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THE EFFECT OF SELECTION ON GENETIC VARIABILITY

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This paper arose from the following apparently paradoxical consideration. If a metric character is subjected to stabilizing selection, which reduces the phenotypic variance in each generation by weeding out extreme deviations from the norm, one would expect the genetic variance to be reduced proportionately in each generation and ultimately to be eliminated altogether. But Crow and Kimura (1970) show that the change in the variance under selection decreases as the number of loci involved increases and must eventually tend to zero when the number of loci becomes effectively infinite. Also, most metric characters investigated in natural and domesticated populations possess much genetic variability, and are likely subject to stabilizing selection (Falconer 1960). The intuitively obvious argument that stabilizing selection will lead to the rapid elimination of genetic variability must therefore be wrong. Why?

To state the problem precisely, consider a metric character whose phenotypic value in an individual, Y , measured before the operation of selection can be regarded as the sum of the genotypic value, G , and an independent environmental deviation, E , so that $Y = G + E$. The total phenotypic variance, V , is therefore the sum of a genetic component, V_G , and an environmental component, V_E , so that

$$V = V_G + V_E. \quad (1)$$

It is emphasized that these variances are assumed to be measured in the population *before* selection; the symbol V' will be used for the total variance measured *after* the operation of selection. The genetic variance may contain an additive and a dominance component,

$$V_G = V_A + V_D; \quad (2)$$

but it is assumed that there are no epistatic components, so that there is no interaction between the effects of different loci. For any one individual, the total genotypic value, G , may therefore be written as the sum of the genotypic values from the different loci affecting the character,

$$G = \sum_{i=1}^N g_i, \quad (3)$$

where g_i is the genotypic value at the i th locus and N is the number of loci involved. It should be noted that g_i depends on both alleles represented at the i th locus and may thus contain a dominance component due to the interaction between the two alleles at that locus. For the population we may write

$$V_G = \text{Var}(G) = \text{Var}\left(\sum_{i=1}^N g_i\right) = \sum_{i=1}^N \text{Var}(g_i) + \sum_{i \neq j} \text{Cov}(g_i, g_j), \quad (4)$$

where $\text{Var}(g_i)$ is the variance at the i th locus and $\text{Cov}(g_i, g_j)$ is the covariance between the i th and j th loci. Under random mating in the absence of selection, the covariances would, of course, be zero, so that equation (4) would take the simpler form

$$V_G = \sum_{i=1}^N \text{Var}(g_i). \quad (5)$$

It should be observed that the genetic variance V_G refers to the variability of the total genotypic values, G , between individuals in the population, *not* to the variability of the g_i 's at different loci in the same individual. Likewise $\text{Var}(g_i)$ refers to the variability at a fixed locus, and $\text{Cov}(g_i, g_j)$ to the covariance at a fixed pair of loci, over different individuals in the population.

We shall now suppose that the number of loci involved is effectively infinite and that the environmental deviation, E , is normally distributed. This model was first studied by Fisher (1918) and is usually called the infinitesimal model. Under random mating in the absence of selection, it can now be shown, not only that the character is normally distributed, but also that the phenotypic values of two or more related individuals follow a multivariate normal distribution in the absence of linkage; this result also holds in the presence of linkage if the related individuals are identical twins or offspring and one or both parents, but not if they are related in any other way. These results are derived in the Appendix. Their importance is that, if the phenotypic values in a set of related individuals are distributed as multivariate normal variables, any regression of one of them on the rest must be linear and homoscedastic. The regression equation and the residual variance can be calculated from the correlations between the individuals by standard regression theory; the calculations for the three special cases considered in this paper are given in the Appendix. Note that these results hold good even when there is dominance, although the correlations between relatives and hence the regression equation depend on the degree of dominance. However, these are only limiting results. When the number of loci is small, the variability will usually be larger in the middle than at the periphery of the regression line or plane, owing to the greater amount of heterozygosis, and dominance will usually cause departures from linearity.

Let us now consider the effect of one generation of selection on the variance in the next generation. In the absence of selection, the joint regression of offspring (Y) on both parents (P_1 and P_2) is given by

$$Y = a + bP_1 + bP_2 + e, \quad (6)$$

where

$$b = \frac{1}{2}h^2 \\ \text{Var}(e) = (1 - \frac{1}{2}h^4)V. \quad (7)$$

These results are derived in the Appendix; h^2 is the heritability in the strict sense, $h^2 = V_A/V$. This regression equation holds good whatever values are attributed to the independent variables, P_1 and P_2 , and is therefore unaffected if we allow selection to operate in the parental generation. If selection acts in such a way as to change the variance in the parental generation from V to $V' = V + dV$, and if individuals mate at random after selection, then the variance in the offspring generation will become

$$\begin{aligned} \text{Var}(Y) &= b^2\text{Var}(P_1) + b^2\text{Var}(P_2) + \text{Var}(e) \\ &= 2b^2(V + dV) + \text{Var}(e) = V + \frac{1}{2}h^4dV. \end{aligned} \quad (8)$$

Thus, the variance of the offspring distribution is changed by the amount $\frac{1}{2}h^4dV$; this must be a change in the genetic variance, since the environmental variance is assumed to be constant. (If selection acts only on one parent, as will be the case for a maternal character like clutch size or egg weight, the change in the variance will only be half the above amount.) Under stabilizing selection, as under most types of selection except disruptive selection, dV will be negative, so that the variance will of course be reduced. But if this process is repeated in each generation, the genetic variance will decrease quite rapidly until the heritability becomes negligible. How can this be reconciled with the conclusion of Crow and Kimura (1970) that there is no change in the variance under selection when the number of loci is effectively infinite and with the fact that the heritability is quite high in many characters which are probably subject to stabilizing selection? Conversely, if dV is positive, one is led by the same argument to the absurd conclusion that fresh genetic variability is continually generated by the action of disruptive selection.

The resolution of this paradox depends on the fact that the genetic variance under selection is the sum of two components, which will be called the equilibrium genetic variance and the disequilibrium contribution. The disequilibrium contribution is the component of the genetic variance due to the phenomenon usually known as "linkage disequilibrium" but which will here be called "joint disequilibrium" since its existence does not depend on linkage; the equilibrium genetic variance is the value which the genetic variance would have in the absence of disequilibrium. From equation (4), we find that the genetic variance can be expressed as

$$V_G = \sum_{i=1}^N \text{Var}(g_i) + \sum_{i \neq j} \text{Cov}(g_i, g_j). \quad (9)$$

The first term on the right-hand side of this equation is the equilibrium genetic variance, and the second term is the disequilibrium contribution.

Under random mating in the absence of selection, the disequilibrium contribution would, of course, be zero; but this will not be true under selection, since one generation of random mating is sufficient to restore Hardy-Weinberg equilibrium at each individual locus but not to restore joint equilibrium between pairs of loci (e.g., Li 1955 or Falconer 1960). In particular, stabilizing selection, which favors values near the mean, will introduce a negative correlation between pairs of loci, part of which will be retained in the next generation, so that the disequilibrium contribution will be negative and the actual genetic variance will be less than the equilibrium genetic variance. If selection were relaxed, joint equilibrium would gradually be restored, and the genetic variance would return to its equilibrium value; if none of the loci were linked, the disequilibrium contribution would be halved in each generation after selection ceases, but linkage would slow down the rate of return to equilibrium.

We shall now show that, under the infinitesimal model, the change in the genetic variance demonstrated in equation (8) is due entirely to the disequilibrium contribution. To prove this, let us consider the effect of one generation of selection on the variance among their grandchildren. In the absence of linkage and selection, the regression of a grandchild on his four grandparents is given by

$$Y = a + bG_1 + bG_2 + bG_3 + bG_4 + e, \quad (10)$$

where

$$b = \frac{1}{4}h^2 \\ \text{Var}(e) = (1 - \frac{1}{4}h^4)V, \quad (11)$$

as derived in the Appendix. This formulation is only valid in the absence of linkage, whereas the formulation of the parent-offspring regression in equations (6) and (7) is also valid in the presence of linkage. If we now allow selection to change the variance in the grandparental generation from V to $V + dV$, then it can be shown in exactly the same way as before that the variance among the grandchildren is

$$\text{Var}(Y) = V + \frac{1}{4}h^4dV. \quad (12)$$

It is essential to this argument that there be no selection in the parental generation, since it is assumed that all surviving individuals in the grandparental generation have the same chance of having grandchildren. Thus, the change in the variance has been halved when compared with its value in the generation immediately following selection (see eq. [8]). This is exactly what would be expected in the absence of linkage if the change in the variance is restricted entirely to the disequilibrium contribution and proves that there has been no permanent change in the equilibrium genetic variance. This argument is based on the assumption that there is no linkage; but there is no reason to suppose that it should not also hold in the presence of linkage, with the modification that the disequilibrium contribution is not halved but is reduced by a fraction somewhat less than one-half in each

generation after selection ceases. The argument on which equation (8) is based is valid in the presence of linkage, so that the disequilibrium contribution in the offspring generation generated by selection in the previous generation is given by $\frac{1}{2}h^4dV$ whether linkage is present or not.

It can be concluded that the change in the variance resulting from one generation of selection does not reflect a permanent change in the equilibrium genetic variance but is due entirely to the correlation between loci induced by selection and therefore disappears rapidly when selection ceases. It must be stressed, however, that this is a limiting result, which is only true exactly when the effective number of loci is infinite. The restriction of the argument to an infinite number of loci cannot be removed in any general way since, for the equilibrium variance to remain constant, there must be no change in the mean gene frequency even if the phenotypic mean changes as a result of selection, which implies an infinite number of loci. It is obvious that selection can cause a permanent change in the equilibrium genetic variance in any actual situation, however large the number of loci may be. The limiting result obtained here shows, however, that the magnitude of any permanent change must decrease as the number of loci involved increases and that when the number of loci is large it is likely to be much less than the temporary change due to the induced correlation between loci, that is, to joint disequilibrium.

Before considering the effect of several generations of selection, we must investigate the effect of the disequilibrium contribution on the genetic structure of the population. To do this, we consider the regression of an individual on one parent and on both grandparents on the same side, which in the absence of linkage is given by

$$Y = a + bP + cG_1 + cG_2, \quad (13)$$

where

$$\begin{aligned} b &= \frac{1}{2}h^2(2 - h^2)/(2 - h^4), \\ c &= \frac{1}{2}h^2(1 - h^2)/(2 - h^4). \end{aligned} \quad (14)$$

The derivation is in the Appendix. If selection acts on the grandparents so as to change their variance from V to $V + dV$, then the covariance of Y and P is given by

$$\begin{aligned} \text{Cov}(Y, P) &= \text{Cov}(bP + cG_1 + cG_2, P) = b\text{Var}(P) + c\text{Cov}(G_1 + G_2, P) \\ &= b\text{Var}(P) + \frac{1}{2}ch^2\text{Var}(G_1 + G_2), \text{ from equations (6) and (7),} \\ &= b(V + \frac{1}{2}h^4dV) + ch^2(V + dV) = \frac{1}{2}(h^2V + \frac{1}{2}h^4dV). \end{aligned} \quad (15)$$

The additive genetic variance in the parental generation is twice the covariance between parent and offspring, which is equal to $h^2V + \frac{1}{2}h^2dV$; this is the sum of the additive genetic variance before selection and of the disequilibrium contribution. The disequilibrium contribution can therefore be regarded as a component of the additive genetic variance.

Let us now consider the effect of several generations of selection. We

shall write A instead of V_A for the additive genetic variance, and we shall suppose that in the i th generation of selection the additive genetic variance is A_i , the phenotypic variance (measured before selection) V_i , the heritability $h_i^2 = A_i/V_i$, and the disequilibrium contribution d_i ; if the additive genetic variance and the phenotypic variance before the onset of selection were A_0 and V_0 , respectively, then

$$\begin{aligned} A_i &= A_0 + d_i, \\ V_i &= V_0 + d_i. \end{aligned} \quad (16)$$

If the effect of selection in the i th generation is to change the phenotypic variance from V_i to $V_i + dV_i$, the disequilibrium contribution in the next generation, in the absence of linkage, is

$$d_{i+1} = \frac{1}{2}d_i + \frac{1}{2}h_i^4 dV_i, \quad (17)$$

since only half the disequilibrium contribution present in the previous generation is preserved. This recurrence relationship allows us to calculate the disequilibrium contribution in successive generations of selection. If dV_i is constant or approaches a limiting value dV^* , then d_i will rapidly tend to a limiting value, d^* , which can be evaluated by putting $d_{i+1} = d_i$ in equation (17); hence,

$$d^* = h^{*4} dV^*, \quad (18)$$

where h^{*2} is the limiting value of the heritability. In many circumstances, dV_i will be proportional to the phenotypic variance, V_i , so that we may write $dV_i = kV_i$. Substituting this value in equation (18) and solving it in terms of V_0 and h_0^2 , we find that

$$d^* = V_0 \{2kh_0^2 - 1 + [1 - 4kh_0^2(1 - h_0^2)]^{\frac{1}{2}}\} / 2(1 - k). \quad (19)$$

We shall now consider the significance of these results under artificial and natural selection.

ARTIFICIAL SELECTION

Consider a mass-selection experiment in which the individuals with the highest phenotypic values are chosen to be the parents of the next generation. If the phenotype is normally distributed in the parent generation with mean M and variance V and if a proportion P is selected then the mean in the selected group of parents is $M + IV^{\frac{1}{2}}$, where I , the intensity of selection, is equal to $f(x)/P$, x being the standard normal deviate corresponding to P and $f(x)$ the standard normal density function at x ; for example, if $P = 0.2$, then $x = 0.8416$, $f(x) = 0.2800$, and $I = 1.400$. The response to selection among their offspring is equal to $Ih^2V^{\frac{1}{2}}$. (See Falconer 1960 for further explanation.) It can be shown similarly that the variance in the selected group of parents is $V[1 - I(I - x)]$, so that the change in the variance as a result of selection is $-I(I - x)V$, which is equal to $-0.7818V$ when $P = 0.2$. Putting $k = -0.7818$ in equation (19) and taking $h_0^2 = \frac{1}{2}$,

we find that the disequilibrium contribution after several generations of selection of this intensity will reach a limiting value of $-0.125V_0$. Thus, if the phenotypic variance was 100 and the additive genetic variance 50 before selection began, then after several generations of selection the phenotypic variance will be reduced to 87.5 and the additive genetic variance to 37.5, so that the heritability is reduced from 0.5 to 0.428. The response to selection is reduced from 7.00 at the beginning of the experiment to 5.61, which is a reduction of 20%. The original values of the variance and heritability will, of course, be rapidly restored when selection ceases. The theoretical course of the selection experiment, calculated from the recurrence relationship contained in equation (17) is shown in table 1.

TABLE 1
THE EFFECT OF DISEQUILIBRIUM IN AN ARTIFICIAL SELECTION EXPERIMENT*

	GENERATION					
	0	1	2	3	4	∞
Disequilibrium contribution ...	0	-9.8	-11.9	-12.4	-12.5	-12.5
Phenotypic variance	100	90.2	88.1	87.6	87.5	87.5
Heritability	0.500	0.446	0.432	0.429	0.428	0.428
Response to selection	7.00	5.93	5.68	5.63	5.61

* Expected values for several parameters in a directional selection experiment computed for four generations. The top 20% of individuals are selected in each generation.

The limiting values are attained very quickly, nearly all the change occurring in the first two generations. If linkage were taken into account, an even larger effect would be expected.

NATURAL SELECTION

The intensity of selection is likely to be considerably less in nature than in artificial breeding experiments. As an example of stabilizing selection, Haldane (1953) has quoted the data of Rendel (1943) on the weights of 960 ducks' eggs of which 64.5% hatched. The mean weight of the original population was 73.9 gm and that of those which hatched 73.8 gm, so that there is no evidence of any directional selection on the mean weight. On the other hand, the variance of the weights was reduced from 52.7 to 43.9, which is highly significant and indicates that eggs of intermediate weight are at a selection advantage compared with either light or heavy eggs. The change in the variance as a result of selection is $dV = -8.8$; but before applying equation (18) we must allow for the fact that egg weight is maternally determined, so that selection acts only on one parent. In this case,

$$d_{i+1} = \frac{1}{2}d_i + \frac{1}{4}h_i^2dV_i, \quad (20)$$

so that in the limit

$$d^* = \frac{1}{2}h^{**}dV^*. \quad (21)$$

Assuming that $h^{**} = 0.6$ (this is the heritability of egg weight in poultry

quoted by Falconer 1960), we conclude that $d^* = -1.6$. If there were no selection, the phenotypic variance would increase from 52.7 to 54.3, and the heritability from 0.60 to 0.61, which is a negligible difference. The important point, however, is that once the phenotypic variance has been reduced from 54.3 to 52.7, due to a reduction in the effective additive genetic variance from 33.2 to 31.6, there will be no further reduction in the variance under continuous stabilizing selection at the same intensity; furthermore, if selection ceases, the small loss of variance will be quickly recovered. Attention must again be drawn to the proviso that this analysis is only strictly valid when the number of loci is effectively infinite and in the absence of linkage.

The effect of joint disequilibrium will probably be similar under directional selection and under stabilizing selection, since both can be regarded as cases of selection for an optimal value. The most plausible model of this type of selection, first considered by Haldane (1953), is that the fitness of an individual with phenotypic value Y is equal to $\exp - c(Y - T)^2$, where T is the optimal value and c determines the intensity of selection. If M and V are the phenotypic mean and variance, this seems equally suitable as a model of stabilizing selection (when $M = T$) or of directional selection (when $M \neq T$). Furthermore, if Y is normally distributed, it is quite easy to show that the change in the mean and variance in the parental generation as a result of selection are, respectively,

$$\begin{aligned} dM &= 2cV(T - M)/(1 + 2cV), \\ dV &= -2cV^2/(1 + 2cV). \end{aligned} \quad (22)$$

The response to selection is $h^2 dM$. Since the disequilibrium contribution is determined by dV , which is independent of M and T , it follows that it will be the same, for this model, under either stabilizing or directional selection.

Finally, mention must be made of disruptive selection, under which it is supposed that extreme phenotypic values (either large or small) are favored at the expense of intermediate values. This form of selection will lead to an increase in the phenotypic variability, so that the disequilibrium contribution will be positive rather than negative. Thus disruptive selection will cause a small increase in the additive genetic variance and, hence, in the heritability.

SUMMARY

If a metric character is determined by an effectively infinite number of loci, selection cannot cause any permanent change in the genetic variance but will cause a temporary change which is rapidly reversed when selection ceases. This change is due entirely to the correlation between pairs of loci which is induced by selection; the correlation is negative, leading to a reduction in the genetic variance under stabilizing or directional selection, and is positive, leading to an increase in the variance under disruptive selection. When selection ceases, the correlation rapidly disappears as joint

equilibrium at pairs of loci is reestablished, and the variance returns to its original value. An expression is derived for the predicted amount of change in the genetic variance due to disequilibrium in the absence of linkage. The change is likely to be small under selection intensities found under natural conditions, but it may be appreciable under intense artificial selection. This limiting result shows that the magnitude of any permanent change in the variance due to selection must decrease as the number of loci involved increases and that, when the number of loci is large, it is likely to be much less than the temporary change due to disequilibrium.

APPENDIX

THE REGRESSION BETWEEN RELATIVES UNDER THE INFINITESIMAL MODEL

Suppose that the genotypic value of an individual, G , is determined by a very large number of additive contributions from different loci,

$$G = \Sigma g_i,$$

where g_i is the contribution of the i^{th} locus; it is therefore assumed that there are no epistatic components of the genetic variance but that there may be a dominance component. If the population is in joint equilibrium at all loci as a result of random mating without selection over many generations, the contributions from different loci will be statistically independent of each other; this will be true even for linked loci, since linkage slows down the rate of approach to equilibrium but does not change the equilibrium ultimately attained. It follows from the central limit theorem that G will be approximately normally distributed.

We will now extend this well-known result to find the joint distribution of a pair of individuals who are related to each other in some specified way but who are otherwise chosen at random from the population. We denote the genotypic values of these individuals—who may be sibs, or parent and child, or grandparent and grandchild, or uncle and nephew—by G and G^* ; and, similarly, we write g_i and g_i^* for the contributions of the i^{th} locus to the respective genotype values. Because the individuals are related, g_i and g_i^* will not be independently distributed but will have some joint distribution. The bivariate form of the central limit theorem states that the joint distribution of G and G^* will be approximately bivariate normal when the number of loci is large, provided that for all pairs of loci the joint distribution of g_i and g_i^* is statistically independent of the joint distribution of g_j and g_j^* .

The above condition is obviously fulfilled for unlinked loci. It also holds for linked loci, provided that the relatives are parent and child or monozygotic twins, but it does not hold for linked loci for any other type of relative. The reason for this is that, at each locus, parent and child always have exactly one gene identical by descent and monozygotic twins always have two genes identical by descent. All other relatives have a variable number of genes identical by descent; for example, sibs may have 0, 1, or 2 identical genes, with probabilities $\frac{1}{4}$, $\frac{1}{2}$, and $\frac{1}{4}$, and grandparent and grandchild are equally likely to have 0 or 1 identical genes. Now, if the number of identical genes is variable, and if we know the number of identical genes at a particular locus, then this provides some information about the number of identical genes at another linked locus—the amount of information being greater, the closer the linkage. Furthermore, knowledge of the genotypic values, g_i and g_i^* , at a locus provides some information about the number of identical genes at that locus, and, hence, about the joint distribution of the genotypic values, g_j and g_j^* , at another linked locus. Hence, the joint distribution of g_j and g_j^* is only independent of the joint distribution of g_i and g_i^* if the loci are unlinked or if the related individuals are parent and child or monozygotic

twins. This conclusion agrees with the results of Schnell (1963), correcting those of Cockerham (1956), on the covariance between relatives in the presence both of linkage and of epistatic effects. Note that the lack of independence between the joint distributions at two linked loci does not have an effect on the covariance between relatives unless there are epistatic effects.

We can conclude that the genotypic values of a pair of individuals related as defined above under the infinitesimal model will follow a bivariate normal distribution in the absence of linkage. This result can be extended in the obvious way to show that the joint distribution of the genotypic values of any number of related individuals will be multivariate normal under the same conditions, and the result will also hold in the presence of linkage for father, mother, and child as well as for one parent and child. Furthermore, if this is true of the genotypic values, then it must also be true of the phenotypic values if the environmental deviations are normally distributed.

Let us now calculate the regression of offspring (Y) on both parents (P_1 and P_2), which can be written (whether or not there is linkage) as

$$Y = a + b_1P_1 + b_2P_2 + e. \quad (\text{A1})$$

From considerations of symmetry, $b_1 = b_2$, so that the regression can be rewritten as

$$Y = a + b(P_1 + P_2) + e. \quad (\text{A2})$$

If V is the phenotypic variance in both generations, then it is known that the covariance of an offspring and one parent is $\frac{1}{2}h^2V$, where h^2 is the heritability in the strict sense (e.g., Falconer 1960). Hence,

$$\begin{aligned} \text{Cov}(Y, P_1 + P_2) &= h^2V, \\ \text{Var}(P_1 + P_2) &= 2V, \\ b &= \frac{\text{Cov}(Y, P_1 + P_2)}{\text{Var}(P_1 + P_2)} = \frac{1}{2}h^2. \end{aligned} \quad (\text{A3})$$

The residual error variance can be evaluated by observing that

$$\text{Var}(Y) = b^2\text{Var}(P_1 + P_2) + \text{Var}(e), \quad (\text{A4})$$

whence

$$\text{Var}(e) = V - 2b^2V = (1 - \frac{1}{2}h^4)V. \quad (\text{A5})$$

Let us now consider the regression of a grandchild on all four of his grandparents, which can (in the absence of linkage) be written as

$$Y = a + b(G_1 + G_2 + G_3 + G_4) + e. \quad (\text{A6})$$

The covariance of a grandchild and one grandparent is known to be $\frac{1}{4}h^2V$. Proceeding in the same way as before, we find that

$$\begin{aligned} b &= \frac{1}{4}h^2, \\ \text{Var}(e) &= (1 - \frac{1}{4}h^4)V. \end{aligned} \quad (\text{A7})$$

Finally, let us consider the regression of an individual on one parent and on both grandparents on the same side, which (in the absence of linkage) can be written as

$$Y = a + bP + c(G_1 + G_2) + e. \quad (\text{A8})$$

The constants b and c can be found from the pair of simultaneous equations

$$\begin{aligned} b\text{Var}(P) + c\text{Cov}(P, G_1 + G_2) &= \text{Cov}(Y, P), \\ b\text{Cov}(P, G_1 + G_2) + c\text{Var}(G_1 + G_2) &= \text{Cov}(Y, G_1 + G_2), \end{aligned} \quad (\text{A9})$$

whose solution is found by substituting for the variances and covariances in terms of h^2 and V :

$$\begin{aligned} b &= \frac{1}{2}h^2(2 - h^2)/(2 - h^4), \\ c &= \frac{1}{2}h^2(1 - h^2)/(2 - h^4). \end{aligned} \quad (\text{A10})$$

The residual error variance can be found in the same way as before, but is not required in the text.

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