

# 6

## Short-term Changes in the Variance

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While we have focused on how selection changes the mean of a character, selection also changes its variance as well. In fact, in many cases, selection is expected to be acting more on the variance than the mean, as would occur in the selection against extreme individuals (stabilizing selection). Selection can change the distribution of genotypic (and hence phenotypic) values by two routes: by changing allele frequencies and by generating gametic-phase disequilibrium. Under the infinitesimal model (Chapter 10), the amount of selection acting on any given locus is expected to be 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. This effect can be important even when allele frequencies are rapidly changing.

Here we examine the effects of selection on the variance under the assumptions of the infinitesimal model, discussing selection with assortative mating as a special case. Under the infinitesimal model, any process that increases the phenotypic variance generates positive disequilibrium, while processes that decrease the phenotypic variance generate negative disequilibrium.

### CHANGES IN VARIANCE DUE TO LINKAGE DISEQUILIBRIUM

In the absence of epistasis, gametic-phase disequilibrium does not change the population mean. 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 average effects of the two alleles at locus  $k$  from a random individual. Since  $\sigma_A^2$  is the variance of the sum of average effects over all loci,

$$\sigma^2 \left( \sum_{k=1}^n (a_1^{(k)} + a_2^{(k)}) \right) = 2 \sum_{k=1}^n \sigma^2 (a^{(k)}) + 4 \sum_{k < j}^n \sigma (a^{(j)}, a^{(k)}) \quad (6.1a)$$

$$= 2 \sum_{k=1}^n C_{kk} + 4 \sum_{k < j}^n C_{jk} \quad (6.1b)$$

where  $n$  the number of loci and  $C_{jk}$  is the covariance between allelic effects at loci

$j$  and  $k$ . We can thus decompose the additive variance as

$$\sigma_A^2 = \sigma_a^2 + d \quad (6.2)$$

where  $\sigma_a^2 = 2 \sum C_{kk}$  is the additive variance in the absence of gametic-phase disequilibrium and the disequilibrium contribution  $d = 4 \sum_{j < k} C_{kj}$  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 covariances are very small.

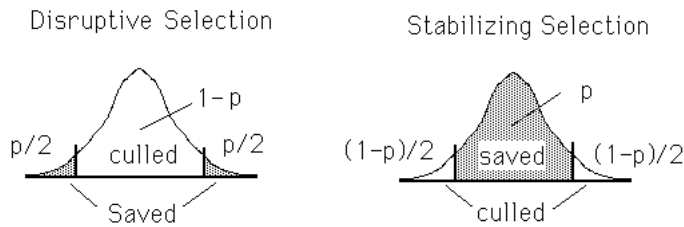
The allelic effects  $a^{(k)}$  (and hence the genic variance  $\sigma_a^2$ ) are altered as allele frequencies change, resulting in a permanent change in  $\sigma_A^2$ . Changes in  $\sigma_a^2$  due to selection strongly depend on the initial distribution of allelic effects and frequencies, both of which are extremely difficult to estimate (Chapters 9, 10). Changes in  $d$ , however, are generally less sensitive to the initial distribution of allelic effects (Sorenson 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 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

$$d(t+1) = \frac{d(t)}{2} \quad (6.3)$$

Thus, with unlinked loci, recombination decreases the genetic variance 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. **Stabilizing selection** (selection against extreme individuals, see Figure 6.1) reduces the phenotypic variance and creates negative  $d$ , while **disruptive selection**

(selection for extreme individuals, see Figure 6.1) 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 (1971b, 1974, 1976a, 1980), whose approach we follow closely. The problem of actually estimating the nature and amount of selection acting on the mean and the variance of a character is the subject of Chapters 12 and 14.



**Figure 6.1** 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 limit, the additive genic variance  $\sigma_a^2$  remains constant and all changes in the additive genetic variance 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 1971b). 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

$$\sigma_z^2(t) = \sigma_E^2 + \sigma_D^2 + \sigma_A^2(t) = \sigma_z^2 + d(t) \quad (6.4a)$$

$$h^2(t) = \frac{\sigma_A^2(t)}{\sigma_z^2(t)} = \frac{\sigma_a^2 + d(t)}{\sigma_z^2 + d(t)} \quad (6.4b)$$

where  $\sigma_z^2 = \sigma_z^2(0)$  is the phenotypic variance before selection in the initial (unselected) population.

Under the infinitesimal model, genotypic values are normally distributed before selection (Bulmer 1971b, 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 normally distributed. Hence, from standard statistical theory (e.g., LW Chapter 8), the regression of offspring phenotypic value  $z_o$  on parental phenotypes is linear and homoscedastic, with

$$z_o = \mu + \frac{h^2}{2}(z_m - \mu) + \frac{h^2}{2}(z_f - \mu) + e \quad (6.5a)$$

where

$$\sigma_e^2 = \left(1 - \frac{h^4}{2}\right) \sigma_z^2 \quad (6.5b)$$

The derivation for Equation 6.5a,b 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 6.5a and assuming random mating and identical selection on both sexes, the phenotypic variance among the offspring from selected parents becomes

$$\begin{aligned} \sigma^2(z_o) &= \frac{h^4}{4} [\sigma^2(z_m^*) + \sigma^2(z_f^*)] + \sigma_e^2 \\ &= \frac{h^4}{2} [\sigma_z^2 + \delta(\sigma_z^2)] + \left(1 - \frac{h^4}{2}\right) \sigma_z^2 \\ &= \sigma_z^2 + \frac{h^4}{2} \delta(\sigma_z^2) \end{aligned} \quad (6.6)$$

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 additive genetic variance  $\sigma_A^2$ . Combining this with Equation 6.3 gives the general recursion for changes in the variance under the infinitesimal model with unlinked loci as

$$d(t+1) = \frac{d(t)}{2} + \frac{h^4(t)}{2} \delta(\sigma_{z(t)}^2) \quad (6.7a)$$

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

$$\begin{aligned} \Delta d(t) &= \Delta \sigma_{z(t)}^2 = \Delta \sigma_A^2(t) \\ &= -\frac{d(t)}{2} + \frac{h^4(t)}{2} \delta(\sigma_{z(t)}^2) \end{aligned} \quad (6.7b)$$

The first term is the decay due to recombination in the disequilibrium contribution from the previous generation, while the second term is amount of the new disequilibrium generated by selection that is passed on to the offspring. At equilibrium,

$$\tilde{d} = \tilde{h}^4 \tilde{\delta}(\sigma_z^2) \quad (6.7c)$$

where tilde denotes an equilibrium value. Equation 6.7 is the analogue of the breeders' equation for predicting changes in variance. Provided the joint distribution of phenotypic and genotypic values remains multivariate normal, under the infinitesimal model the complete dynamics of the phenotypic distribution are described by Equation 6.7 and the breeders' equation  $R(t) = h^2(t)S(t)$ , where  $S(t)$  is the selection differential in generation  $t$ . Equation 6.7 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  $\sigma_A^2$  (and, in turn, the response to selection) cannot be predicted without knowing the  $d$  value in the starting population.

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**Example 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 1971b).

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

An alternative, and instructive, approach to the phenotypic regression argument leading to Equation 6.7 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 1971b), 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

$$A_o = \frac{1}{2}A_m + \frac{1}{2}A_f + e \quad (6.8a)$$

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 1971b, Felsenstein 1981, Tallis 1987). Since  $e$  is the residual of a regression, it is independent of both  $A_f$  and  $A_m$  (LW Chapters 3, 8). Taking variances and assuming random mating (so that  $A_f$  and  $A_m$  are independent),

$$\begin{aligned} \sigma_{A_o}^2(t+1) &= \sigma_{A_o}^2(t+1) = \sigma^2 \left( \frac{A_m(t)}{2} + \frac{A_f(t)}{2} \right) + \sigma_e^2 \\ &= \frac{1}{4} \left[ \sigma_{A_m}^2(t) + \sigma_{A_f}^2(t) \right] + \frac{1}{2} \sigma_A^2(0) \\ &= \frac{1}{2} \sigma_{A^*}^2(t) + \frac{1}{2} \sigma_a^2 \end{aligned} \quad (6.8b)$$

where  $\sigma_{A^*}^2(t)$  is the variance of the breeding values of the selected parents [with assortative mating, Equation 6.8b has an additional term,  $\sigma(A_m, A_f)/2$ ]. Equation 6.8b 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 6.8a 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 expected variance between the mean values of different families. Positive disequilibrium ( $d > 0$ ) increases the between-family component while negative disequilibrium ( $d < 0$ ) decreases it (Wright 1921, Reeve 1953).

An especially important implication of the constant within-family segregation variance is that it tends to largely restore a normal distribution of breeding values following selection. As Equation 6.8a 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 6.8a 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 6.7 and 6.8a 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 (the residual variances are constant, see LW Chapter 3). Consequences of departures from linearity were briefly discussed in Chapter 5 and are developed more fully in Chapter 10. 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 first assume that these departures from normality can be ignored.

### Accounting for Inbreeding and Drift

The effects of drift and regular inbreeding can also be easily accommodated by the infinitesimal model (Verrier et al. 1989, Gibson and Mackay 19xx). The segregation variation (the variance in the residual  $e$  in Equation 6.8a) is just half the additive genic variance of the parents. When genetic drift is present, the genic variance

variance in generation  $t$  becomes

$$\sigma_a^2(t) = \sigma_a^2(0) \left(1 - \frac{1}{2N_e}\right)^t \quad (6.9a)$$

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

$$\Delta d(t) = -\frac{d(t)}{2} \left(1 + \frac{1}{N_e}\right) - \frac{1}{2} \left(1 - \frac{1}{N_e}\right) \kappa h^2(t) \sigma_A^2(t) \quad (6.9b)$$

Hence, when the population size is finite, the variance in any particular generation can be computed by iterating Equation 6.9a and b. Using these update values for the additive variance, one can then use the breeders' equation to predict the change in mean.

Similarly, when the parents are inbred, the segregation variance is also corresponding reduced. Assuming no correlation between the parents (such as would occur with strict selfing), the additive variance recursion equation under the infinitesimal assumptions becomes

$$\sigma_A^2(t+1) = \frac{1}{4} \left[ \sigma_{A_{ma}}^2(t) + \sigma_{A_{fe}}^2(t) \right] + \frac{\sigma_a^2(t)}{2} \left[ 1 - \frac{f_{ma}(t)}{2} - \frac{f_{fe}(t)}{2} \right] \quad (6.9c)$$

where  $f_{ma}$  and  $f_{fe}$  denotes the variance amount of inbreeding in male and female parents.

## 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 6.7. In the general case, this requires numerical iteration to obtain the equilibrium heritability and genetic variance. However, when the phenotypic variance after selection can be written as

$$\sigma_{z^*}^2 = (1 - \kappa) \sigma_z^2 \quad (6.10)$$

where  $\kappa$  is a constant independent of the variance, analytic solutions for the equilibrium variances and heritability can be obtained. Truncation selection — both as we have defined it for directional selection (Chapter 5) and **double truncation** giving disruptive or stabilizing selection (Figure 6.1) — satisfies Equation 6.10. As shown in Table 6.1, for truncation selection  $\kappa$  is strictly a function of the fraction  $p$  of the population saved and the type of truncation used (Figure 6.2).

**Table 6.1** Changes in the phenotypic variance under the various schemes of single- and double-truncation given in Figure 6.1. Assuming the character is normally distributed

before selection, the phenotypic variance after selection is given by  $\sigma_{z^*}^2 = \kappa \sigma_z^2$ , where  $\kappa$  is a function of the fraction  $p$  of individuals saved. Here  $\varphi$  denotes the unit normal density function and  $z_{[p]}$  satisfies  $\Pr(U \leq z_{[p]}) = p$  [so that  $\Pr(U > z_{[1-p]}) = p$ ], where  $U$  is a unit normal random variable. 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

$$\begin{aligned} \kappa &= \frac{\varphi(z_{[1-p]})}{p} \left( \frac{\varphi(z_{[1-p]})}{p} - z_{[1-p]} \right) \\ &= \bar{i} (\bar{i} - z_{[1-p]}) \end{aligned}$$

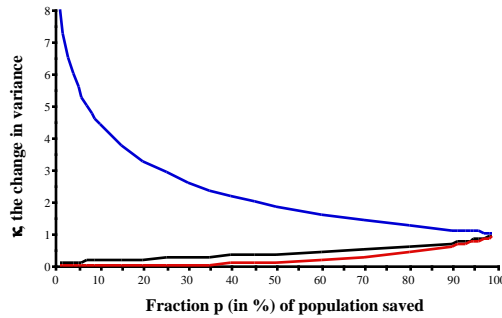
**Stabilizing Truncation Selection:** Middle fraction  $p$  of the distribution saved

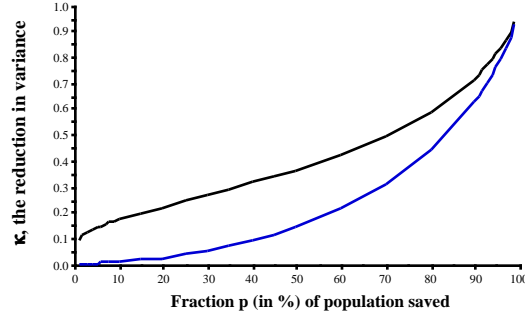
$$\kappa = \frac{2 \varphi(z_{[1/2+p/2]}) z_{[1/2+p/2]}}{p}$$

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

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


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**Figure 6.2** The fraction  $\kappa$  of phenotypic variance that remains after truncation selection (Figure 6.1) as a function of the fraction  $p$  of individuals saved. **Top:** The uppermost curve (values of  $\kappa > 1$ ) corresponds to disruptive selection, while the lower two curves ( $\kappa < 1$ ) correspond to directional (middle curve) and stabilizing selection (lower curve). **Bottom:** Closeup for directional (upper curve) and stabilizing selection (lower curve).

Suppose selection is such that Equation 6.10 is satisfied. We allow for differential selection on the sexes by letting 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),

$$\sigma_{z^*}^2(t) = \frac{\sigma^2(z_f^*(t))}{2} + \frac{\sigma^2(z_m^*(t))}{2} = (1 - \kappa)\sigma_z^2(t) \quad (6.11a)$$

where

$$\kappa = \frac{\kappa_f + \kappa_m}{2} \quad (6.11b)$$

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

$$\delta(\sigma_{z^*}^2(t)) = \sigma_{z^*}^2(t) - \sigma_z^2(t) = -\kappa\sigma_z^2(t) = -\kappa\frac{\sigma_A^2(t)}{h^2(t)} \quad (6.11c)$$

Substituting this into Equation 6.7a recovers the result of Bulmer (1974),

$$\begin{aligned} d(t+1) &= \frac{d(t)}{2} - \frac{\kappa}{2}h^2(t)\sigma_A^2(t) \\ &= \frac{d(t)}{2} - \frac{\kappa}{2}\frac{[\sigma_a^2 + d(t)]^2}{\sigma_z^2 + d(t)} \end{aligned} \quad (6.12)$$

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

$$\tilde{\sigma}_A^2 = \sigma_z^2 \theta, \quad \text{where} \quad \theta = \frac{2h^2 - 1 + \sqrt{1 + 4h^2(1 - h^2)\kappa}}{2(1 + \kappa)} \quad (6.13a)$$

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Since

$$\tilde{\sigma}_z^2 = \sigma_z^2 + (\tilde{\sigma}_A^2 - \sigma_A^2) = \sigma_z^2(1 + \theta - h^2) \quad (6.13b)$$

it follows that the equilibrium heritability is

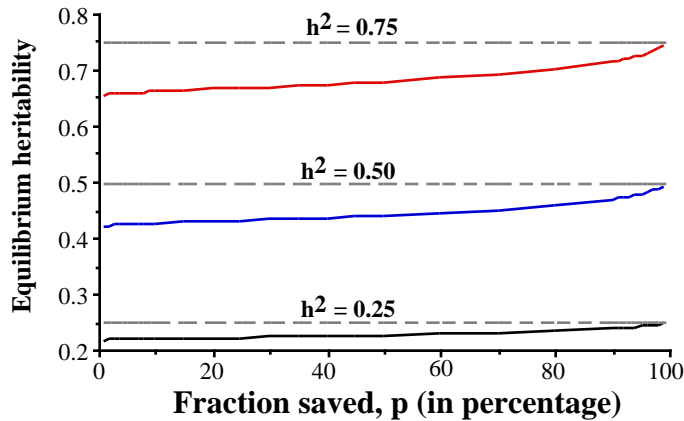
$$\tilde{h}^2 = \frac{\tilde{\sigma}_A^2}{\tilde{\sigma}_z^2} = \frac{\theta}{1 + \theta - h^2} \quad (6.13c)$$

### Directional Truncation Selection

From Table 6.1, direction (or single) truncation selection on a normally-distributed character gives  $\kappa < 1$  and hence the within-generation phenotypic variance is reduced by selection, generating negative disequilibrium and reducing the additive genetic variance. The variance after selection is also often expressed in terms of the selection intensity,  $\bar{i}$ , which is completely determined by the fraction of the population saved  $p$  (Equation 4.14b). Recalling LW Equation 2.15 gives

$$\sigma_{z^*}^2 = [1 - \bar{i} (\bar{i} - z_{[1-p]})] \sigma_z^2 \quad (6.14)$$

The stronger selection (i.e., the smaller the value of  $p$ ), 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 2). 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 6.3, the reduction in heritability is greatest when selection is strongest (i.e., when the fraction saved  $p$  is small).



**Figure 6.3** 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  $3/4$ ,  $1/2$ , and  $1/4$ .

**Example 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,

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

Likewise, evaluating the unit normal gives  $\varphi(0.84) = 0.2803$ , so that (Equation 4.14c)

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

From Table 6.1, the fraction of variance remaining after selection becomes

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

Hence, Equation 6.12 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, 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. How does this reduction in  $\sigma_A^2$  influence the per generation change in mean,  $R(t)$ ? Since  $\bar{i}$  is unchanged, but  $\hat{h}^2$  and  $\sigma_z^2$  change over time, Equation 4.3 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 equilibrium per-generation value of  $\tilde{R} = 1.4 \cdot 0.43 \cdot \sqrt{87.41} = 5.6$ .

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 6.3) 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.

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 6.2). 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.

**Table 6.2** 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 8) 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).

	$\widehat{h}^2(t)$			$\widehat{\sigma}_A^2(t)$		
	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

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

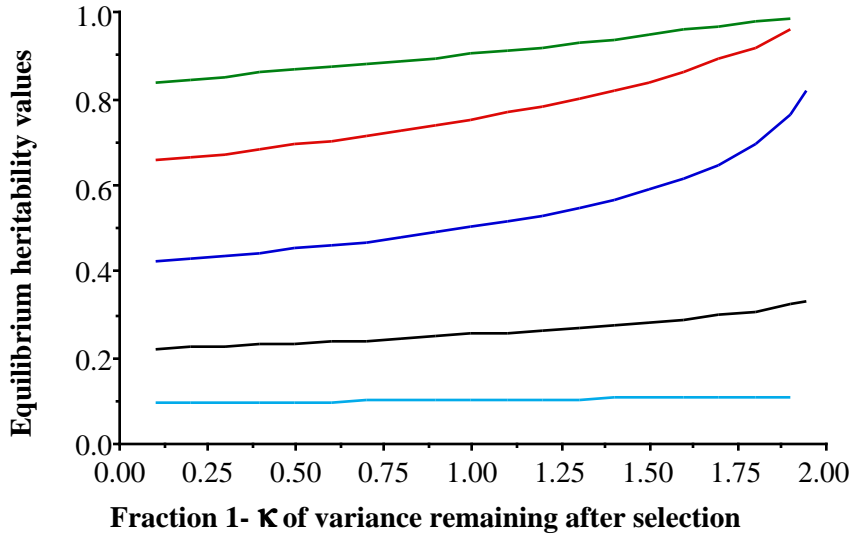
As discussed in Chapter 4, Griffing (1960a,b) showed when additive epistasis is present, gametic-phase disequilibrium increases the response to directional selection, with the response augmented by  $S\sigma_{AA}^2/2\sigma_z^2$ . This (transient) increase in rate of response has been termed the **Griffing 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 (we refer to this change in additive variance as 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.

### Double Truncation Selection

Table 6.1 and Figure 6.2 shows that  $\kappa < 1$  under stabilizing double-truncation selection, so that selection reduces the within-generation phenotypic variance and generates negative disequilibrium. Conversely,  $\kappa > 1$  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 6.4 plots the equilibrium heritability values as a function of the initial heritabilities and the mean fraction  $\kappa$  of phenotypic variance retained after selection.

Consideration of Equation 6.13 shows that under stabilizing selection, the value  $\theta = \widehat{\sigma}_A^2/\sigma_z^2$  (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 ( $\gamma \geq 1 + [4h^2(1 - h^2)]^{-1}$ ) 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 under disruptive selection with  $p$  values ensuring no positive real root of Equation 6.13. 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.



**Figure 6.4** Equilibrium  $h^2$  values as a function of the fraction of variance remained after selection  $\kappa$  and initial heritability  $h^2$ . Curves (from top to bottom) correspond to  $h^2$  values of 0.9 (upper curve), 0.75, 0.5, 0.25, and 0.1 (lower curve).

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 3 illustrates.

**Example 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) in the other. To obtain  $\kappa$  from Table 6.1, first note that for stabilizing selection,  $z_{[1/2+p/2]} = z_{[0.75]}$ , while for disruptive selection  $z_{[1-p/2]} = z_{[0.75]}$ . From normal tables,  $\Pr(U \leq 0.675) = 0.75$ , hence  $z_{[0.75]} = 0.675$ . 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, hence

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

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	6.22	6.72	39.35	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

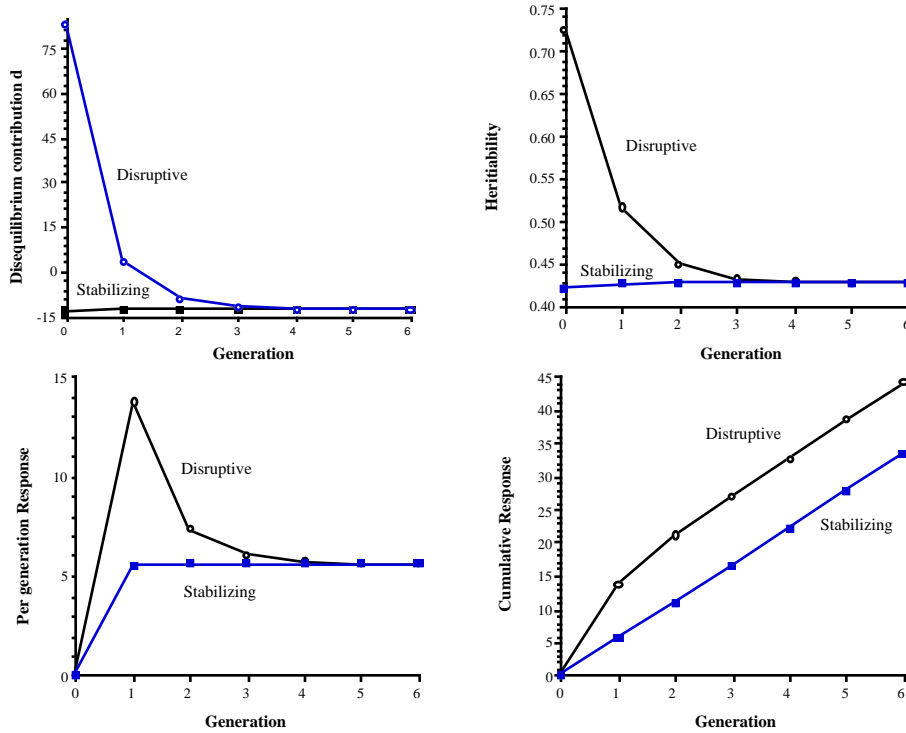
**Stablizing selection**

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 6.2, 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 6.2, 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 6.2). 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 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)

How well do the qualitative predictions from the infinitesimal model for stabilizing and disruptive selection hold up? Experiments examining the effects of selection on variance are reviewed by Prout (1962b), 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 genetic and environmental changes. For example, if heterozygotes have smaller environmental variances, selection favoring heterozygotes will also reduce the environmental variance of the population. Likewise, stabilizing selection could select for increased canalization, lowering the environmental variance (LW Chapter 11).

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 1962b), 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). 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 reanalysis 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 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 in many of these studies, reflecting changes in either the environmental and/or nonadditive genetic variances. On the other hand, for *Drosophila* development time, Prout (1962a) 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. The most direct test of the infinitesimal model predictions for disruptive selection were those of Sorensen (1980) and Sorensen and Hill (1983), 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 in Under Normalizing Selection**

While double-truncation is the common mode of artificial stabilizing selection, the standard model for stabilizing selection in natural populations is to assume **normalizing** (or **non-optimal**) selection (Weldon 1895, Haldane 1954),

$$W(z) = \exp\left(-\frac{(z - \hat{\mu})^2}{2\omega}\right) \tag{6.15}$$

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

$$\mu^* = \mu + \frac{\sigma^2}{\sigma^2 + \omega}(\hat{\mu} - \mu) \quad \text{and} \quad \sigma_*^2 = \sigma^2 - \frac{\sigma^4}{\sigma^2 + \omega} \tag{6.16a}$$

This follows as a special case of the general Gaussian fitness function discussed in Chapter 15. Note that we cannot write  $\sigma_{z^*}^2 = \kappa \sigma_z^2$ , 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 15). Thus, we can apply the breeders' equation to predict changes in the mean and Equation 6.6 to predict changes in the variance (under the infinitesimal model). Here,

$$S = \frac{\sigma^2}{\sigma^2 + \omega}(\hat{\mu} - \mu) \quad \text{and} \quad \delta(\sigma_{z^*}^2) = -\frac{\sigma^4}{\sigma^2 + \omega} \tag{6.16b}$$

The change in mean is thus given by

$$R(t) = h^2(t) S(t) = h^2(t) \frac{\sigma_z^2(t) [\hat{\mu} - \mu(t)]}{\sigma_z^2(t) + \omega} \tag{6.17}$$

implying that the mean converges to  $\hat{\mu}$ . From Equation 6.6, the change in the disequilibrium contribution is given by

$$d(t+1) = \frac{d(t)}{2} - \frac{h^4(t)}{2} \frac{\sigma_z^4(t)}{\sigma_z^2(t) + \omega} = \frac{d(t)}{2} - \frac{1}{2} \left[ \frac{(\sigma_a^2 + d_t)^2}{\sigma_z^2 + d(t) + \omega} \right] \quad (6.17)$$

Note that both directional and stabilizing selection can simultaneously occur with normalizing selection — if  $\mu \neq \hat{\mu}$ , the mean also changes under selection. The change in mean is given by the breeders equation with the appropriate heritability, the change in variance is obtained from the change in the disequilibrium contribution.

Example 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  $\tilde{h}^2 = (50 - 6.46)/(100 - 6.46) = 0.465$ . 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  $\tilde{\sigma}_z^2 = 100 - 6.46 = 93.54$ .

## 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**) generate negative  $d$ , decreasing  $\sigma_A^2$ .

**Results Using the Infinitesimal Model**

Assortative mating is easily incorporated into the infinitesimal model (Fisher 1918, Bulmer 1980). With assortative mating, the parental phenotypic values  $z_f^*$  and  $z_m^*$  are correlated. Note by using  $z^*$  that we have allowed for the possibility of selection prior to mate choice. Writing the variance of a sum as  $\sigma^2(x + y) = \sigma_x^2 + \sigma_y^2 + 2\rho_{xy}\sigma_x\sigma_y$ , Equation 6.8b can be modified to allow for correlation between the parental values, from which it follows that Equation 6.11b is extended to account for assortative mating by considering

$$\gamma = \frac{\kappa_f + \kappa_m}{2} + \rho\sqrt{\kappa_f \kappa_m} \tag{6.18}$$

**correct for new definition of gamma, kappa**

and Equation 6.12 holds using this extended definition of  $\gamma$ . 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 ( $\kappa_f = \kappa_m = 1$ ),  $\gamma = 1 + \rho$  and previous results for assortative mating (LW Equations 7.18-7.20) follow immediately from Equations 6.12, 6.7c, and 6.13 (respectively). More generally, when the amount of selection and assortative mating change each generation,

$$d(t + 1) = \frac{d(t)}{2} + \frac{\gamma(t) - 1}{2} h^2(t) \sigma_A^2(t) \tag{6.19}$$

where  $\gamma(t)$  is given by Equation 6.18 with  $\kappa$  and  $\rho$  taking on the 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  $\kappa(1 + \rho)$ . Positive gametic-phase disequilibrium is generated when  $\gamma > 1$ , while negative disequilibrium is generated if  $\gamma < 1$ . Note that if  $\kappa < 0.5$ ,  $\kappa(1 + \rho) < 1$  and no amount of positive assortative mating can generate positive disequilibrium. However, for all values

of  $\kappa$ , there is some amount of negative assortative mating such that  $\kappa(1 + \rho) < 1$ . Even if selection generates positive disequilibrium (such as disruptive selection), it is possible to find an amount of negative assortative mating such that the net change is to produce 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), Smith and Hammond (1987), and Tallis and Leppard (1988a). Their 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 is at most six percent, consistent with the very small increases seen in experiments. As Example 5 demonstrates, 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.

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**Example 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 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,  $\gamma - 1 = \rho = 0.5$ ) is given by iterating Equation 6.19

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

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

**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 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? Since selection is the same in both sexes,  $\gamma = \kappa(1 + \rho)$ . From Example 2,  $\kappa = 0.213$ , giving the change in  $d$  in the assortatively mated population as

$$d(t + 1) = \frac{d(t)}{2} + \frac{0.213 \cdot 1.5 - 1}{2} h^2(t) \sigma_A^2(t)$$

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 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.