\tilde{d}$  which has one admissible solution (the constraint being that  $\tilde{\sigma}_A^2 \geq 0$ ). Solving gives

$$\tilde{\sigma}_A^2 = \sigma_z^2 \theta, \quad \text{where} \quad \theta = \frac{2h^2 - 1 + \sqrt{1 + 4h^2(1 - h^2)\kappa}}{2(1 + \kappa)} \quad (13.13a)$$

Since  $\tilde{\sigma}_A^2 - \sigma_A^2 = \tilde{d}$ , we can write

$$\tilde{\sigma}_z^2 = \sigma_z^2 + (\tilde{\sigma}_A^2 - \sigma_A^2) = \sigma_z^2(1 + \theta - h^2) \quad (13.13b)$$

it follows that the equilibrium heritability is

$$\tilde{h}^2 = \frac{\tilde{\sigma}_A^2}{\tilde{\sigma}_z^2} = \frac{\theta}{1 + \theta - h^2} \quad (13.13c)$$

We can also (Gomez-Raya and Burnside 1990) express the equilibrium heritability as

$$\tilde{h}^2 = \frac{-1 + \sqrt{1 + 4h^2(1 - h^2)\kappa}}{2\kappa(1 - h^2)} \quad (13.13d)$$

### Changes in a Correlated Character

Suppose the joint distribution of phenotypic values for our trait under selection  $z$  and two other correlated traits  $x$  and  $y$  is multivariate normal. If the within-generation change in the phenotypic values of  $z$  is given by Equation 13.10, then classic results (Pearson 1903) for the

multivariate distribution imply that the variance in  $x$  following selection on (only)  $z$  is given by

$$\sigma^2(x^*) = (1 - \kappa \rho_{x,z}^2) \sigma^2(x) \quad (13.14a)$$

implying

$$\delta [\sigma^2(x)] = -\kappa \rho_{x,z}^2 \sigma^2(x) \quad (13.14b)$$

where  $\rho_{x,z}$  is the correlation between traits  $x$  and  $z$ . Thus if selection reduces the variance in  $x$  ( $0 < \kappa < 1$ ), then the variance in any correlated character is also reduced, *independent* of the sign of the correlation. Likewise, the covariance between  $x$  and  $y$  following selection on  $z$  is given by

$$\sigma(x^*, y^*) = \sigma(x, y) - \kappa \frac{\sigma(x, z)\sigma(y, z)}{\sigma_z^2} \quad (13.14c)$$

and hence

$$\delta [\sigma(x, y)] = -\kappa \frac{\sigma(x, z)\sigma(y, z)}{\sigma_z^2} \quad (13.14d)$$

These results will prove useful when we consider the Bulmer effect for multivariate traits (Chapter 31), such as selection on an index (Chapters 32-33), or selection using BLUP (Chapter 34).

#### Directional Truncation Selection: Theory

Directional truncation results in a reduction in the phenotypic variance following selection, and hence generates negative  $d$  and a corresponding reduction in the additive variance and heritability. When the trait is normally-distributed, recalling LW Equation 2.15 and our Equation 10.26a gives

$$\sigma_{z^*}^2 = [1 - \bar{i} (\bar{i} - x_{[1-p]})] \sigma_z^2 \quad (13.15a)$$

and hence

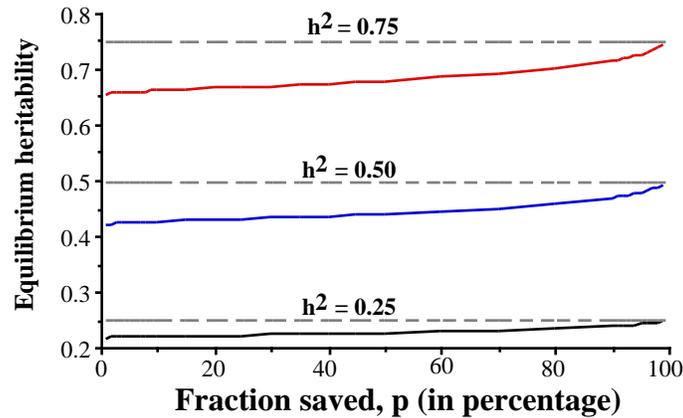
$$\kappa = \bar{i} (\bar{i} - x_{[1-p]}) \quad (13.15b)$$

The stronger the selection (i.e., the smaller the value of  $p$  and hence the larger the value of  $\bar{i}$ ), the larger the amount of disequilibrium generated and the greater the reduction in additive variance. Since the response to selection depends on the additive genetic variance in the selected *parents*, the response to selection in the first generation is unaffected (assuming the parents from the base population are in gametic-phase equilibrium). However, in the next two or three generations, essentially all of the reduction in  $h^2$  due to buildup of negative  $d$  occurs, after which heritability remains constant (see Example 13.2). Equations 13.13a-d give the equilibrium (or **asymptotic**) variances and heritabilities. The ratio of the asymptotic to initial (assuming  $d = 0$ ) response is given by

$$\frac{\tilde{R}}{R(0)} = \frac{\tilde{i} \tilde{h} \tilde{\sigma}_A}{\bar{i} h(0) \sigma_A(0)} = \sqrt{\frac{\tilde{h}^2}{h^2(0) [1 + \kappa \tilde{h}^2]}} \quad (13.16)$$

as obtained by Gomez-Raya and Burnside (1990).

Following the relaxation of selection,  $h^2$  is expected to increase as disequilibrium decays. The equilibrium heritabilities are a function of the initial heritability and the fraction of the population saved. As shown in Figure 13.3, the reduction in heritability is greatest when selection is strongest (i.e., when the fraction saved  $p$  is small) and heritability is high.



**Figure 13.3.** Equilibrium heritability values under directional (truncation) selection as a function of the fraction  $p$  saved and the initial heritability  $h^2$ . The three curves correspond to initial heritability values of 0.75, 0.5, and 0.25.

**Example 13.2.** Suppose directional truncation selection is performed (equally on both sexes) on a normally distributed character with  $\sigma_z^2 = 100$ ,  $h^2 = 0.5$ , and  $p = 0.20$  (the upper 20 percent of the population is saved). From normal distribution tables,

$$\Pr(U \leq 0.84) = 0.8, \quad \text{hence} \quad x_{[0.8]} = 0.84$$

Likewise, evaluating the unit normal gives  $\varphi(0.84) = 0.2803$ , so that (Equation 10.26a)

$$\bar{i} = \varphi(0.84)/p = 0.2803/0.20 = 1.402$$

From Equation 13.15b, the fraction of variance removed by selection is

$$\kappa = 1.402(1.402 - 0.84) = 0.787.$$

Hence, Equation 13.12 gives

$$d(t + 1) = \frac{d(t)}{2} - 0.394 \frac{[50 + d(t)]^2}{100 + d(t)}$$

Starting selection in generation 0 on a base population in gametic-phase equilibrium,  $d(0) = 0$ , iteration yields

Generation	0	1	2	3	4	5	$\infty$
$d(t)$	0.00	-9.84	-11.96	-12.45	-12.56	-12.59	-12.59
$\sigma_A^2(t)$	50.00	40.16	38.04	37.55	37.44	37.41	37.41
$h^2(t)$	0.50	0.45	0.43	0.43	0.43	0.43	0.43

Essentially all of the decline in additive variance occurs in the first three generations.

An important point to note is that the within-generation reduction in the variance is close to 80%, but the resulting decrease in the phenotypic variance (at equilibrium) is  $100 - 12.59 = 87.41$ , only a 13% decrease. Thus, as we found for the response in mean using the breeders' equation, only a fraction of the within-generation response is transmitted between generations. With the change in mean, this arises because only a fraction of the variation is due to additive-genetic variance. This is a contributing fraction with the change in variance as well, but a new complication is the additional variation generated by mendelian sampling each generation, partly mitigating the impact of selection in decreasing the variance.

We can also obtain these equilibrium additive variance directly from Equation 13.13a, where  $\tilde{\sigma}_A^2 = \sigma_z^2 \theta = 100 \theta$ , where

$$\theta = \frac{2 \cdot 0.5 - 1 + \sqrt{1 + 4 \cdot 0.5 \cdot (1 - 0.5) \cdot 0.787}}{2(1 + 0.787)} = 0.374$$

and hence  $\tilde{\sigma}_A^2 = 37.4$ , as found by iteration. Likewise, Equation 13.13c gives the equilibrium heritability at

$$\tilde{h}^2 = \frac{\theta}{1 + \theta - h^2} = \frac{0.374}{1 + 0.374 - 0.5} = 0.43$$

Again, this matches the value found by iteration.

How does this reduction in  $\sigma_A^2$  influence the per-generation change in mean,  $R(t)$ ? Since the selection  $\bar{i}$  is unchanged (being entirely a function of the fraction  $p$  of adults saved), but  $h^2$  and  $\sigma_z^2$  change over time, Equation 10.6b gives the response as

$$R(t) = h^2(t) \bar{i} \sigma_z(t) = 1.402 h^2(t) \sqrt{\sigma_z^2 + d(t)} = 1.402 h^2(t) \sqrt{100 + d(t)}$$

Response declines from an initial value of  $R = 1.4 \cdot 0.5 \cdot 10 = 7$  to an asymptotic per-generation value of  $\bar{R} = 1.4 \cdot 0.43 \cdot \sqrt{87.41} = 5.6$ . Thus if we simply used the Breeders' equation to predict change in mean over several generations without accounting for the Bulmer effect, we would have *overestimated* the expected response by 25 percent.

### Directional Truncation Selection: Experimental Results

How well do these predictions hold up for directional selection? Somewhat surprisingly, not many experiments have directly examined these issues. One reason is that the predicted change in  $h^2$  under directional selection is usually expected to be small (Figure 13.3) and hence laborious to detect (requiring very large sample sizes). One indirect study is that of Atkins and Thompson (1986), who subjected blackface sheep to selection for increased bone length. Following 18 years of selection, realized heritability was estimated to be  $0.52 \pm 0.02$ . Using the infinitesimal model, they predicted the expected base population heritability should be 0.57, in agreement with the estimated base population heritability of  $0.56 \pm 0.04$ . Further, the infinitesimal model predicts a 10% decrease in phenotypic variance. The observed values were a 9% decrease in the upwardly-selected line and an 11% decrease in the downwardly-selected line.

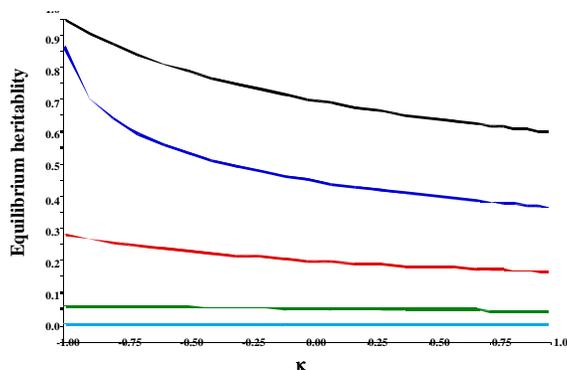
**Table 13.2** Heritability and additive genetic variance in an experimental population undergoing directional selection on abdominal bristle number in *Drosophila melanogaster*. The base population is denoted by B. At the third generation of selection (H3) and the following three generations of relaxed selection (C7, in generation 7),  $h^2$  was estimated from the response to divergent selection (Chapter 14) and  $\sigma_A^2$  subsequently estimated by  $\hat{h}^2 \sigma_z^2$ . The standard error for  $\hat{h}^2$  in all cases was 0.04. From Sorensen and Hill (1982).

	$\hat{h}^2(t)$			$\hat{\sigma}_A^2(t)$		
	B	H3	C7	B	H3	C7
Replicate 1	0.42	0.45	0.59	3.63	5.83	7.66
Replicate 2	0.38	0.26	0.26	2.96	2.28	2.08

A more direct study is that of Sorensen and Hill (1982), who subjected two replicate lines of *Drosophila melanogaster* to directional truncation selection on abdominal bristle number for four generations and then relaxed selection (Table 13.2). They interpreted their data as being consistent with the presence of a major allele (or alleles) at low frequency in the base population. These alleles are lost by sampling accidents in some replicates (e.g., replicate 2 which shows no net increase in additive variance). If not lost, they are expected to increase rapidly in frequency due to selection, increasing additive variance (replicate 1), with this increase being partly masked by generation of negative disequilibrium with other loci. Once selection stops, disequilibrium breaks down, resulting in a further increase in additive variance (compare the additive variance in lines H3 and C7 in replicate 1). Hence, even when major alleles are present, generation of gametic-phase disequilibrium reduces the rate of selection response.

### Effects of Epistasis: Does the Griffing Effect Overpower the Bulmer Effect?

As discussed in Chapter 11, Griffing (1960a,b) showed when additive epistasis is present, gametic-phase disequilibrium increases the response to directional selection, with the response augmented by  $S\sigma_{AA}^2/2\sigma_z^2$ . This (transient) increase in rate of response has been termed the **Griffing effect**. Thus, in the presence of additive epistasis, disequilibrium is on one hand expected to increase the rate of response, while it is also expected to decrease the rate of response by decreasing additive genetic variance (the Bulmer effect). Which change is more important? Based on a small simulation study, Mueller and James (1983) concluded that if epistatic variance is small relative to additive variance and the proportion of pairs showing epistasis is also small, the Bulmer effect dominates the Griffing effect, and disequilibrium reduces the response to selection. This is clearly an interesting area for more work.



**Figure 13.4** Equilibrium  $h^2$  values as a function of  $\kappa$  (recall that  $1 - \kappa$  is the fraction of variance following selection) initial heritability  $h^2$ . Curves (from top to bottom) correspond to  $h^2$  values of 0.75 (upper curve), 0.5, 0.25, 0.1, and 0.05 (lower curve).

### Double Truncation Selection: Theory

Table 13.1 and Figure 13.2 show that  $\kappa > 0$  under stabilizing double-truncation selection, so that selection reduces the within-generation phenotypic variance and generates negative disequilibrium. Conversely,  $\kappa < 0$  for disruptive selection, with selection increasing the within-generation variance and generating positive disequilibrium. Hence, when the infinitesimal model assumptions hold, heritability is expected to decrease under stabilizing selection and increase under disruptive selection. Figure 13.4 plots the equilibrium heritabil-

ity values as a function of the initial heritabilities and the fractional change  $\kappa$  of phenotypic variance retained after selection.

Consideration of Equation 13.13 shows that under stabilizing selection, the value  $\theta = \tilde{\sigma}_A^2/\sigma_z^2$  (which measures the fraction of the initial phenotypic variance that is additive genetic variance at equilibrium) satisfies  $0 < \theta < h^2$ . Similarly, under disruptive selection  $\theta > h^2$ , with one twist. If disruptive selection is sufficiently strong ( $\kappa < -[4h^2(1-h^2)]^{-1}$ ) there is no positive real root for  $\theta$ , and the variance increases without limit in the infinitesimal model (Bulmer 1976a). This is a consequence of the infinite number of loci in the infinitesimal limit. What happens under these conditions with a finite number of loci is suggested from simulation studies of Bulmer (1976a), who examined the behavior when disruptive selection generated sufficiently negative  $\kappa$  values to ensure no positive real root of Equation 13.13. Bulmer assumed 12 identical additive diallelic loci (alternative alleles contributing 1 and 0, respectively, to the genotypic value). After a few generations, this population showed essentially complete disequilibrium, with most individuals having values 0, 12, and 24 (with frequencies 1/4, 1/2, 1/4). At equilibrium, the population behaved like a single locus segregating two alleles (contributing 0 and 12), each with frequency 1/2. Thus, the expectation when there is no positive real solution for  $\tilde{\sigma}_A^2$  is that the population approaches a state of essentially complete disequilibrium.

The approach to the equilibrium value  $\tilde{d}$  also behaves differently under disruptive selection. Under directional and stabilizing selection, the majority of reduction in the additive variance occurs in the first few generations. However, the increase in the variance to its equilibrium value under disruptive selection requires many more generations, as Example 13.3 illustrates.

**Example 13.3.** Consider a normally distributed character with  $\sigma_z^2 = 100$  and  $h^2 = 0.5$  in a random mating population. To compare the effects of stabilizing and disruptive selection, suppose in one replicate disruptive selection is practiced via double truncation with  $p = 0.5$  (the upper and lower 25 percent of the population are saved), while stabilizing selection with  $p = 0.5$  (the upper and lower 25 percent are culled) is practiced in the other. To obtain  $\kappa$  from Table 13.1, first note that for stabilizing selection,  $x_{[1/2+p/2]} = x_{[0.75]}$ , while for disruptive selection  $x_{[1-p/2]} = x_{[0.75]}$ . From normal tables,  $\Pr(U \leq 0.675) = 0.75$ , giving  $x_{[0.75]} = 0.675$ . Likewise,  $\varphi(0.675) = 0.318$ . Hence

$$\kappa = \pm(2 \cdot 0.318 \cdot 0.675)/0.5 = \pm 0.858$$

where the plus is used for stabilizing selection, the minus for disruptive selection. Equation 13.2 becomes

$$d(t+1) = \frac{d(t)}{2} \mp 0.429 \frac{[50 + d(t)]^2}{100 + d(t)}$$

where (since the equation is in terms of  $-\kappa$ ) the minus corresponds to stabilizing selection, the plus for disruptive selection. Starting selection on a base population with  $d = 0$ , iteration yields

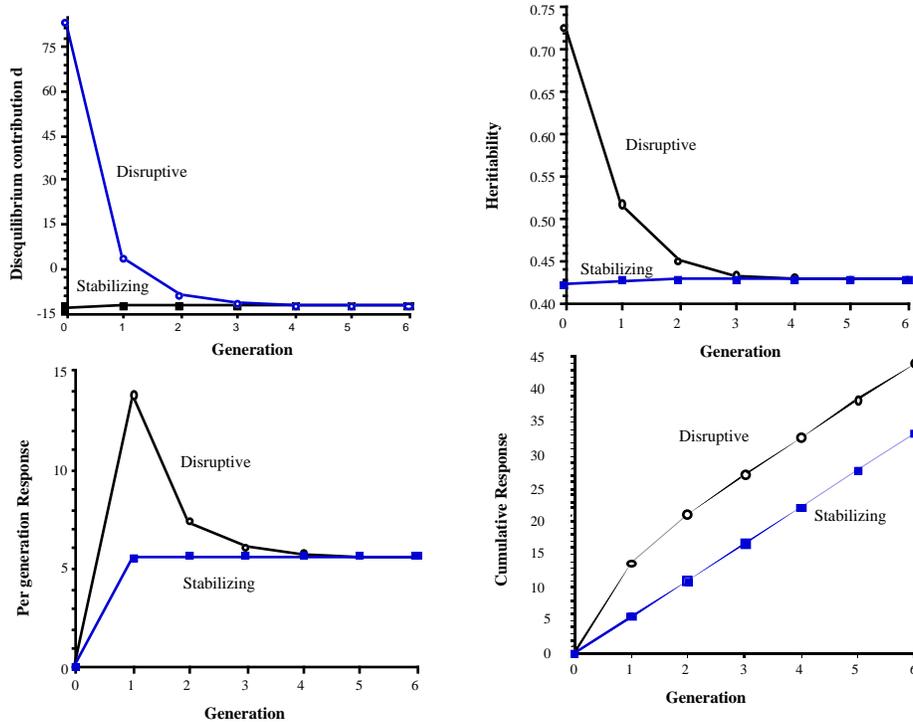
	Disruptive selection							
Generation	0	1	2	3	4	5	20	$\infty$
$d(t)$	0.00	10.73	19.65	27.22	33.72	39.34	74.83	82.69
$\sigma_A^2(t)$	50.00	60.37	69.65	77.22	83.72	89.34	124.83	132.69
$h^2(t)$	0.50	0.55	0.58	0.61	0.63	0.64	0.71	0.73

**Stablizing selection**

Generation	0	1	2	3	4	5	$\infty$
$d(t)$	0.00	-10.73	-12.77	-13.20	-13.29	-13.31	-13.32
$\sigma_A^2(t)$	50.00	39.27	37.23	36.80	36.71	36.69	36.68
$h^2(t)$	0.50	0.44	0.43	0.42	0.42	0.42	0.42

Note that with disruptive selection, the absolute change in  $d$  is much greater and the time to approach equilibrium considerably longer than with stabilizing selection.

As mentioned above, the previous history of selection strongly influences its future response. Suppose that after the equilibrium value of  $d$  has been reached, both of the two above populations are then subjected to directional selection. In particular, assume directional truncation selection with the upper 20 percent of the population being saved. The recursion equation for the disequilibrium contribution is given in Example 13.2, but now the initial disequilibrium values are  $d_0 = -13.32$  for the population with a previous history of stabilizing selection and  $d_0 = 82.69$  for the population with a previous history of disruptive selection. If stabilizing/disruptive selection is stopped for  $k$  generations before truncation selection,  $d_0$  is replaced by  $d_0/2^k$ . From Example 13.2, the per-generation response is  $R(t) = h^2(t) 1.4 \sigma_z(t)$ , which is plotted below for both populations. Initially there is a large difference between the replicates, but after three generations both have essentially the same rate of response, converging to  $\tilde{d} = -12.59$  and hence a per-generation response of  $\tilde{R} = 5.6$ , the equilibrium value under directional truncation selection (see Example 13.2). The replicate originally subjected to disruptive selection shows a higher cumulative response, due to larger responses resulting from higher heritabilities in the first few generations. This difference in cumulative response is permanent — it does not decay away once selection stops.



Hence, we expect that if an unselected base population is divided into three replicates, one first subjected to disruptive selection, another to stabilizing selection, and the third to no

selection, that when directional selection is applied on these replicates, the largest response should occur in the disruptively-selected replicate and the smallest in the replicate that first underwent stabilizing selection. This pattern is indeed seen in artificial selection experiments on *Drosophila* sternopleural bristles (Thoday 1959) and wing length (Tantawy and Tayel 1970).

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### Double Truncation Selection: Experimental Results

Experiments examining the effects of selection on variance are reviewed by Prout (1962), Thoday (1972), Soliman (1982), and Hohenboken (1985). One complication with many of the experimental results is that only phenotypic variances are examined, so that care must be taken to distinguish between changes in genetic, versus environmental, variances.

As expected under the infinitesimal model, several experiments using stabilizing artificial selection (typically by double-truncation) show a reduction in the phenotypic variance that is largely due to reduction in the additive variance, relative to control populations. Examples include sternopleural bristle number (Gibson and Bradley 1974), developmental time (Prout 1962), wing venation (Scharloo 1964, Scharloo et al. 1967), and wing length (Tantawy and Tayel 1970) in *Drosophila melanogaster*, and developmental time in *Tribolium castaneum* (Soliman 1982). Gibson and Bradley (1974) found that some of the decrease in the phenotypic variance was also due to a decrease in the environmental variance as well. Other experiments observed different results. For example, selection on sternopleural bristle number by Gibson and Thoday (1963) resulted in no change in the phenotypic variance because the decrease in additive variance was apparently countered by an increase in the environmental variance (strictly speaking, the increase was in the residual variance, which could include nonadditive genetic variances as well as environmental effects). Likewise, 95 generations of stabilizing selection on pupa weight in *T. castaneum* by Kaufman et al. (1977) resulted in a decrease in the additive variance, but only a slight decrease in the heritability, reflecting a corresponding decrease in the residual variance as well. Bos and Scharloo (1973a,b) observed no decrease in the phenotypic variance following stabilizing selection on *Drosophila* body size. Grant and Mettler (1969) observed a significant increase in variance in one replicate and a significant decrease in the other for two replicate lines subjected to stabilizing selection for a *Drosophila* behavioral trait (escape behavior). Falconer (1957) reported no decrease in additive variance when abdominal bristle number in *Drosophila melanogaster* was subjected to stabilizing selection. However, a re-analysis by Bulmer (1976a) suggested that indeed a reduction in variance occurred, close to the value predicted from the infinitesimal model. Hence, the conclusion is that while reductions in the environmental variance itself sometimes occur, the reduction in the additive variance is often the main source for the observed decrease in phenotypic variance.

Disruptive selection experiments generally show rather large increases in the phenotypic variance. Increases in the heritability and/or additive variance were observed in *Drosophila* for sternopleural bristle number (Thoday 1959, Millicent and Thoday 1961, Barker and Cummins 1969) and wing venation traits (Scharloo 1964, Scharloo et al. 1967), and for pupal weight in *Tribolium* (Halliburton and Gall 1981). Increases in the residual variance were also seen many of these studies, reflecting changes in either the environmental and/or nonadditive genetic variances. On the other hand, for *Drosophila* development time, Prout (1962) observed that the heritability actually decreased relative to the base population, indicating that the large increase observed in phenotypic variance was due to changes in the residual variance. Robertson (1970) observed an increase in the phenotypic variance following disruptive selection on sternopleural bristles, but no corresponding (significant) increase in heritability.

While a change in variance is one prediction from the infinitesimal model, a more striking

prediction is that happens to the variance upon relaxation of selection. Here any gametic-phase disequilibrium generated by selection quickly decays (for unlinked loci). Thus, more solid support would come from experiments that also follow the variance upon relaxation of selection. This was done by Sorensen (1980) and Sorensen and Hill (1982), who selected on abdominal bristle number in *Drosophila melanogaster*. They observed large changes in the phenotypic variance, with realized heritability increasing from 0.37 to 0.69 in two generations of selection. Following four generations of no selection, heritability decreased to 0.44 (the standard error for all heritability estimates was 0.04). This pattern is expected given the decay of gametic-phase disequilibrium (which here is positive and hence inflates the additive variance). Clearly, more experiments examining whether the variance changes immediately after selection is stopped are needed to obtain a general picture for the role of disequilibrium.

### RESPONSE UNDER NORMALIZING SELECTION

While double-truncation is the common mode of artificial stabilizing selection, one standard model assumed for stabilizing selection in natural populations is **normalizing** (or **nor-optimal**) selection (Weldon 1895, Haldane 1954),

$$W(z) = \exp\left(-\frac{(z - \hat{\mu})^2}{2\omega}\right) \tag{13.17}$$

The optimal value is  $z = \hat{\mu}$ , and the strength of selection is given by  $\omega$ . If  $\omega \gg \sigma^2$ , fitness falls off slowly and selection is weak, while if  $\omega \ll \sigma^2$  selection is strong. If phenotypes are normally distributed before selection with mean  $\mu$  and variance  $\sigma^2$ , after selection phenotypes remain normally distributed with new mean and variance

$$\mu^* = \mu + \frac{\sigma^2}{\sigma^2 + \omega}(\hat{\mu} - \mu) \quad \text{and} \quad \sigma_{z^*}^2 = \sigma^2 - \frac{\sigma^4}{\sigma^2 + \omega} \tag{13.18a}$$

This follows as a special case of the general Gaussian fitness function discussed in Chapter 41. Note that we cannot write  $\sigma_{z^*}^2 = (1 - \kappa)\sigma_z^2$ , as here  $\kappa$  is no longer a constant, but rather a function of  $\sigma_z^2$ . However, under this model the distribution of genotypes remains normal after selection and hence parent-offspring regressions remain linear throughout (Chapter 41). Thus, we can apply the breeders' equation to predict changes in the mean and Equation 13.6 to predict changes in the variance (under the infinitesimal model). Here,

$$S = \frac{\sigma^2}{\sigma^2 + \omega}(\hat{\mu} - \mu) \quad \text{and} \quad \delta(\sigma_{z^*}^2) = -\frac{\sigma^4}{\sigma^2 + \omega} \tag{13.18b}$$

The change in mean is thus given by

$$R(t) = h^2(t)S(t) = h^2(t)\frac{\sigma_z^2(t)[\hat{\mu} - \mu(t)]}{\sigma_z^2(t) + \omega} \tag{13.19}$$

implying that the mean converges to  $\hat{\mu}$ . From Equation 13.6, the change in the disequilibrium contribution is given by

$$d(t+1) = \frac{d(t)}{2} - \frac{h^4(t)}{2}\frac{\sigma_z^4(t)}{\sigma_z^2(t) + \omega} = \frac{d(t)}{2} - \frac{1}{2}\frac{[\sigma_a^2 + d(t)]^2}{\sigma_z^2 + d(t) + \omega} \tag{13.20}$$

Note that both directional and stabilizing selection can simultaneously occur with normalizing selection — if  $\mu \neq \hat{\mu}$ , the mean also changes under selection. However, observe from

both Equations 13.18a and 13.20 that the change in variance is independent of the current mean value  $\mu$ .

---

**Example 13.4.** Suppose normalizing selection occurs on a normally distributed character with  $\sigma_z^2 = 100$ ,  $h^2 = 0.5$ , and  $\omega = 200$ . The dynamics of the disequilibrium contribution follow

$$d(t+1) = \frac{d(t)}{2} - \frac{1}{2} \frac{(50 + d(t))^2}{300 + d(t)}$$

implying at equilibrium that  $\tilde{d}$  satisfies

$$\tilde{d}(\sigma^2 + \tilde{d} + \omega) + (\sigma_A^2 + \tilde{d})^2 = \tilde{d}(300 + \tilde{d}) + (\tilde{d} + 50)^2 = 0$$

which gives  $\tilde{d} = -6.46$ , hence  $\tilde{h}^2 = (50 - 6.46)/(100 - 6.46) = 0.47$ . Most of this reduction in heritability occurs in the first few generations. In the first generation of selection, variances are those in the base population, hence we let selection start in generation 0.

Generation	0	1	2	3	4	5	$\infty$
$d_t$	0	-4.17	-5.64	-6.16	-6.35	-6.42	-6.46
$\sigma_A^2(t)$	50.00	45.83	44.37	43.84	43.65	43.58	43.54
$h^2(t)$	0.50	0.48	0.47	0.47	0.47	0.47	0.47

Thus, under the infinitesimal model, the distribution reaches equilibrium with the phenotypes (before selection) normally distributed with mean  $\hat{\mu}$  and variance  $\tilde{\sigma}_z^2 = 100 - 6.46 = 93.54$ .

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## SELECTION WITH ASSORTATIVE MATING

Recall (LW Chapter 7) that assortative mating changes the additive genetic variance relative to a random mating population, mainly by generating gametic-phase disequilibrium. Assortative mating results in some inbreeding (measured by a slight increase in homozygosity), but if the number of loci is large, the deviation of genotypes from Hardy-Weinberg frequencies is expected to be small. In the limiting infinitesimal model, no changes in genotypic frequencies occur at single loci, although large changes in variance can occur due to gametic-phase disequilibrium. Positive assortative mating (where  $\rho$ , the phenotypic correlation between mates, is positive) generates positive gametic-phase disequilibrium, increasing  $\sigma_A^2$ , while negative assortative mating ( $\rho < 0$ , also referred to as **disassortative mating**) generates negative  $d$ , decreasing  $\sigma_A^2$ . As with selection, these changes in the variance are temporary, with values returning to those in the base population after a few generations of random mating.

### Results Using the Infinitesimal Model

Assortative mating is easily incorporated into the infinitesimal model (Fisher 1918, Bulmer 1980). We assume assortative mating follows selection, so that the selected parental phenotypic values  $z_f^*$  and  $z_m^*$  are correlated. Returning to Equation 13.5a, the variance in the offspring is given by

$$\sigma^2(z_o) = \frac{h^4}{4} \sigma^2(z_m^* + z_f^*) + \sigma_e^2 \quad (13.21a)$$

Writing the variance of a sum as  $\sigma^2(x + y) = \sigma_x^2 + \sigma_y^2 + 2\rho_{xy}\sigma_x\sigma_y$ , this becomes

$$\sigma^2(z_o) = \frac{h^4}{4} \left( \sigma^2(z_m^*) + \sigma^2(z_f^*) + 2\rho\sigma(z_f^*)\sigma(z_m^*) \right) + \sigma_e^2 \quad (13.21b)$$

Assuming selection is such that  $\sigma^2(z_x^*) = (1 - \kappa_x)\sigma_z^2$  for  $x = f$  or  $m$ , Equation 13.21b becomes

$$\sigma^2(z_o) = \frac{h^4}{2} \sigma_z^2 \left( 1 - \frac{\kappa_f + \kappa_m}{2} + \rho \sqrt{(1 - \kappa_f)(1 - \kappa_m)} \right) + \sigma_e^2 \quad (13.21c)$$

Comparing this with Equation 13.11a and 13.12, we see that with assortative mating, Equation 13.12 holds with

$$\kappa = \frac{\kappa_f + \kappa_m}{2} - \rho \sqrt{(1 - \kappa_f)(1 - \kappa_m)} \quad (13.21d)$$

Likewise, Equations 13.13a-13c hold with  $\kappa$  replaced by Equation 13.21d. This generalization is due to Tallis (1987, Tallis and Leppard 1988a), and has been extended to multiple characters by Tallis and Leppard (1988b). If there is no selection ( $\kappa_f = \kappa_m = 0$ ),  $\kappa = -\rho$  and previous results for assortative mating (LW Equations 7.18-7.20) follow immediately from Equations 13.12, 13.7c, and 13.13 (respectively). More generally, when the amount of selection and assortative mating change each generation,

$$d(t+1) = \frac{d(t)}{2} - \frac{\kappa(t)}{2} h^2(t) \sigma_A^2(t) \quad (13.22)$$

where  $\kappa(t)$  is given by Equation 13.21d with  $\kappa_f$ ,  $\kappa_m$  and  $\rho$  taking on values for generation  $t$ .

Thus under the infinitesimal model, analyzing the joint effects of assortative mating and selection is straightforward. When selection is the same in both sexes, the effect of assortative mating is to change  $\kappa$  to  $\kappa - \rho(1 - \kappa)$ . Negative gametic-phase disequilibrium is generated when this quantity is positive (indicating a reduction in variance), while positive disequilibrium is generated when it is negative. Note that if  $\kappa > 0.5$ ,  $\kappa - \rho(1 - \kappa) > 0$  and no amount of positive assortative mating can generate positive disequilibrium. However, for all values of  $\kappa$ , there is some amount of assortative mating such that  $\kappa - \rho(1 - \kappa) > 0$ . Even if selection generates positive disequilibrium ( $\kappa < 0$  such as with disruptive selection), sufficiently strong negative assortative mating ( $\rho < \kappa/[1 - \kappa]$ ) generates negative disequilibrium, reducing the additive genetic variance.

### Assortative Mating and Enhanced Response

Since positive assortative mating increases the additive genetic variance, Breese (1956) and James and McBride (1958) suggested that response to selection could be increased by employing assortative mating. Since the response to selection depends on the additive variance of the parents, at least one generation of assortative mating is required in order for the response to differ from random mating. Experimental support from an increase in response with assortative mating is mixed. McBride and Robertson (1963) observed a slight (but not statistically significant) increase in response to selection on abdominal bristle number in *Drosophila melanogaster* when parents were assortatively mated. Wilson et al. (1965) also reported a slight (but not significant) increase in response with assortative mating for *Tribolium* pupal weight, while Sutherland et al. (1968) found no effect of assortative mating when selecting on 6-week body weight in mice.

The efficiency of assortative mating under truncation selection has been examined in detail by Baker (1973), DeLange (1974), Fernando and Gianola (1986), Smith and Hammond (1987), Tallis and Leppard (1988a), and Shepherd and Kinghorn (1994). The general conclusion is that the relative increase in response is greatest when  $h^2$  is large and selection is

weak. However, unless the population is subjected to multiple generations of assortative mating before selection, if selection is strong, the increase (for individual selection under the infinitesimal model) is at most six percent, consistent with the very small increases seen in experiments. However, if there are a small number of loci, Fernando and Gianola found a larger effect, which largely appeared to arise from faster allele frequency change under assortative mating (as opposed to generation of positive disequilibrium). Likewise, Shepherd and Kinghorn (1994) found a much larger effect when selection and assortative mating is done using estimating breeding values (BLUP Selection, Chapter 34) as opposed to simple individual selection. As Example 13.5 demonstrates, under the infinitesimal model the per-generation increase in response of a population subjected to multiple rounds of assortative mating prior to selection rapidly decays to zero once selection starts.

**Example 13.5.** Starting with a base population initially in gametic-phase equilibrium with  $h^2 = 0.5$ ,  $\sigma_z^2 = 100$ , individuals are positively assortatively mated (with  $\rho = 0.5$ ) for  $k$  generations before being subjected to directional truncation selection with  $p = 0.20$ . What is the difference in response to one generation of selection in these assortatively mated populations relative to a random mating base population? From Example 13.2, the response in generation  $t$  is given by  $R(t) = 1.4 h^2(t) \sigma_z(t)$ . The expected disequilibrium contribution following  $k$  generations of assortative mating (here,  $\kappa = -\rho = -0.5$ ) is given by iterating Equation 13.22

$$d(t + 1) = \frac{d(t)}{2} + 0.25 h^2(t) \sigma_A^2(t)$$

where  $d(0) = 0$ ,  $h^2(0) = 0.5$  and  $\sigma_A^2(0) = 50$ . Iteration gives:

**Single generation response following  $k$  generations of assortative mating**

k	0	1	2	3	5	10	$\infty$
$d(t)$	0.00	6.25	10.57	13.58	17.17	20.09	20.71
$h^2(t)$	0.50	0.53	0.55	0.56	0.57	0.58	0.59
$R(t)$	7.00	7.64	8.06	8.35	8.69	8.95	9.01
$R(t)/R_{rm}$	1.00	1.09	1.15	1.19	1.24	1.28	1.29

where  $R_{rm} = 7.00$  is the response under random mating. While there can be up to a 29% increase in the rate of response to a single generation of selection, it is extremely inefficient to assortatively mate a population for several generations before applying selection. Once selection starts,  $d$  decays very rapidly, greatly reducing  $R/R_{rm}$ . Assuming  $d(0) = 20.71$ , after a single generation of selection,  $d(1) = -3.84$ ,  $h^2 = 0.48$ , and the response in the next generation drops to 6.59 (compared to a response of 5.98 under random mating).

**Example 13.6.** Consider the same population and selection parameters as in the previous example, but now selection and assortative mating occur simultaneously starting at generation 0. What is the difference in response if random mating occurs in one replicate and assortative mating with  $\rho = 0.5$  in the other? From Example 13.2,  $\kappa_f = \kappa_m = 0.787$ , giving

$$\kappa - \rho(1 - \kappa) = 0.787 - 0.5(1 - 0.787) = 0.6805$$

and (from Equation 13.22), the change in  $d$  in the assortatively mated population becomes

$$d(t + 1) = \frac{d(t)}{2} - \frac{0.6805}{2} h^2(t) \sigma_A^2(t)$$

Assuming the initial population is in gametic phase disequilibrium ( $d(0) = 0$ ), iteration yields

Generation	0	1	2	3	4	5	$\infty$
$d(t)$	0.00	-8.51	-10.66	-11.22	-11.37	-11.42	-11.43
$h^2(t)$	0.50	0.45	0.44	0.44	0.44	0.44	0.44
$R(t)$	7.00	6.07	5.82	5.76	5.74	5.74	5.74
$R_{rm}(t)$	7.00	5.92	5.66	5.62	5.61	5.60	5.60
$R(t)/R_{rm}(t)$	1.00	1.03	1.03	1.02	1.02	1.02	1.02

where  $R_{rm}(t)$ , the response in generation  $t$  of random mating, comes from Example 13.2. Thus, there is at most a 3% increase in the rate of response. Note that the response in the first generation (generation 0) is the same in both populations — the response to selection depends on the variance of the parents, and in the first generation both populations have the same variance (as  $d(0) = 0$  in each).

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### Disruptive Selection, Assortative Mating, and Reproductive Isolation

We would be remiss if we did not mention a historical interest between disruptive selection and assortative mating as a mechanism for reproductive isolation. In the early 1960's the general view was that speciation (reproductive isolation between populations) required geographic (or other) isolation, a view strong championed by Mayr (1963). However, the idea that **sympatric speciation** (Maynard-Smith 1962, 1966) could develop without the need for such isolation was bolstered by an experimental observation by Thoday and Gibson (1962). They observed that disruptive selection on sternopleural bristle in *D. melanogaster* seemed generate two distinct groups (high vs. low files) that appeared to assortatively mate (individuals with intermediate phenotype were absent from the population, where they would be expected if random mating occurred). The idea is that crosses between high and low parents generate less fit offspring (having intermediate values) and that natural selection occurred to generate preferential mating over the short time course of this experiment. Thus, it appeared, only 12 generations of disruptive selection had generated partial reproductive isolation within a population. However, this striking observations was not repeatable (Scharloo et al. 1967, Charbora 1968, Baker and Cumming 1968, Thoday and Gibson 1970). Indeed, Scharloo (1971) suggested that the base population for selection used by Thoday and Gibson might have been composed of flies from different geographic origins, and hence already possessing partial isolation that was uncovered, rather than evolved, by Thoday and Gibson's experiments. While Today and Gibson's interpretation of their experiments is now largely discounted, the notion of **reinforcement** (the evolution of mating preferences to reduce the production of less fit hybrids when diverged populations come back into contact), remains an important concept (Noor 1999, Servedio and Noor 2003).

### SELECTION IN THE PRESENCE OR HERITABLE VARIATION IN $\sigma_E^2$

Our assumption to this point has been that the environmental variation is homoscedastic (constant across genotypes), and hence not subject to modification by selection, so that changes in the phenotypic variance  $\sigma_z^2 = \sigma_G^2 + \sigma_E^2$  are entirely due to changes in  $\sigma_G^2$ . However, a fairly universal (and very striking) observation is that most traits examined in an outbred population show at least some genetic variation. One can imagine that sensitivity to the environment is one such trait (Waddington 1957, Hill 2007), and one measure of this is the environmental variance. This raises the possibility that there is heritable variation in  $\sigma_E^2$ , offering another pathway to change the phenotypic variance.

One immediate question is how this added complication might comprise standard equa-

tions of selection response. Further, there are also situations where direct selection on  $\sigma_E^2$  is desired. Due to the design of many agricultural production systems, there is often an economic advantage (which may be considerable) to more uniformity in the trait, even while one is trying to improve its mean. Examples include more uniform harvesting time or meat quality. In both cases, we are trying to improve the mean while shrinking the variance. More generally, we may wish to improve an index that weights both mean *and* variance, such as  $I = a_1\mu_z + a_2\sigma_z^2$ . Schemes that directly select the environmental variance (such as using an index of family mean and variance under a sire design) are examined in Chapter 34.

We start our discussion of selection on  $\sigma_E^2$  by first considering the different scales over which environmental sensitivity can act. Next, we review evidence for genetic heterogeneity in  $\sigma_E^2$  before turning to several models for the transmission of any heritable differences in  $\sigma_E^2$  from parent to offspring. With this machinery in hand, we consider univariate treatments of selection on the environmental variance. Chapter 34 expands our analysis by placing the joint selection of *both* trait mean and variance in the proper multiple-trait/selection index framework. Chapter 47 examines developmental issues related to  $\sigma_E^2$ .

The major results developed below (and in Chapter 34) are as follows. First, direct selection on the mean can result in (generally) weak selection on  $\sigma_E^2$ , but when the breeding values for trait mean and  $\sigma_E^2$  are correlated, selection on the mean can have a significant effect on  $\sigma_E^2$ . Second, stabilizing or distributive selection generally only results in a small change in the variance through reduction or expansion of  $\sigma_E^2$ , with most of the short-term response (under the infinitesimal model) coming from changes in the additive variance of the trait itself. Third, as shown in Chapter 34, significant response can occur in the environmental variance by directly targeting it, for example by using a selection index on family variance. Hence, while phenotypic selection (directional, stabilizing, or disruptive selection) on trait value often has little effect on  $\sigma_E^2$ , dramatic response can occur by using schemes that directly utilize additive-genetic variance in  $\sigma_E^2$ .

### Micro-environmental Variance, Developmental Noise and Canalization

Genotypes can potentially vary in their sensitivity to the environment at many different scales of resolution. At the level of macro-environments (such as different growing regions for a crop or major differences in temperature within a species range), there can be strong genotype-environment interactions (differences in the *mean* performance of the same genotype across environments). For example, one genotype may be relatively insensitive to macro-environmental differences, having roughly the same mean value over a number of diverse environments. In contrast, another genotype might show significant differences in mean value over these environments (e.g., Chapters 20, 38, 39, and 41). As the opposite end of the scale, there is development variance, for example as measured by differences in homologous structures on the left and right sides of bilaterally-symmetric organisms (Chapter 47, LW Chapter 11). Presumably this within-individual variation almost entirely reflects “noise” in the developmental process — variation in the end product of the same genotype in the same environment. At intermediate scales are small differences in the environment that different individuals within the same macro-environment might experience. Variation at all of these scales influences the environmental variance of a trait, and presumably one could select at any of these levels to modify the environmental variance. This range of scales is reflected in the standard decomposition of the environmental variation into general influences that effect all individuals within that environment (be it a common growing region or a common material environment) and specific environmental effects that are uniquely experienced by an individual (such as micro-environmental variation or developmental noise). In artificial or natural selection, we can often (but not always!) account for macro-environment differences. When we can do so, selection response under significant  $G \times E$  can be treated as a multiple-trait selection problem (Chapters 38 and 39). Within a given macro-environment,

the remaining environmental variation arises from variation in micro-environments and from developmental noise. It is these two factors that we will consider here, and if different genotypes have different sensitivities to micro-environmental factors and/or developmental noise, they will also display genetic variation in  $\sigma_E^2$ .

Waddington (1942) introduced the notion of **canalization**, wherein the organism is developmentally buffered against small perturbations, be they environmental or genetic, so that a wide range of genotypes and environments end up at essentially the same developmental end-product. He also stressed that canalization is an *evolved system* (Waddington 1957, 1959), and hence to some extent is a selectable trait. We examine canalization and related features of development in detail in Chapter 47. An important, but subtle, distinction is between **environmental canalization** (canalization against environmental noise) and **genetic canalization** (canalization against genetic variation). Selection on environmental canalization is akin to selection on the environmental variance, and hence our results here (and in Chapter 34) will have some bearing on this issue. One straight-forward way to measure environmental canalization is through the measurement of the amount of fluctuating asymmetry (differences in homologous trait values on the left and right side of bilaterally-symmetric organism; Chapter 47, LW Chapter 11). Selection on genetic canalization is a deeper issue, and examined in Chapter 47.

### Evidence for Heritable Variation in Environmental Variance

The idea that different genotypes may have different environmental sensitivities (and hence different environmental variances) is not a new one. Robertson and Reeve (1952) and Lerner (1954) noted that inbred lines often had larger environmental variances relative to their outbred counterparts (see Whitlock and Fowler (1999) for a recent example). This led Lerner to propose that **genetic homeostasis** (developmental buffering across environments) was facilitated by heterozygosity, with environment sensitivity ( $\sigma_E^2$ ) increasing with homozygosity. More recently it has been observed that developmental noise (typically measured by the amount of fluctuating asymmetry) often decreases with increasing levels of protein heterozygosity (reviewed in Mitton and Grant 1984; Livshits and Kobylansky 1985; Zouros and Foltz 1987; Chakraobty 1987), a point we will return to shortly.

Indirect evidence for variation in environmental sensitivity comes from numerous examples in dairy cattle of heterogeneity in variance across herds (Hill et al 1983, Lofgren et al 1985, Brotherstone and Hill 1986, Boldman and Freeman 1990, Dong and Mao 1990, Short et al. 1990, Visscher and Hill 1992). While between-herd differences in environmental sensitivity could account for this heterogeneity across herds, so could differences in genetic variances. In reality, both likely contribute. A more direct observation for heritable variation in environmental sensitivity is that traits usually respond to selection to either increase or decrease the amount of fluctuating asymmetry in a trait (Chapter 47; LW Chapter 11).

There are several additional lines of evidence consistent with genetic variation in the environmental variance. First, although we have noted differences in  $\sigma_E^2$  between inbred and outbred lines, there are also significant differences *among* inbred lines. This is commonly seen in crops (Chapter 20), and Mackay and Lyman (2005) also observed different amounts of environmental variation for bristle number across inbred lines of *Drosophila* from a common source population.

Second, one of the early QTL mapping projects in maize (Edwards et al. 1987) noted that different marker genotypes not only differed in their means, but also in their variances. This could arise from differences in environmental variances across QTL genotypes, but it could also arise because of linkage of a marker to multiple QTL and hence from the segregation variance differing across marker genotypes.

The third line of evidence comes from statistical models (developed in the next section) that allow for an explicit test (given an appropriate experimental design) of genetic variation

in the residual error. Such evidence has been found for fecundity in sheep (SanCristobal-Gaudy et al. 2001), body weight in the snail *Helix aspersa* (Ros et al. 2004), and litter size in pigs (Sorensen and Waagepetersen 2003). The caveat with these results is the (as yet unproved) concern that violations of the underlying statistical models may lead to an incorrect suggestion that such genetic variation exists when in fact it is absent.

The final line of evidence comes from heritable variation of the within-family variance. Again, this is an old observation, as Van Vleck (1968) and Clay et al. (1979) observed significant sire differences in the variation in milk yield in dairy cattle across half-sib families. In a more recent study, Rowe et al. (2006) also found significant sire variation in the within-family residual variance for 35-day body weight in broiler chickens. While suggestive, variation between sires in their within-family variance can also arise from genetic segregation. In particular, heteroscedasticity of family variances is a classic (but weak) test for the presence of a major gene, with heterozygous parents having sibs a larger within-family variance than homozygous parents. (LW Chapter 13).

Taken as a whole, these observations collectively suggest that heritable variation in the environmental variation likely exists for many (perhaps most!) traits, and that selection on the phenotypic variance can result in a response in part due to changes in the overall environmental variance of the population. Indeed, recall that this was seen in several of the stabilizing/disruptive selection experiments we previously reviewed.

### Modeling Genetic Variation in $\sigma_E^2$

The basic starting model is that the phenotypic value of an individual of genotype  $i$  can be written as

$$z_i = \mu + G_i + E, \quad \text{where } E \sim (0, \sigma_i^2) \quad (13.23a)$$

(The notation  $x \sim N(\mu, \sigma^2)$  denotes that  $x$  comes from a distribution with mean  $\mu$  and variance  $\sigma^2$ .) For ease of development, we generally assume that the trait is entirely additive so that  $G = A_m$ , the breeding value for the mean. Taking the expectation (we use roman E for expectation and italic  $E$  for environmental values), the population environmental variance is the average of the  $\sigma_i^2$ ,

$$\sigma_E^2 = E[\sigma_i^2] \quad (13.23b)$$

If we are working with a series of pure lines (e.g., Chapter 20), we can estimate  $\sigma_i^2$  for each line, and the transmission (within a line) from parent to offspring is trivial since they are clones. The more interesting problem arises when dealing with an outbred population. In this case we have to deal with both estimation and the vexing issue of modeling transmission. Models allowing for heterogeneity of environmental variance were introduced in the 1990's (Foulley et al. 1992, Foulley and Quaas 1995, Cullis et al 1996), but these ignored the question of selection (and evolution) of the environmental variance itself. The first formal models strictly looking at the evolution of the environmental variance were population-genetic models by Gavrillets and Hastings (1994) and Wagner et al. (1997), and breeding-value based models by SanCristobal-Gaudy et al. (1998).

Gavrillets and Hastings (1994) assumed some underlying environmental value  $e$  (such as temperature), with different genotypes having different sensitivity  $\gamma_i$ , so that

$$E = \gamma_i e, \quad \text{where } e \sim (0, \sigma_e^2) \quad (13.24a)$$

This **multiplicative model** is simply the Finlay-Wilkinson model for genotype-environmental interactions (Chapter 39), and was also used by Wagner et al. (1997). Under Equation 13.24a, the conditional environmental variance (given the genotypic value and its environmental sensitivity) is

$$\sigma^2[E | G, \gamma_i] = \gamma_i^2 \sigma_e^2 \quad (13.24b)$$

Taking the expected value over  $\gamma$  gives the unconditional environmental variance (Example 13.7),

$$\sigma_E^2 = (\mu_\gamma^2 + \sigma_\gamma^2) \sigma_e^2 \quad (13.24c)$$

Under the multiplicative model, the environmental variance for the population decreases by selecting  $\mu_\gamma$  to zero and/or by decreasing the variance  $\sigma_\gamma^2$ . The problematic issue here is modeling the change in the distribution of the genotypic-specific sensitivities  $\gamma$ . The simplest approach is to assume  $\gamma$  is an entirely additive quantitative trait, so that  $\gamma = A_v$ , namely the breeding value for the environmental variance. In this framework, the mean breeding value of the population of offspring is the mean breeding value of their parents (Chapter 10). Similarly, the variance in breeding values in the offspring is given by the machinery developed in this chapter, namely the base additive variance plus half (for unlinked loci) the disequilibrium value in the selected parents.

Equation 13.24c lead Gavrillets and Hasting to comment on the relationship between developmental noise and heterozygosity. Lerner assumed this was causative – higher levels of heterozygosity resulted in decreased environmental variance. However, Gavrillets and Hastings noted that when  $\mu_\gamma^2 = 0$ , as might occur with selection to decrease  $\sigma_E^2$ , then the environmental variance is proportional to the additive genetic variance  $\sigma_\gamma^2 = \sigma_{A_v}^2$ . Recalling a result from Chakraborty (1987), namely that for an additive trait, the genetic variance is a decreasing function of the number of heterozygous loci, they note that the correlation between heterozygosity and  $\sigma_E^2$  simply falls out as a consequence of their model, rather than from any functional relationship between the two. Conversely, if Lerner was correct and the relationship between  $\sigma_E^2$  and heterozygosity is indeed casual, Zhivotovsky and Feldman (1992) note that if  $\sigma_E^2$  is a decreasing function of heterozygosity, the equilibrium mean value of a trait under stabilizing selection may not coincide with the optimal fitness value.

If we allow for dominance in the quantitative-trait formulation of  $\gamma$ , we now have  $\gamma = A_v + D_v$ , where the dominance value  $D_v$  is *not* transmitted from parent to offspring. Further, by construction  $D_v$  has a mean value of zero and under the infinitesimal model, the dominance variance is not changed by selection (a point developed earlier in this chapter). Under this extension, the mean environmental variance becomes

$$\sigma_E^2 = (\mu_{A_v}^2 + \sigma_{A_v}^2) \sigma_e^2 + \sigma_{D_v}^2 \sigma_e^2 \quad (13.24d)$$

While selection can reduce the first component (first to  $\sigma_{A_v}^2 \sigma_e^2$  by driving the mean breeding value to zero, and then reducing  $\sigma_{A_v}^2$  by generating negative disequilibrium), the second component involving non-additive variance remains unchanged. Hence, implicit in assuming a breeding value for this (or any of the other models discussed below) is that any non-transmissible genetic variation in  $\sigma_E^2$  remains unchanged by selection. Genetic variation in  $\sigma_E^2$ , by itself, is not sufficient for a selection response, as at least part of this variation must be transmissible under the breeding scheme used.

While we have presented the multiplicative model within a breeding-value framework, this was not explicitly done by Gavrillets and Hastings (1994), who (coming from a population-genetics background) were more concerned with evolution of the environmental variance than estimating  $A_v$ . Conversely, SanCristobal-Gaudy et al. (1998), coming from an animal breeding background, were also concerned with estimation. They did so by modeling  $E$  using an **exponential model**,

$$E = \exp\left(\frac{A_v}{2}\right) \cdot e, \quad \text{where } e \sim N(0, \sigma_e^2) \quad \text{and} \quad A_v \sim N(\mu_{A_v}, \sigma_{A_v}^2) \quad (13.25a)$$

The assumption that  $e$ ,  $A_v$ , as well as  $A_m \sim N(\mu_{A_m}, \sigma_{A_m}^2)$ , are all uncorrelated and normally distributed allowed SanCristobal-Gaudy et al. (1998) to obtain likelihood estimators the

breeding values for the environmental variance ( $A_v$ ) and trait mean ( $A_m$ ). They explicitly considered estimation under either a sire design (using half sib values to estimate  $A_v$  and  $A_m$  of the parent) or using a repeatability model (Chapter 15) where repeated measurements on a single individual are used to estimate its breeding values. SanCristobal-Gaudy et al. (2001) extend this approach to threshold traits (in particular, litter size). Bayesian estimators under this model are developed by Sorensen and Waagepetersen (2003) and Ros et al. (2004).

Given  $A_v$ , the conditional distribution of the environmental variance becomes

$$\sigma^2(E | A_v) = \sigma_e^2 \exp(A_v) \quad (13.25b)$$

which follows by recalling  $(e^a)^2 = e^{2a}$ . Hence, the environmental variance is a constant ( $\sigma_e^2$ ) multiplied by a scaling factor that is a function of the breeding value  $A_v$  for the environmental variance. Decreasing  $A_v$  results in an individual with reduced environmental sensitivity (reduced  $\sigma_E^2$ ). The constant  $\sigma_e^2$  can be interpreted as the environmental variance for an individual with an environmental breeding value of zero. The exponential model is also called the **log-additive model**, as the breeding value is additive on the log of the variance scale,

$$\ln[\sigma^2(E | A_v)] = \ln(\sigma_e^2) + A_v, \quad (13.25c)$$

As detailed in Example 13.7, the expectation of Equation 13.25b gives the mean environmental variance as

$$\sigma_E^2 = \sigma_e^2 \exp(\mu_{A_v} + \sigma_{A_v}^2/2) \quad (13.25d)$$

Equation 13.25d shows that either decreasing the mean breeding value  $\mu_{A_v}$ , or its additive variance  $\sigma_{A_v}^2/2$ , decreases the environmental variance. Comparison of Equations 13.24c and 13.25d shows one subtle difference between the multiplicative and exponential models. Under the former, the minimal population environmental variance occurs when  $\mu_{A_v} = 0$ , with any deviation from this increasing the average environmental variance in the population. By contrast, under the exponential model, decreasing  $\mu_{A_v}$  always decreases the average value of  $\sigma_E^2$  in the population. Thus, under the exponential model,  $\sigma_e^2$  can be selected to be arbitrary small, while under the multiplicative model, it has a lower bound set by  $\sigma_{A_v}^2$  (and more generally by the dominance variance as well, see Equation 13.24d).

**Table 13.3.** Models for heritable variation in the environmental value  $E$ . The basic model is  $z = \mu + A_m + E$ , where  $z$  is the trait value and  $A_m \sim N(\mu_{A_m}, \sigma_{A_m}^2)$  the breeding value for trait value. The table gives the assumed form of  $E$  for different models as a function of the breeding value in variance  $A_v \sim N(\mu_{A_v}, \sigma_{A_v}^2)$ , some intrinsic environmental value  $e \sim N(0, \sigma_e^2)$ , and the unit normal  $U \sim N(0, 1)$ .

Model	$E$	$\sigma^2(E   A_v)$	$\sigma^2(E)$
Multiplicative	$A_v e$	$A_v^2 \sigma_e^2$	$(\mu_{A_v}^2 + \sigma_{A_v}^2) \sigma_e^2$
Exponential (or log-additive)	$\exp(A_v/2) e$	$\sigma_e^2 \exp(A_v)$	$\sigma_e^2 \exp[\mu_{A_v} + \sigma_{A_v}^2/2]$
Additive	$\sqrt{A_v + \sigma_e^2} \cdot U$	$A_v + \sigma_e^2$	$\mu_{A_v} + \sigma_e^2$

Our last formulation for modeling genetic variation in  $E$  was suggested by Hill and Zhang (2004) and Mulder et al (2007),

$$E = U \cdot \sqrt{\sigma_e^2 + A_v}, \quad \text{where } U \sim N(0, 1) \quad \text{and} \quad A_v \sim N(\mu_{A_v}, \sigma_{A_v}^2) \quad (13.26a)$$

This is the **additive model**, as the environmental variance for an individual with breeding value  $A_v$  is simply

$$\sigma^2(E | A_v) = \sigma_e^2 + A_v \quad (13.26b)$$

with the constraint on the breeding value that  $\sigma^2 + A_v > 0$ . The additive model is a *local*, as selection to decrease  $A_v$  can eventually result in the model constraints being violated, generating a negative variance. Under the additive model, the mean population value for the environmental variance is simply

$$\sigma_E^2 = E[\sigma_e^2 + A_v] = \sigma_e^2 + \mu_{A_v} \quad (13.26c)$$

Unlike the multiplicative and exponential models, changes in  $\sigma_E^2$  under the additive model depend only on changes in the mean breeding value, and not its variance. Mulder et al. (2007) develop a simple regression estimator for  $A_v$  that is examined in detail in Chapter 34.

The additive model has the advantage of being much more tractable, but the disadvantage that it breaks down when the breeding value becomes sufficiently negative ( $\sigma_e^2 + A_v < 0$ ). In contrast, the exponential model has additivity on the log of the variance scale, which is a nice statistical feature, as log variances are approximately normally distributed (Box 1953, Layard 1973). Mulder et al. (2007) discuss additional connections between the additive and exponential models.

**Example 13.7.** Here we derive the unconditional variances for the models summarized in Table 13.3. Consider the multiplicative model first, where

$$\sigma_E^2 = E[\gamma^2 \sigma_e^2] = \sigma_e^2 E[\gamma^2]$$

Recalling that  $\sigma_x^2 = E[x^2] - \mu_x^2$ , rearranging gives

$$\sigma_E^2 = \sigma_e^2 E[\gamma^2] = \sigma_e^2 (\sigma_\gamma^2 + \mu_\gamma^2)$$

Turning to the exponential model (Equation 13.25a), since by construction  $E$  has expected value zero, the variance of  $E$  is simply the expected value of  $E^2$ ,

$$\sigma_E^2 = E \left[ (\sigma_e \exp\{A_v/2\})^2 \right] = \sigma_e^2 E \left[ (\exp\{A_v/2\})^2 \right] = \sigma_e^2 E[\exp(A_v)]$$

where the expected value with respect to the distribution of breeding values  $A_v$ . The last expectation is computed by noting for a normal with mean  $\mu$  and variance  $\sigma^2$ , that  $E[e^x] = \exp(\mu + \sigma^2/2)$ , which follows using the standard expression for the moment generating function  $E[e^{tx}]$  upon setting  $t = 1$  (Johnson and Kotz 1970). Since we assumed  $A_v \sim N(\mu_{A_v}, \sigma_{A_v}^2)$ , the environmental variance for the population becomes

$$\sigma_E^2 = \sigma_e^2 \exp \left( \mu_{A_v} + \frac{\sigma_{A_v}^2}{2} \right)$$

### $h_v^2$ , the Heritability of the Environmental Variance

Under the additive model framework (Equation 13.26), Mulder et al. (2007) suggest a definition for the heritability of the environmental variance. Their motivation is that the heritability

of a trait is the slope in the regression of the breeding value of an individual on its phenotype. As shown in Chapter 34,  $A_v$  is a linear function of the square  $z^2$  of phenotypic value. From regression theory (LW Chapter 3), the slope of the regression of  $A_v$  on  $z^2$  is simply their covariance divided by the variance of the predictor variable,

$$h_v^2 = \frac{\sigma(A_v, z^2)}{\sigma^2(z^2)} \quad (13.27a)$$

By definition,  $\sigma(A_v, z^2) = \sigma(A_v, A_v) = \sigma^2(A_v)$ , while (assuming  $z$  is normally distributed)  $\sigma^2(z^2) = 2\sigma_z^4 + 3\sigma_{A_v}^2$ . Thus the heritability becomes

$$h_v^2 = \frac{\sigma^2(A_v)}{2\sigma_z^4 + 3\sigma_{A_v}^2} \quad (13.27b)$$

Table 13.4 reviews the (limited) estimates of  $h_v^2$  from the literature. Note that the estimated heritabilities are low, typically less than five percent. However, also note that the evolvability (the coefficient of genetic variation, measured by the standard deviation of additive variance divided by the mean trait value  $\sigma_{A_v}/\sigma_E^2$ , Houle 1992), is large. Although selection may be difficult (given the low heritability), there is much variation to exploit (high evolvability).

**Table 13.4.** Literature estimates of the heritability  $h_v^2$  and evolvability  $\sigma_{A_v}/\sigma_E^2$  of the environmental variance (after Mulder et al. 2007).

Species	Trait	$h_v^2$	$\sigma_{A_v}/\sigma_E^2$	Reference
Pig ( <i>Sus</i> )	Meat pH	0.039	0.402	SanCristobal-Gaudy et al. (1998)
	Litter size	0.026	0.307	Sorensen & Waagepetersen (2003)
Sheep ( <i>Ovis</i> )	Litter size	0.048	0.509	SanCristobal-Gaudy et al. (2001)
Snail ( <i>Helix</i> )	Body weight	0.017	0.580	Ros et al. (2004)
Chicken ( <i>Gallus</i> )	Body weight (male)	0.029	0.399	Rowe et al. (2006)
	Body weight (female)	0.031	0.318	Rowe et al. (2006)

### Translating The Response in $A_v$ into Response in $\sigma_E^2$

A number of authors have modeled the response in the phenotypic variance when there are heritable differences in environmental sensitivity (Gavrilets and Hastings 1994; Wagner et al. 1997; SanCristobal-Gaudy et al. 1998, 2001; Sorensen and Waagepetersen 2003; Ros et al. 2004; Hill and Zhang 2004; Mulder et al. 2007, 2008). A critical step in each of these models is treating phenotypic value and residual variance as two separate (and potentially correlated) traits, both with heritable variation. While some models (Gavrilets and Hastings 1994, Wagner et al. 1997, Hill and Zhang 2004) are based on strict population-genetic models (following the changes in individual alleles), most are based on schemes that assign breeding values to the heritable component of  $\sigma_E^2$  (Table 13.3). Breeding values have nice features, especially under the infinitesimal model. The mean breeding value in the offspring is simply the mean breeding values of the parents (Chapter 10), while changes in the variance in breeding values from parent to offspring have a simple form under the infinitesimal model (Equation 13.8b). Using the expressions given Table 13.3 allows us to map changes in  $\mu_{A_v}$ , the mean breeding value for environmental sensitivity, into changes in  $\sigma_E^2$ . The simplest case is under the additive model (Equation 13.26b). Let the response  $R_{A_v}$  denote the change in

the mean breeding value of the selected parents from the mean breeding value of the entire population. The resulting change in  $\sigma_E^2$  becomes

$$\begin{aligned}\Delta\sigma_E^2(t) &= \sigma_E^2(t+1) - \sigma_E^2(t) = [\mu_{A_v}(t+1) + \sigma_e^2] - [\mu_{A_v}(t) + \sigma_e^2] \\ &= \mu_{A_v}(t) + R_{A_v}(t) - \mu_{A_v}(t) = R_{A_v}(t)\end{aligned}\quad (13.28a)$$

showing that the change in the environmental variance is simply the change in mean breeding value.

Things are a bit more complex under the multiplicative and exponential models, as the mean population value  $\sigma_E^2$  for the environmental variance is a non-linear function of the mean (and variance) of the breeding values. Assume no change in the additive variance of environmental sensitivities. Under the multiplicative model (Equation 13.24a), the change in  $\sigma_E^2$  given a change in breeding values becomes

$$\begin{aligned}\Delta\sigma_E^2(t) &= \left([\mu_{A_v}(t) + R_{A_v}(t)]^2 + \sigma_{A_v}^2\right)\sigma_e^2 - (\mu_{A_v}^2(t) + \sigma_{A_v}^2)\sigma_e^2 \\ &= [2\mu_{A_v}(t)R_{A_v}(t) + R_{A_v}^2(t)]\sigma_e^2\end{aligned}\quad (13.28b)$$

Change in the variance in breeding value in the parents can similarly be accounted for by using Equation 13.8b to obtain the variance of  $A_v$  in the offspring and substituting this into Equation 13.28b. Finally, under the exponential model (again assuming no change in  $\sigma^2(A_v)$ ,

$$\begin{aligned}\Delta\sigma_E^2(t) &= \sigma_e^2 \exp[\mu_{A_v}(t) + R_{A_v}(t) + \sigma_{A_v}^2/2] - \sigma_e^2 \exp[\mu_{A_v} + \sigma_{A_v}^2/2] \\ &= \sigma_e^2 \exp[\mu_{A_v} + \sigma_{A_v}^2/2] (\exp[R_{A_v}(t)] - 1) \\ &= \sigma_E^2(t) \cdot (\exp[R_{A_v}(t)] - 1)\end{aligned}\quad (13.28c)$$

### Selection Response in $\sigma_E^2$

Equation 13.28 allows us to map a change  $R$  in mean breeding values under the different models for heritable environmental sensitivity into an expected change in  $\sigma_E^2$ . Responses in  $A_v$  can arise through two different routes. First, the nature of selection can act directly upon  $A_v$ , and we consider such responses here. Second, selection may act directly upon the breeding values  $A_m$  for the trait mean, and if  $A_v$  and  $A_m$  are correlated, an indirect response (Chapter 30) in  $A_v$  occurs. This is examined in detail in Chapter 34. How important such indirect responses are is unclear, as to date only a few studies have attempted to estimate the correlation between  $A_m$  and  $A_v$ . If the coefficient of variation  $\sigma_z/\mu_z$  is to remain roughly constant under selection, then we expect  $A_m$  and  $A_v$  to be positively correlated, with larger breeding values for the trait resulting in larger environmental variances. Ros et al. (2004) did indeed find a positive correlation between the two breeding values for adult weight in the snail *Helix aspersa*. On the other hand, Sorensen and Waagepetersen (2003) found evidence of negative genetic correlations between breeding values for trait mean and the environmental variance for pig litter size.

To examine how selection on a phenotype maps into selection on  $A_m$  and  $A_v$ , we first consider a quadratic fitness model of stabilizing selection. Here, the expected fitness of an individual with phenotypic value  $z$  is

$$W(z) = 1 - s(z - \theta)^2. \quad (13.29a)$$

where  $\theta$  is the optimal trait value and  $s$  the strength of stabilizing selection. This is a weak selection model, as  $W \geq 0$  only for sufficiently small  $s$  relative to the total variance of  $z$ . Note

that if we take  $s < 0$ , then Equation 13.29a becomes a model of (weak) disruptive selection. Gavrillets and Hastings (1994) examined how this phenotypic fitness function translates into selection on  $(A_m, A_v)$  under the multiplicative model. To do so, replace  $z$  by  $A_m + A_v e$  and take the expectation over  $e$ . Noting that

$$(A_m + A_v e - \theta)^2 = (A_m - \theta)^2 + 2(A_m - \theta)A_v e + A_v^2 e^2,$$

and that  $E[e] = 0$ ,  $E[e^2] = \sigma_e^2$ , the expected fitness becomes

$$\begin{aligned} W(A_m, A_v) &= 1 - s E[(A_m + A_v e - \theta)^2] \\ &= 1 - s [(A_m - \theta)^2 + 2(A_m - \theta)A_v E[e] + A_v^2 E[e^2]] \\ &= 1 - s [(A_m - \theta)^2 + A_v^2 \sigma_e^2] \end{aligned} \quad (13.29b)$$

Under, under phenotypic stabilizing selection, there is selective pressure to shift the breeding value of the trait towards  $\theta$  and to decrease  $A_v$  towards zero (and hence direct selection to decrease  $\sigma_E^2$ ). There are two important consequences of this. First, the reduction in phenotypic variance can be much greater than predicted from the simple reduction in the additive variance from the Bulmer effect. Second, there can be cases where the heritability will *increase* under stabilizing selection. Since both additive and environmental variances are decreased, if the decrease in environmental variance is sufficiently larger,  $h^2$  can actually increase.

Now consider the consequences of heritable variation in  $\sigma_E^2$  for the change in variance under directional selection. Gavrillets and Hastings note that under the simple model of directional selection

$$W(z) = 1 + sz, \quad (13.30a)$$

the mean fitness for breeding values under the multiplicative model becomes

$$W(A_m, A_v) = 1 - sE(A_m + eA_v) = 1 - sA_m + A_v E(e) = 1 - sA_m \quad (13.30b)$$

Thus, under this setting, there is no direct selection on the breeding value of the environmental variance. However, if the breeding values  $A_m$  and  $A_v$  are correlated, selection on  $A_m$  also induces a positive change in  $A_v$  (if they are positively correlated), and a decrease in  $A_v$  if they are negatively correlated. While the additive genetic variance in the trait declines from the Bulmer effect, the phenotypic variance can increase provided conditions are such for an increase in  $\sigma_E^2$ .

A similar outcome was noted Hill and Zhang (2004), and Mulder et al. (2007) who assume the additive model for  $\sigma_E^2$  (Equation 13.26b) and truncation selection on a normally-distributed trait. Recall (Equation 10.26) that for a given fraction  $p$  saved under truncation selection on a normally-distributed trait, the selection intensity is  $\bar{i} = \varphi(x_{[1-p]})/p$ , where  $\varphi(x)$  is the unit normal distribution function evaluated at  $x$ , while for a unit normal  $U$   $x_{[1-p]}$  satisfies  $\Pr(U \geq x_{[1-p]}) = p$ . For the special case where  $A_m$  and  $A_v$  are uncorrelated, the expected responses become simply

$$R_{A_m} = h_m^2 \bar{i} \sigma_z \quad (13.31a)$$

$$R_{A_v} = h_v^2 \bar{i} x_{[1-p]} \sigma_z^2 \quad (13.31b)$$

where  $h_m^2$  is the heritability for trait mean and  $h_v^2$  is the heritability for the additive component of trait variance (Equation 13.27b). Equation 13.31a is simply our standard result for the change in mean under truncation selection, while 13.31b is the analog for the change in the breeding value of the variance (under the additive model). Equation 13.31 assumes the trait distribution is approximately normal, and this break downs at extreme trait values

when here is heritable variation in  $\sigma_E^2$  (as  $z$  is no longer normally distributed). Hence, for strong selection these results are biased. As developed in Chapter 34, when  $A_M$  and  $A_v$  are correlated, the response equations are considerably more complex than given by Equation 13.31.

**Example 13.8.** Consider a trait with  $h_m^2 = 0.3$  and  $h_v^2 = 0.03$  (which is a typical value of the known estimates, see Table 13.4). Suppose  $\sigma_z^2 = 100$ . What is the expected response in the mean and  $\sigma_E^2$  following a single generation of truncation selection with  $p = 0.1$ ? Noting that  $\Pr(U > 1.282) = 0.1$ , we have  $x_{[1-p]} = 1.282$  and  $\bar{v} = \varphi(1.282)/0.1 = 1.755$ , giving

$$R_{A_m} = 0.3 \cdot 1.755 \cdot 10 = 5.265, \quad \text{and} \quad R_{A_v} = 0.03 \cdot 1.755 \cdot 1.282 \cdot 100 = 6.750$$

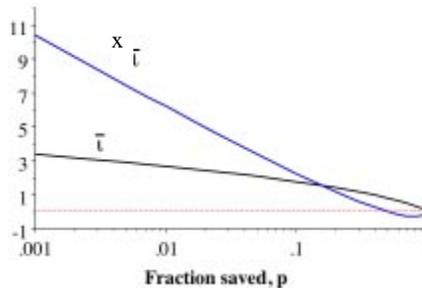
Here selection increased the mean by 5.3 and the environmental variance by 6.75. Now consider stronger selection,  $p = 0.01$ . Here  $x_{[1-p]} = 2.326$  and  $\bar{v} = 2.666$ , giving

$$R_{A_m} = 0.3 \cdot 2.666 \cdot 10 = 7.998, \quad \text{and} \quad R_{A_v} = 0.03 \cdot 2.666 \cdot 2.326 \cdot 100 = 18.603$$

This is roughly a 50% increase in the mean, but a 275% increase in the variance.

As Example 13.8 illustrates, as truncation selection becomes stronger, there is a disproportionate change in the variance relative to the mean, as selection is choosing outliers, and hence more strongly influenced by genotypes with larger variances. The effect on  $\sigma_E^2$  from directional selection on trait value is thus expected to be greatest under strong selection (Hill and Zhang 2004).

We can quantify this for our model. Inspection of Equation 13.31 shows that the strength of selection on the mean is  $\bar{v}$ , while the strength of selection on  $\sigma_E^2$  is  $\bar{v}x_{[1-p]}$ . As shown in Figure 13.5, from large  $p$  (weak selection as most of the population is saved), selection on the mean dominates, but the two strengths of selection are equal around  $p = 0.16$ , below which selection on the variance is stronger. Further, for  $p > 0.5$  (more than half the population is saved),  $x_{[1-p]} < 0$  and hence weak directional selection results in a slight *decrease* in  $\sigma_E^2$  (Hill and Zhang 2004). The effect is largest around  $p = 0.80$  (only 20 percent of the population culled) and even here the strength of selection is still fairly modest, with  $\bar{v}x = -0.3$ . This slight decrease in  $\sigma_E^2$  under weak directional selection occurs because only low outliers are selected *against*, and such genotypes tend to have slightly higher variances.



**Figure 13.5.** The relative strengths of selection on the mean ( $\bar{v}$ ) and variance ( $x\bar{v}$ ) under truncation selection as a function of the fraction  $p$  saved. The two strengths of selection are equal around  $p = 0.16$ . Note for  $p > 0.5$  there is (weak) selection to *decrease* the variance, as the curve for  $x\bar{v}$  dips below the line indicating a value of zero.

Chapter 34 examines the joint response in the more complex case where the two breeding values are correlated. As expected given our earlier discussion in this chapter, linkage-disequilibrium generated after a generation of selection changes  $\sigma_{A_m}^2$ ,  $\sigma_{A_v}^2$  and  $\sigma_z^2$ , and hence both heritabilities. We examine this in Chapter 34 in a broader context that also considers any change in the genetic covariances  $\sigma_{A_m, A_v}$  as well.

In conclusion, the simple predictions for the change in phenotypic variance (decreasing under directional and stabilizing selection, increasing under disruptive selection) no longer universally hold when there is heritable variation in  $\sigma_E^2$ . The behavior of the additive genetic variance in the trait still follows Bulmer's results (under the infinitesimal model), but predicting the behavior of the total phenotypic variance requires additional information. Bull (1987) notes that when there is selection for the phenotypic variance to change, evolution may favor the displacement of genetic variation by environmental variance, and vice-versa, depending on the ecological setting. Thus, as Bull points out, "environmental and genetic factors may thus compete to produce a given selected level of phenotypic variance."

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