

Lecture 8

Advanced Topics in Selection

Bruce Walsh. July 2005. Asian Institute on Statistical Genetics

We conclude our discussion of selection response by considering two final topics. First, we further consider the response to multivariate selection and introduce the concept of **index selection**, selection on a weighted index of trait values. Next, we consider long-term selection response, starting with infinite population models before turning to the effects of drift and the role of new mutations in long-term response.

More on Multivariate Response

Importance of Genetic Correlations

Recall from Lecture 7 that the multivariate breeders equation can be written (in matrix form) as

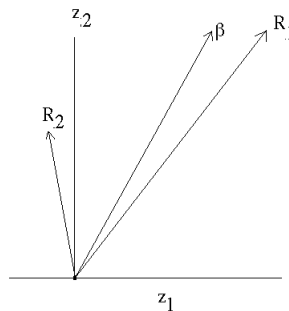
$$\mathbf{R} = \mathbf{G}\mathbf{P}^{-1}\mathbf{S} = \mathbf{G}\boldsymbol{\beta}$$

where $\boldsymbol{\beta}$, the **directional selection gradient**, measures the direct effect of selection acting on each character. A positive value of β_i means direct selection to increase trait i , $\beta_i < 0$ implies selection to decrease the value of trait i , and $\beta_i = 0$ implies no direct selection on trait i . Thus, $\boldsymbol{\beta}$ is a vector that points in the direction that selection is trying to move the trait. However, the response vector is $\mathbf{G}\boldsymbol{\beta}$, not $\boldsymbol{\beta}$. Recall that when multiplying a vector by a matrix that the resulting new vector typically results in a rotation (change in angle) and a scaling (change in length) of the original vector. Thus, the response vector $\mathbf{G}\boldsymbol{\beta}$ is usually different from our objective, $\boldsymbol{\beta}$.

To see this, suppose the (additive) genetic covariance structure for two traits under selection is as follows: $\sigma_A^2(1) = 6$, $\sigma_A^2(2) = 8$ and $\sigma(A_1, A_2) = 4$. Further suppose that there is direct selection to increase trait 1 by one unit and trait two by two units. The resulting \mathbf{G} , $\boldsymbol{\beta}$ and response vector becomes

$$\mathbf{R}_1 = \mathbf{G}\boldsymbol{\beta} = \begin{pmatrix} 6 & 4 \\ 4 & 8 \end{pmatrix} \begin{pmatrix} 1 \\ 2 \end{pmatrix} = \begin{pmatrix} 14 \\ 20 \end{pmatrix}$$

As shown below, this is close to, but departs a bit from, the desired response (compare \mathbf{R}_1 and $\boldsymbol{\beta}$).



Now suppose that the additive genetic covariance between traits is negative, with $\sigma(A_1, A_2) = -4$. The resulting response becomes

$$\mathbf{R}_2 = \mathbf{G}\boldsymbol{\beta} = \begin{pmatrix} 6 & -4 \\ -4 & 8 \end{pmatrix} \begin{pmatrix} 1 \\ 2 \end{pmatrix} = \begin{pmatrix} -2 \\ 12 \end{pmatrix}$$

Thus, even though we select to *increase* trait one, the response results in a *decrease* in this trait, due to the negative genetic correlation. This is an example of a **genetic constraint** — the pattern of genetic variances and covariances is such that it is difficult to cleanly select a desired response. If we are trying to increase two traits, a negative genetic correlation between them hinders our response. Likewise, a positive genetic correlation hinders our response if we are trying to select the traits in opposite directions.

Index Selection

The typical way to select on a combination of traits is to construct a weighted index of trait values,

$$I = \sum_{i=1}^n a_i z_i$$

and select directly on the I values of individuals. Here a_i is the weight on trait i and we are considering selection on n traits. A very compact way to express such sums is to once again use matrix notation. If \mathbf{a} denotes a column vector, then \mathbf{a}^T denotes its **transpose**, which yields a row vector. For example,

$$\mathbf{a} = \begin{pmatrix} a_1 \\ a_2 \\ \vdots \\ a_n \end{pmatrix}, \quad \text{then} \quad \mathbf{a}^T = (a_1 \quad a_2 \quad \cdots \quad a_n)$$

With this notation, our selection index becomes

$$I = \mathbf{a}^T \mathbf{z} = \sum_{i=1}^n a_i z_i$$

The other bit of matrix notation that will prove useful is that of a **quadratic product**,

$$\sum_{i=1}^n \sum_{j=1}^n A_{ij} z_i z_j$$

which can be compactly written in matrix form as

$$\mathbf{z}^T \mathbf{A} \mathbf{z}$$

Quadratic products arise when we compute the variance of a sum. For example,

$$\sigma_I^2 = \sigma^2 \left(\sum_{i=1}^n a_i z_i \right) = \mathbf{a}^T \sigma^2(\mathbf{z}) \mathbf{a} = \mathbf{a}^T \mathbf{P} \mathbf{a} \quad (8.1)$$

here \mathbf{P} is the phenotypic covariance matrix associated with the vector \mathbf{z} , i.e., $P_{ij} = \sigma(z_i, z_j)$. Equation 8.1 is the phenotypic variance of the index I . Similarly, the additive genetic variance of the index becomes

$$\sigma_{A_I}^2 = \mathbf{a}^T \mathbf{G} \mathbf{a} \quad (8.2)$$

Thus, the heritability of the selection index becomes

$$h_I^2 = \frac{\sigma_{A_I}^2}{\sigma_I^2} = \frac{\mathbf{b}^T \mathbf{G} \mathbf{b}}{\mathbf{b}^T \mathbf{P} \mathbf{b}} \quad (8.3)$$

Thus if \bar{i} is the selection intensity on I , the response in I becomes

$$R_I = \bar{i} h_I^2 \sigma_I = \bar{i} \cdot \frac{\mathbf{b}^T \mathbf{G} \mathbf{b}}{\mathbf{b}^T \mathbf{P} \mathbf{b}} \sqrt{\mathbf{b}^T \mathbf{P} \mathbf{b}} = \bar{i} \cdot \frac{\mathbf{b}^T \mathbf{G} \mathbf{b}}{\sqrt{\mathbf{b}^T \mathbf{P} \mathbf{b}}} \quad (8.4)$$

At the risk of mentioning the obvious, the index I is a standard univariate character and hence all of our previous univariate results apply.

How does selection on this index change the vector of underlying character means? It can be shown that the vector of selection differentials for the underlying traits becomes

$$\mathbf{S} = \left(\frac{\bar{i}}{\sigma_I} \right) \cdot \mathbf{P} \mathbf{b} \quad (8.5a)$$

and the vector of responses of the individual trait means becomes

$$\mathbf{R} = \mathbf{G} \mathbf{P}^{-1} \mathbf{S} = \left(\frac{\bar{i}}{\sigma_I} \right) \cdot \mathbf{G} \mathbf{b} = \bar{i} \cdot \frac{\mathbf{G} \mathbf{b}}{\sqrt{\mathbf{b}^T \mathbf{P} \mathbf{b}}} \quad (8.5b)$$

Equation 8.5b shows that the vector of responses \mathbf{R} is unchanged if the index weights are rescaled from \mathbf{b} to $c \cdot \mathbf{b}$, as the constant c cancels out.

One use of a selection index is to combine trait values with information from molecular markers linked to QTLs. This approach is called **MAS**, or **Marker Assisted Selection**. Here an index is constructed that incorporates both the phenotypic value of the trait and the genotypic value for molecular markers that previous work has indicated are linked to QTLs for the trait of interest.

The Smith-Hazel Index

A classic (and very useful) result is the **Smith-Hazel selection index**. Suppose we wish to select in such a way as to maximize the response on the linear combination of traits $\mathbf{a}^T \mathbf{z}$. Smith (1936) and Hazel (1945) found that the response is maximized by selecting on the index $I_s = \mathbf{b}_s^T \mathbf{z}$, where the index weights are given by

$$\mathbf{b}_s = \mathbf{P}^{-1} \mathbf{G} \mathbf{a} \quad (8.6)$$

Example 8.1. Suppose our index is

$$I = z_1 + 2z_2, \quad \text{giving } \mathbf{a} = \begin{pmatrix} 1 \\ 2 \end{pmatrix}$$

Suppose the genetic covariance matrix is as above,

$$\mathbf{G} = \begin{pmatrix} 6 & -4 \\ -4 & 8 \end{pmatrix}$$

and (to make life easy) assume $\sigma^2(z_1) = \sigma^2(z_2) = 10$ and $\sigma(z_1, z_2) = 0$. The resulting Smith-Hazel weights are

$$\mathbf{b}_s = \mathbf{P}^{-1} \mathbf{G} \mathbf{a} = \frac{1}{10} \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} \begin{pmatrix} 6 & -4 \\ -4 & 8 \end{pmatrix} \begin{pmatrix} 1 \\ 2 \end{pmatrix} = \begin{pmatrix} -2/10 \\ 12/10 \end{pmatrix}$$

Since what matters is how the index weights scale with respect to each other, we can write the Smith-Hazel weights as

$$\mathbf{b}_s = \begin{pmatrix} -1 \\ 6 \end{pmatrix}$$

Long-Term Response

So far we been considering the **short-term** response to selection, namely the expected response over a few generations. Focusing back on univariate traits, the initial response is just h^2S . As long as the initial heritability does not change, the response in each future generation is just h^2S . However, as allele frequencies change (either by selection or drift), the genetic variances, and hence h^2 , changes as well. Thus, after a sufficient number of generations, the values expected under short-term response no longer hold, as h^2 has now changed considerably. **Long-term** response deals with the expected response after many generations (typically 20 or more). As we will see, the simple initial genetic variances really offer little insight into long-term response.

Idealized Response in a Large Population

The general pattern expected in long-term response to directional selection is roughly as follows. In the absence of segregating major genes, additive variance (and hence response) is roughly constant over the first few generations giving a nearly linear response (Figure 8.1). There is a slight reduction in the variance due to the generation of gametic-phase disequilibrium, but this is generally small. As generations proceed, sufficient allele frequency change accrues to significantly alter genetic variances. At this point, additive variance can either increase or decrease, depending on the starting distribution of allelic frequencies and effects. Assuming no input of new variation (from mutation or migration), the additive variance generated from the initial variation in the base population eventually declines. Ultimately, a **selection limit** or **plateau** is reached, reflecting fixation of all favorable alleles and loss of additive genetic variance at those loci still segregating (e.g., loci overdominant for the character under selection). If both major and minor alleles influence the character, an initial rapid response due to large changes in allele frequencies at major loci is followed by a much longer period of slower response due to allele frequency changes at loci having smaller effects. Such differences in rates of response can make it difficult to determine whether a selection limit has actually been reached. As the genetic variation in the base population becomes exhausted, the effects of new mutations become extremely important for continued response.

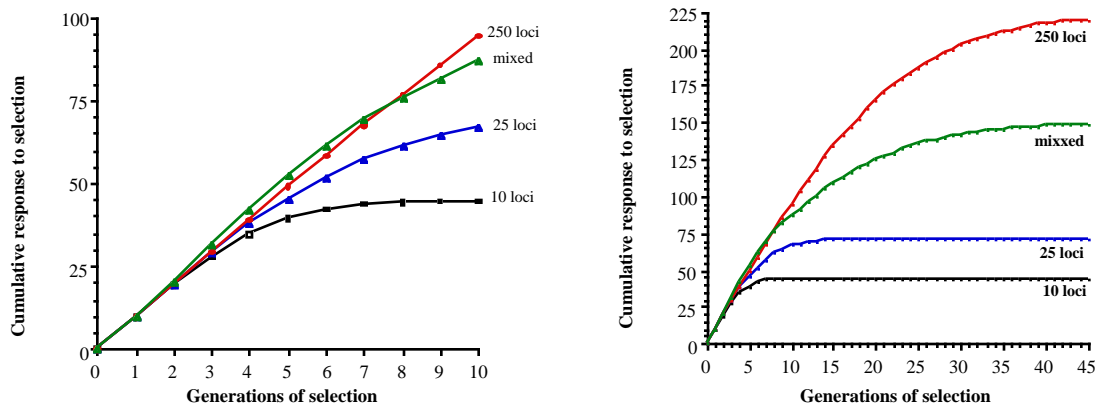


Figure 8.1. Examples of the expected response to selection, here assuming truncation selection (with the upper 20% saved), n identical diallelic loci (at each, the genotypes $AA : Aa : aa$ have genotypic values $2a : a : 0$, and all loci have the same frequency (p) of A). All populations start with $\sigma_A^2(0) = 100$ and $h^2(0) = 0.5$. Curves marked 10, 25, and 250 loci correspond to populations with initial allele frequency $p = 0.5$ and a values of 4.47, 2.82, and 0.89, respectively. The mixed population consists of 5 identical major loci with $p = 0.25$, $a = 5.16$ and 125 identical minor loci with $p = 0.5$, $a = 0.89$. **Left:** Short-term response over the first 10 generations. **Right:** Response over the first 40 generations. Note that the total response increases with the number of loci. In the infinitesimal model limit, the response remains linear over all generations.

Figure 8.1 illustrates differences in the long-term response for four hypothetical populations with the same initial heritability but different numbers of loci. All show essentially the same response over the first few generations. By generation five, allele frequencies have changed enough in the 10- and 25-locus populations to show reduced response, while the 250-locus population shows a roughly constant response through 20–25 generations. The mixed population (5 major loci, each with initial frequency of the favored allele $p = 0.25$, 125 minor loci with $p = 0.5$) shows an enhanced response relative to the others in generations 3 – 7. This results from an increase in heritability as the frequencies of alleles with large effects increase from $1/4$ to $1/2$, increasing the additive variance contributed by these loci. If rare recessives are present, there can be a considerable time lag until the enhanced response appears.

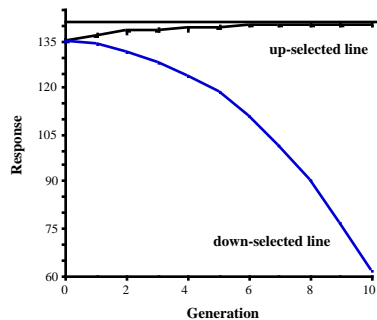


Figure 8.2. With strong directional dominance, an apparent selection limit can result when alleles favored by selection are dominant. Here the genotypes $\mathbf{AA} : \mathbf{Aa} : \mathbf{aa}$ have values $2a : 2a : 0$, and we ignore epistasis and gametic-phase disequilibrium. The population consists of 25 identical loci, with $a = 2.82$ and initial frequency $p_A = 0.8$. Truncation selection with the upper (or lower) 20% of the population saved is assumed. If all loci are fixed for the favored allele, the selection limit is 141 (indicated by the horizontal line). There is little response to upward selection and the population appears at a selection limit, even though there is still considerable genetic variation. Conversely, the down-selected line responds very rapidly.

If alleles are favored by selection are dominant, response slows down considerably as these alleles become common, reflecting the rarity of homozygous recessives. In such cases, response can be so slow that the population appears to be at a limit. However, as Figure 8.2 demonstrates, reverse selection on these populations can result in a fairly rapid response. This apparent limit due to the very slow removal of recessives can be partly overcome by inbreeding. By increasing the frequency of homozygotes relative to a random mating population, inbreeding greatly improves the efficiency of selection against heterozygotes, allowing favorable dominant alleles to be more rapidly fixed.

Example 8.2. Falconer (1971) examined an apparent limit in a mouse line selected for increased litter size. Four sublines were created from this plateaued line and subjected to inbreeding and selection. Selection on a new line formed by crossing these inbred-selected lines gave an improvement of 1.5 mice/litter over the original limit. Falconer's interpretation was that many recessive alleles decreasing litter size were segregating in the apparently plateaued line, some of which were lost in during inbreeding within sublines. Crossing the inbred-selected lines generated a population segregating fewer recessives (i.e., fixed for more of the dominant alleles), facilitating response.

Drift and Long-term Response

It is important that we make the distinction between long-term and **asymptotic** response. The former is the total response attributable to the existing variation at the time of selection, while the latter is the expected eventual rate of response due to the input of new mutation. When the effective population size is small, essentially all of the observed response is due to the initial variation, with the population reaching an apparent selection limit until the appearance of new mutations allows

further response. In larger populations, however, these two components of response (that from initial variation and that from new mutations) become more difficult to separate, and no selection limit may be observed when in fact all of the initial variation has become exhausted. Much of the theory of long-term response has ignored mutation and we examine this first. Even though this theory is unrealistic over very long time scales, it often provides a good description of how the population exhausts its initial variation.

Basic Theory

We expect response to selection in very small populations to be significantly influenced by drift, showing less total response than expected in larger populations starting with the same initial genetic variance. A fairly extensive theory examining the effects of drift on long-term response (the utilization of the existing genetic variation) has been developed, starting with the extremely influential paper of Robertson (1960). Most of this theory is based on summing over single-locus results, and we take this approach unless stated otherwise (this assumes that epistasis and linkage effects can be ignored). As before, we first consider a single diallelic locus (which we index as locus i) where the genotypes $aa : Aa : AA$ have genotypic values (for the character under selection) of $0 : a(1+k) : 2a$. Let p_t denote the frequency of A at this locus at generation t , $\Delta_i(t)$ be the contribution to total response from this locus in generation t , and $u_i(p_0)$ the probability that A is ultimately fixed at this locus given it starts at frequency p_0 . The total response is given by summing over all loci, $R(t) = \sum_i \Delta_i(t)$. Under drift, both p_t and $\Delta_i(t)$ are random variables and (assuming genotypes are in Hardy-Weinberg proportions) are related by

$$\begin{aligned}\Delta_i(t) &= m_i(p_t) - m_i(p_0) \\ &= 2a \left[p_t - p_0 + k \left(p_t(1 - p_t) - p_0(1 - p_0) \right) \right]\end{aligned}\quad (8.7a)$$

where $m_i(p)$ is the mean contribution from locus i given allele A is at frequency p . The expected contribution (at generation t) from this locus is

$$E[\Delta_i(t)] = 2a \left[E(p_t) - p_0 + k \left(E[p_t(1 - p_t)] - p_0(1 - p_0) \right) \right]\quad (8.7b)$$

Since A is ultimately either fixed ($p_\infty = 1$) or lost ($p_\infty = 0$), $E(p_t)$ converges to

$$1 \cdot u_i(p_0) + 0 \cdot [1 - u_i(p_0)] = u_i(p_0)$$

while $E[p_t(1 - p_t)]$ converges to zero. Thus, the limiting expected contribution from locus i is

$$E[\Delta_i(\infty)] = 2a \left[u_i(p_0) - p_0 - k \left(p_0(1 - p_0) \right) \right]\quad (8.8a)$$

Two cases of special interest are when A is additive ($k = 0$), in which case

$$E[\Delta_i(\infty)] = 2a [u_i(p_0) - p_0]\quad (8.8b)$$

and when A is recessive ($k = -1$),

$$E[\Delta_i(\infty)] = 2a [u_i(p_0) - p_0^2]\quad (8.8c)$$

The variance (and indeed all higher moments) of the total response at the selection limit are easily computed, as $\Delta_i(\infty)$ takes on only two values,

$$\begin{aligned}\Delta_i(\infty) &= 2a - m_i(p_0) \quad \text{with probability } u_i(p_0) \\ &= 0 - m_i(p_0) \quad \text{with probability } 1 - u_i(p_0)\end{aligned}\quad (8.9)$$

In particular,

$$\begin{aligned}\sigma^2 [\Delta_i(\infty)] &= E [\Delta_i^2(\infty)] - \left(E [\Delta_i(\infty)] \right)^2 \\ &= 4a^2 u_i(p_0) [1 - u_i(p_0)]\end{aligned}\quad (8.10a)$$

With weak selection, $u_i(p) \simeq p$ (i.e., the allelic dynamics are governed by drift), implying

$$\sigma^2 [R(\infty)] \simeq 4 \sum a^2 p_0 (1 - p_0) \quad (8.10b)$$

If all loci are additive, this is simply $2\sigma_A^2(0)$, the expected between-line divergence under pure drift. Under sufficiently strong selection, almost all favorable alleles are fixed and the variance is close to zero as $u_i(p) \simeq 1$.

The variance in the selection limit across replicate lines has a direct bearing on whether further response can occur by crossing plateaued lines and then reselecting. If drift has played a significant role in response, a line formed by crossing replicate plateaued lines should show further response to selection, as each line should be fixed for a considerable number of unfavorable alleles.

Robertson's Theory of Selection Limits

Equations 8.7 - 8.10 are fairly general, assuming only Hardy-Weinberg, no epistasis, and that single-locus results can be added across loci. To proceed further, we need explicit expressions for $u_i(p)$ (to describe the limit) and $E(p_t)$ and $E[p_t(1 - p_t)]$ (to describe the dynamics). The most complete description, due to Robertson (1960), is for additive alleles, and gives the expected response from locus i after t generations of selection as

$$\Delta_i(t) = 2a[E(p_t) - p_0] \simeq 2N_e \left(1 - e^{-t/2N_e}\right) \left(\frac{Sa}{\sigma_z^2}\right) 2a p_0(1 - p_0) \quad (8.11a)$$

This can be simplified further by noting that $2a^2 p_0(1 - p_0)$ is the initial additive variance contributed by the locus. Since we assumed no gametic-phase disequilibrium, summing over all loci gives

$$R(t) \simeq 2N_e \left(1 - e^{-t/2N_e}\right) \frac{S\sigma_A^2(0)}{\sigma_z^2} \quad (8.11b)$$

Note that $S\sigma_A^2(0)/\sigma_z^2 = S h^2(0)$ is the expected response in the first generation, provided that the conditions for the breeders' equation hold. Thus,

$$R(t) \simeq 2N_e \left(1 - e^{-t/2N_e}\right) R(1) \quad (8.12a)$$

giving an expected limiting total response of

$$R(\infty) \simeq 2N_e R(1) \quad (8.12b)$$

Thus the total expected response is simply $2N_e$ times the initial response, as first suggested by Dempster (1955) and formally derived by Robertson (1960).

Another quantity of interest is the expected half-life of response, $t_{0.5}$, the time required to obtain half the final response. Recalling Equation 17.21a and solving $1 - e^{-t_{0.5}/2N_e} = 1/2$ gives the expected half-life as

$$t_{0.5} = N_e \ln 2 \simeq 1.4N_e \quad (8.13)$$

This is an upper limit with the half-life decreasing as the product $N_e\bar{i}$ increases. An observed half-life considerably below that predicted by Equation 8.13 suggests that a large portion of the response is due to fixation of favorable alleles by selection, as selection (when it dominates) changes allele frequencies much faster than drift.

Mutation and Long-term Response

There is strong evidence that new mutants contribute to selection response even during the short time scales of many “long-term” laboratory experiments. The selection limit resulting from drift and selection removing all initial genetic variation is thus an artifact of time scale as it ignores this mutational contribution. Even if an observed limit is due to a balance between natural and artificial selection, new mutations with less deleterious pleiotropic effects on fitness can arise, resulting in further response.

Letting σ_m^2 represent the mutation variance, then the expected additive genetic variance at generation t is given by

$$\sigma_A^2(t) \simeq 2N_e\sigma_m^2 + [\sigma_A^2(0) - 2N_e\sigma_m^2] \exp(-t/2N_e) \quad (8.14)$$

Setting $\sigma_A^2(0) = 0$ gives the additive variance contributed entirely from mutation as

$$\sigma_{A,m}^2(t) \simeq 2N_e\sigma_m^2 [1 - \exp(-t/2N_e)] \quad (8.15a)$$

Hence, the rate of response at generation t from mutational input is

$$r_m(t) = \bar{i} \frac{\sigma_{A,m}^2(t)}{\sigma_z} \simeq 2N_e \bar{i} \frac{\sigma_m^2}{\sigma_z} [1 - \exp(-t/2N_e)] \quad (8.15b)$$

For $t \gg 2N_e$, the per-generation response approaches an asymptotic limit of

$$r_m(\infty) = 2N_e \bar{i} \frac{\sigma_m^2}{\sigma_z} \quad (8.16)$$

Assuming $\sigma_A^2(0) = 0$, half this rate occurs when $t \simeq 1.4N_e$ (Hill 1982b,c). There are several ways to intuit the value of the asymptotic limit. From Robertson’s theory, we expect the final response to be $2N_e$ times the initial response $R(0)$, which for new mutants arising in any particular generation is $R(0) = \bar{i} \sigma_m^2 / \sigma_z$. Alternatively, note that the equilibrium additive variance is $2N_e\sigma_m^2$, which gives an Equation 8.16.

Of some interest is the expected number of generations until response from mutational input exceeds that contributed by the initial variation. Let t^* be the generation when the per-generation response from both sources is equal. Here the initial additive variance remaining at generation t^* equals the new additive variance generated by generation t^* ,

$$\sigma_A^2(0) \exp(-t^*/2N_e) = 2N_e \sigma_m^2 [1 - \exp(-t^*/2N_e)]$$

This equation has the solution

$$t^* = 2N_e \ln(1 + \Psi) \quad (8.17a)$$

where $\Psi = \sigma_A^2(0)/(2N_e\sigma_m^2)$. Denoting the initial heritability by h^2 , a little rearrangement give

$$\Psi = \frac{h^2}{(1 - h^2) 2N_e (\sigma_m^2/\sigma_E^2)} \quad (8.17b)$$

The average value of σ_m^2/σ_E^2 is approximately 0.005. Using this value, it is seen that t^* is only rather weakly dependent on N_e (see Figure 8.3). If $\Psi \ll 1$, so that the expected additive variance at the mutation-drift equilibrium exceeds the initial additive variance ($\sigma_A^2(0) \ll 2N_e\sigma_m^2$), then using the approximation $\ln(1+x) \simeq x$ for small $|x|$, we have

$$t^* \simeq 2N_e\Psi = \frac{h^2}{(1-h^2)(\sigma_m^2/\sigma_E^2)} \quad (8.17c)$$

Again using $\sigma_m^2/\sigma_E^2 = 0.005$ gives $t^* \simeq 200h^2/(1-h^2)$. This translates into 11, 22, and 67 generations until the rate of response from mutational input exceeds the rate of response due to initial variation for h^2 values of 0.05, 0.10, and 0.25 (respectively).

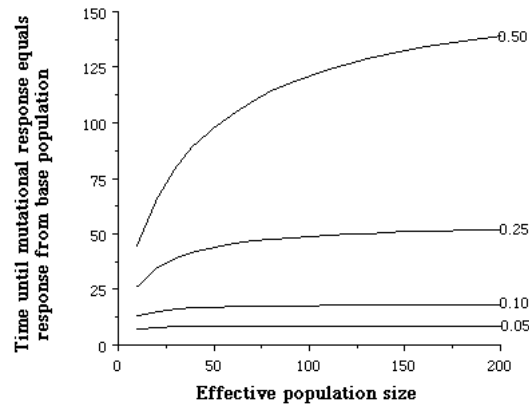


Figure 8.3. The expected generation at which response due to mutational input equals the response due to initial variation in the base population. The four curves correspond to initial heritabilities of 0.05, 0.10, 0.25 and 0.50.

Lecture 8 Problems

1. Suppose traits 1 and 2 have the following covariance structure and selection differential

$$\mathbf{P} = \begin{pmatrix} 10 & 5 \\ 5 & 10 \end{pmatrix}, \quad \mathbf{G} = \begin{pmatrix} 8 & -5 \\ -5 & 8 \end{pmatrix}, \quad \mathbf{S} = \begin{pmatrix} 5 \\ 5 \end{pmatrix}$$

- a: What is β ?
- b: What is the expected response?
- c: Suppose our goal is to maximize response on the index $I = z_1 + 3z_2$. What are the Smith-Hazel weights?

Solutions to Lecture 8 Problems

1. Suppose traits 1 and 2 have the following covariance structure

a:

$$\mathbf{P}^{-1} = \frac{1}{10 \cdot 10 - 5 \cdot 5} \begin{pmatrix} 10 & -5 \\ -5 & 10 \end{pmatrix} = \frac{1}{75} \begin{pmatrix} 10 & -5 \\ -5 & 10 \end{pmatrix}$$
$$\boldsymbol{\beta} = \mathbf{P}^{-1}\mathbf{S} = \frac{1}{75} \begin{pmatrix} 10 & -5 \\ -5 & 10 \end{pmatrix} \begin{pmatrix} 5 \\ 5 \end{pmatrix} = \begin{pmatrix} 1/3 \\ 1/3 \end{pmatrix}$$

b:

$$\mathbf{R} = \mathbf{G}\boldsymbol{\beta} = \begin{pmatrix} 8 & -5 \\ -5 & 8 \end{pmatrix} \begin{pmatrix} 1/3 \\ 1/3 \end{pmatrix} = \begin{pmatrix} 1 \\ 1 \end{pmatrix}$$

Note that for both traits, $h^2 = \sigma_A^2/\sigma_P^2 = 8/10 = 0.8$, so that (just considering each trait independently), the expected response would be $h^2S = 0.8 \cdot 5 = 4$, much greater than what is observed.

c: For this index,

$$\mathbf{a} = \begin{pmatrix} 1 \\ 3 \end{pmatrix}$$

Here

$$\mathbf{b}_s = \mathbf{P}^{-1}\mathbf{G}\mathbf{a} = \frac{1}{75} \begin{pmatrix} 10 & -5 \\ -5 & 10 \end{pmatrix} \begin{pmatrix} 8 & -5 \\ -5 & 8 \end{pmatrix} \begin{pmatrix} 1 \\ 3 \end{pmatrix} = \begin{pmatrix} -2.2 \\ 3 \end{pmatrix}$$

So the Smith-Hazel index is to select on

$$I = -2.2z_1 + 3z_2$$

which is identical to selecting using the weights

$$I = -z_1 + 1.36z_2$$