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The heritability of fitness: some single gene models

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Summary. Because directional selection exhausts additive-genetic variance, it is frequently claimed that the heritability of fitness should be very close to zero. However, mutation-selection balance generates a certain amount of additive-genetic variance, so that even parent-offspring measures of heritability may be greater than zero at equilibrium. Intra-generation heritability may also be non-zero, providing the potentials for genetic change following environmental change.

Key words: Heritability – Fitness – Additive-genetic variance

Introduction

The purpose of this note is to review the possibility that equilibrial additive-genetic variance in fitness need not be zero, whether estimated from parent-offspring correlation or regression or within generation correlation.

In 1930, Fisher enunciated what he called the fundamental thorem of natural selection: "The rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time." What Fisher called the genetic variance is now generally termed the additive-genetic variance. As Fisher noted, this theorem has the implication that "there will be no additive-genetic variance in fitness in random mating populations at equilibrium under natural selection, independent of the mode of selection, so that individuals of different genotype will all produce progeny with the same average fitness" (Charlesworth 1987). As has frequently been noted, the theorem is not in fact, as Fisher had initially hoped, a biological analogue of the second law of thermodynamics, in that it does not have that law's generality. However, it is usually accepted that it applies in a qualitative way.

As noted by Haldane (1949), if all possible matings in a population produce offspring with the same mean fitness, there will be no parent-offspring correlation (or regression) in fitness and, consequently, no change in fitness over time; this is, of course, the equilibrium to which Charlesworth referred. Thus, it has frequently been claimed that traits closely related to fitness should have low heritability as compared with traits not directly related to fitness (e.g., Falconer 1981). Of course, the question then should be asked, which traits fall into each category? For example, in conditions of crowding and seasonal uncertainty, the ability of grasses to produce multiple tillers or flowering stems, rather than just a main stem and culm, may be highly advantageous, yet not in more favorable environments. For one grass, common wheat, heritability of tillering ability has been estimated in a variety of studies to be between 0.03 and 0.80 (Merrit 1988). It is not clear what inference regarding the relation of tillering to fitness may be drawn.

The inference discussed above about "fitness traits" related essentially to parent-offspring heritability. Within a generation, as again noted by Haldane (1949), the situation is rather different; this will be considered further below. More recently, Eshel and Hamilton (1984) have noted that if there are cyclical variations in the environment such that fitnesses are not constant but also vary cyclically, parent-offspring correlations will not be zero and natural selection will occur at a rate given by the additive variance in fitness. In this paper, we wish to consider both parent-offspring correlations in fitness and sib-sib correlations in fitness, in order to access the gener-

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ality of the conclusion that equilibrial heritability of fitness should be close to zero and that, in any given generation, the opportunity for natural selection on traits related to fitness will be low, apart from stabilizing or "purifying" selection. We shall show that the more important question is whether the fitnesses, even if constant, are unassociated between family members from generation to generation.

Mousseau and Roff (1987) present substantial evidence for the existence in natural populations of high heritabilities for traits under strong selection, in contrast to the expectation of Charlesworth (1987).

We only consider random mating populations; assortative mating may complicate both estimation and interpretation of parent-offspring regression (Fisher 1918; Gimelfarb 1985).

Parent-offspring correlations

For a balanced polymorphism at equilibrium, as shown by Haldane (1949), the parent-offspring correlation in fitness is zero. However, as noted by Eshel and Hamilton (1984), away from equilibrium, this correlation will in general be non-zero, and in most cases will be positive, though it can be negative. The table below shows the frequencies of different parent-offspring combinations. Taking $0 \leq \alpha, \beta, \gamma \leq 1$ as arbitrary fitnesses, the subscripts t, $t+1$ denoting successive generations, we can write out the covariance as follows:

$$\mathrm{COV_{OP}} = [(\beta_t - \alpha_t)\, P\, q + (\gamma_t - \beta_t)\, R\, p]\, [(\beta_{t+1} - \alpha_{t+1})\, p + (\gamma_{t+1} - \beta_{t+1})\, q] .$$

Although this expression is much simpler than that of Eshel and Hamilton (1984), theirs can be obtained by some (tricky) calculations if one sets $\alpha = 1 + \alpha^1$, $\beta = 1 + \beta^1$, $\gamma = 1 - \alpha^1$. (Subscripts are omitted and α^1, β^1 denote their α, β.)

Then $\mathrm{COV_{OP}} > 0$ if both of the expressions in square brackets are greater than zero or if both are less than zero. From this expression for the covariance, we note that if $\gamma \geq \beta \geq \alpha$ in both parent and offspring generations or $\gamma \leq \beta \leq \alpha$ in both generations, $\mathrm{COV_{OP}} \geq 0$. We also note that if fitnesses do not vary between generations and there is random mating, then $\mathrm{COV_{OP}} = p\, q\, [(\beta - \alpha)\, p + (\gamma - \beta)\, q]^2 \geq 0$. [This is Eq. (1.7) of Ewens (1979).] Further, if the rank order of fitnesses is reversed between generations, $\mathrm{COV_{OP}} \leq 0$, as noted by Eshel and Hamilton (1984). However, this is a very strong condition to impose on the fitness values. (It is sufficient, but not necessary. If $\beta > \alpha$, γ or $\beta < \alpha$, γ, $\mathrm{COV_{OP}} < 0$ is possible for certain P, Q, R, p, q.)

We can also obtain the parent-offspring regression coefficient, b_{OP}, from $\mathrm{COV_{OP}}$ and $\mathrm{Var_P} = (\beta - \alpha)^2 P + (\gamma - \beta)^2 R - [(\beta - \alpha) P - (\gamma - \beta) R]^2$ for the case of constant fitnesses. The parent-offspring correlation may be similarly derived.

Many special cases of (mostly constant) fitness values have been considered in the literature. Table 1 shows several of these, using standard notation (μ = mutation rate per gene per generation, $0 \leq s, t \leq 1$ = diminutions in fitness, $0 \leq h \leq 1$ = scale factor for dominance). Our reasons for presenting so many examples, which may all be simply derived from the covariance expression given above and the marginal variance, are twofold. First, the magnitudes may be relatively very different. Secondly, we wished to collect for comparison the various cases presented in the literature for parent-offspring and sib-sib correlations (Table 4). The model of one-sided frequency dependence is very crude, but there is evidence that the phenomenon is real (Knoppien 1985), so we have used the simplest possible model for illustrative purposes (cf. Haldane and Jayakar 1967; Zonta and Jayakar 1988). In addition, it should be noted that a non-zero parent-offspring correlation is expected in the case of a sex-linked balanced polymorphism (Leach and Mayo 1967). Table 1 shows clearly that the use of the regression coefficient (which agrees with the O-P correlation in these cases) may lead to artificial results, since it is the quotient of two quantities both of which may be very small. In fact, if one calculates b_{OP} analytically as $b_{\mathrm{OP}} = \mathrm{COV_{op}}/\mathrm{Var_P}$ then the nominator and the denominator contain the same multiplicative term pq, which cancels, but is almost zero at mutation-selection equilibrium and is zero at the pure selection equilibrium. Therefore, in some cases of Table 1 we have $b_{\mathrm{OP}} \approx 1/2$, whereas $\mathrm{COV_{OP}}$ and hence the additive genetic variance is very small. The same problem seems to occur in the paper of Eshel and Hamilton (1984), who showed that under cyclical selection P-O correlation in fitness is most of the time one-half. However, $\mathrm{COV_{OP}}$ will be almost zero if the cycles are long – which is the most favourable case for high P-O correlation.

Overall, we can conclude that the absence of a parent-offspring correlation does not imply the absence of natural selection, as noted originally by Wright (1935). Indeed, so diverse are the possible outcomes of different types of single gene selection that it would be unwise to make any predictions. What one can say is that for plausible values of h, s, t, and μ (e.g., $h = 0.5$, $s = t = 0.01$, $\mu = 10^{-5}$), the parent-offspring correlations will be high for some cases in the absence of environmental contributions to variability in fitness. However, the contribution to the variance in fitness of a single gene is low (of the order of the mutation rate) and, hence, any single gene's contribution, in the presence of substantial environmental variability (e.g., a coefficient of variation of 10% for fitness with a mean close to unity), will be very small. Because there are many genes with variability maintained by mutation-selection balance, overall the heritability cannot be negligible.

Table 1. Parent-offspring correlations in fitness at equilibrium. (Where no reference is provided, the results have been derived using the formula on page 4)

Selection	Fitness			Mutation ($A_2 \rightarrow A_1$)	COV_{OP}	b_{OP}
	A_1A_1	A_1A_2	A_2A_2			
Dominant	$1-s$	$1-s$	1	μ	μs	$\frac{1}{2}\left(1-\frac{\mu}{2s}\right)$ ($\frac{1}{2}\sqrt{(1-s)}$; Haldane 1949)
Intermediate	$1-s$	$1-hs$	1	μ	μhs	$\frac{1}{2}\left(1-\frac{\mu}{2hs}\left(2-\frac{1}{h}\right)^2\right)$
Frequency dependent	$1-ps$	1	1	μ	μ/s	$s\left(1-\left(\frac{\mu}{s}\right)^{1/3}\right)^3 \Big/ (1-\mu)$
Recessive	$1-s$	1	1	μ	$\mu s\sqrt{\mu/s}$	$\sqrt{\frac{\mu}{s}}$
Overdominant	$1-s$	1	$1-t$	μ	$\mu^2 s/t$	$\frac{(s+t)^2\mu^2}{s^2t^4}$ (Haldane 1949; Penrose 1964)

The cases considered here are very simple, but many other fitness schemes that exhibit the important point mentioned earlier, i.e., lack of independence of parent and offspring fitnesses, could be considered. Tables 2 and 3 illustrate a fitness scheme possible only with long generation intervals and a long period of parental care as with humans and various other animals. In this system, proposed by Yokoyama (1987) as a model for the interaction of an individual's own phenotype with that of its parents, the fitness of an individual manifesting a trait is $1-\delta$ and this is reduced multiplicatively by a factor $(1-\varepsilon)$ for each affected parent. For generality, all three genotypes can manifest the trait with probabilities f_1, f_2, f_3. We have not worked out the entire consequences of this scheme because it is so artificial, but present it as an example of a case where parental phenotypes and genotypes and the mating types determine offspring fitnesses, noting that in this case, we cannot conclude that the offspring of all genotypes will have equal mean fitness, so that parent-offspring correlation will not be zero. We present the model in detail to illustrate the complexity of even the simplest, crudest model that is aimed at incorporating behavioural feedback.

Fraternal correlations in fitness

Using the method of Haldane (1949), we can readily derive comparable correlation coefficients for sibs. Some of these are set out in Table 4. It can be easily shown, in addition, that in each case only a part of the genetical variance is dominance variance; part is additive. (For example, in the dominant case, $V_A = p^3 q s^2$, $V_D = p^2 q^2 s^2$ in the population at large.)

The important point to note here is that at any time there is the potential for substantial additive-genetic variance in fitness within a generation from the joint effects of all of the different types of gene action, so that if the environment changes, whether in the cyclical manner proposed by Eshel and Hamilton (1984), or in a monotonic fashion, or in a discontinuous fashion, or indeed arbitrarily adaptive, evolution can occur, utilizing the variation maintained by either overdominance or mutation selection balance.

Discussion

If equilibrial additive-genetic variance is not zero, at any time, there will be substantial potential for natural selection, regardless of whether the population is in equilibrium or not. The only equilibrium that does not offer the potential for genetic change given environmental change is the trivial and unlikely state of complete homozygosity for all relevant genes.

There are many other possible influences on the additive-genetic variance in fitness and the consequent heritability. For example, Wade and McCauley (1980) have defined a "populational heritability", i.e., the proportion of the total variance in a trait attributable to genetic variation among different demes or subpopulations relative to the phenotypic variation among the means of such subpopulations. Tachida and Cockerham (1987) have considered the implications of such an approach for

Table 2. Fitness scheme of Yokoyama (1987). See text for details

	No. parents affected	Frequency	A_1A_1	A_1A_2	A_2A_2
$A_1A_1 \times A_1A_1$	0	$P^2(1-f_1)^2$	$f_1(1-\delta)$ $1-f_1$		
	1	$P^2\,2f(1-f_1)$	$f(1-\delta)(1-\varepsilon)$ $(1-f_1)(1-\varepsilon)$		
	2	$P^2 f_1^2$	$f_1(1-\delta)(1-\varepsilon)^2$ $(1-f_1)(1-\varepsilon)^2$		
$A_1A_1 \times A_1A_2$	0	$2PQ(1-f_1)(1-f_2)$	$\frac{1}{2}f_1(1-\delta)(1-\varepsilon)^2$ $\frac{1}{2}(1-f_1)(1-\varepsilon)^2$	$\frac{1}{2}f_2(1-\delta)$ $\frac{1}{2}(1-f_2)$	
	1	$2PQ((1-f_1)f_2+f_1(1-f_2))$	$\frac{1}{2}f_1(1-\delta)(1-\varepsilon)^2$ $\frac{1}{2}(1-f_1)(1-\varepsilon)^2$		
	2	$2PQ f_1 f_2$	$\frac{1}{2}f_1(1-\delta)(1-\varepsilon)^2$ $\frac{1}{2}(1-f_1)(1-\varepsilon)^2$		
$A_1A_1 \times A_2A_2$	0	$2PR(1-f_1)(1-f_3)$		$f_2(1-\delta)$ $1-f_2$	
	1	$2PR((1-f_1)f_3+f_1(1-f_3))$		$f_2(1-\delta)$ $1-f_2$	
	2	$2PR f_1 f_3$		$f_2(1-\delta)$ $1-f_2$	
$A_1A_2 \times A_1A_2$	0	$Q^2(1-f_2)^2$	$\frac{1}{4}f_1(1-\delta)(1-\varepsilon)^2$ $\frac{1}{4}(1-f_1)(1-\varepsilon)^2$	$\frac{1}{2}f_2(1-\delta)$ $\frac{1}{2}(1-f_2)$	$\frac{1}{4}f_3(1-\delta)$ $\frac{1}{4}(1-f_3)$
	1	$Q^2\,2f_2(1-f_2)$	$\frac{1}{4}f_1(1-\delta)(1-\varepsilon)^2$ $\frac{1}{4}(1-f_1)(1-\varepsilon)^2$		$\frac{1}{4}f_3(1-\delta)$ $\frac{1}{4}(1-f_3)$
	2	$Q^2 f_2^2$	$\frac{1}{4}f_1(1-\delta)(1-\varepsilon)^2$ $\frac{1}{4}(1-f_1)(1-\varepsilon)^2$		$\frac{1}{4}f_3(1-\delta)$ $\frac{1}{4}(1-f_3)$
$A_1A_2 \times A_2A_2$	0	$2QR(1-f_2)(1-f_3)$		$\frac{1}{2}f_2(1-\delta)$ $\frac{1}{2}(1-f_2)$	$\frac{1}{2}f_3(1-\delta)$ $\frac{1}{2}(1-f_3)$
	1	$2QR((1-f_2)f_3+f_2(1-f_3)$		$\frac{1}{2}f_2(1-\delta)$ $\frac{1}{2}(1-f_2)$	$\frac{1}{2}f_3(1-\delta)$ $\frac{1}{2}(1-f_3)$
	2	$2QR f_2 f_3$		$\frac{1}{2}f_2(1-\delta)$ $\frac{1}{2}(1-f_2)$	$\frac{1}{2}f_3(1-\delta)$ $\frac{1}{2}(1-f_3)$
$A_2A_2 \times A_2A_2$	0	$R^2(1-f_3)^2$			$\frac{1}{2}f_3(1-\delta)$ $\frac{1}{2}(1-f_3)$
	1	$R^2\,2f_2(1-f_3)$			$\frac{1}{2}f_3(1-\delta)$ $\frac{1}{2}(1-f_3)$
	2	$R^2 f_3^2$			$\frac{1}{2}f_3(1-\delta)$ $\frac{1}{2}(1-f_3)$

change in genetic variance over time and have concluded that, in most cases, the so-called populational heritability will increase over time to a maximum and decline thereafter, under panmixia with migration between demes and random extinction of demes. Examination of their results suggests that it will not be possible in any particular case to determine whether or not there should be substantial additive-genetic variance for a fitness-related trait at any particular time.

As a second example of a process that may be expected to allow the persistence of non-zero heritability for a trait closely associated with fitness, consider two traits whose functions are closely interdependent, but whose genetic determination is largely independent. In such a case, directional selection on one trait may be ineffective if the second trait is subject to stabilizing selection and has substantial additive-genetic variation (Bürger 1986). [Equations (11.4) and (19.6) of Falconer (1981) are not appropriate for this example because Bürger considered two traits under selection, one under directional, the other under stabilizing selection, whereas Falconer considered that only one trait is under selection, and obtained the correlated response of the second trait.]

In this discussion, we have not referred to actual estimates of heritability for fitness-related or other traits. However, we should note that estimates can vary extremely widely. For example, Woldehawariat et al. (1977) reported the results shown in Table 5 for birth weight (a trait known to display evidence of stabilizing selection) and final feedlot weight (a trait further removed from natural selection) in beef cattle. Neither trait is usually regarded as a "fitness trait", and each has quite high heritability, compared with, e.g., litter size in sheep (range 0.01–0.29; Purvis et al. 1987). Further, heritability can be

Table 3. Means and variances in fitness for the genotypic scheme of Table 2

		Mean	Variance
$A_1A_1 \times A_1A_1$	0	$1-f_1\delta$	$(1-f_1)f_1\delta^2$
	1	$(1-f_1\delta)(1-\varepsilon)$	$(1-\varepsilon)^2 f_1\delta^2(1-f_1)$
	2	$(1-f_1\delta)(1-\varepsilon)^2$	$(1-\varepsilon)^4 f_1\delta^2(1-f_1)$
$A_1A_1 \times A_1A_2$	0	$\frac{1}{2}(2-\delta(f_1+f_2))$	$\frac{1}{2}(1-\frac{1}{2}(f_1+f_2))(f_1+f_2)\delta^2$
	1	$\frac{1}{2}(2-\delta(f_1+f_2))(1-\varepsilon)$	$\frac{1}{2}(1-\frac{1}{2}(f_1+f_2))(f_1+f_2)\delta^2(1-\varepsilon)^2$
	2	$\frac{1}{2}(2-\delta(f_1+f_2))(1-\varepsilon)^2$	$\frac{1}{2}(1-\frac{1}{2}(f_1+f_2))(f_1+f_2)\delta^2(1-\varepsilon)^4$
$A_1A_1 \times A_2A_2$	0	$1-f_2\delta$	$(1-f_2)f_2\delta^2$
	1	$(1-f_2\delta)(1-\varepsilon)$	$(1-f_2)f_2\delta^2(1-\varepsilon)^2$
	2	$(1-f_2\delta)(1-\varepsilon)^2$	$(1-f_2)f_2\delta^2(1-\varepsilon)^4$
$A_1A_2 \times A_1A_2$	0	$1-\frac{1}{4}\delta(f_1+2f_2+f_3)$	$\frac{1}{4}(f_1+2f_2+f_3)(1-\frac{1}{4}(f_1+2f_2+f_3))\delta^2$
	1	$(1-\frac{1}{4}\delta(f_1+2f_2+f_3))(1-\varepsilon)$	$\frac{1}{4}(f_1+2f_2+f_3)(1-\frac{1}{4}(f_1+2f_2+f_3))\delta^2(1-\varepsilon)^2$
	2	$(1-\frac{1}{4}\delta(f_1+2f_2+f_3))(1-\varepsilon)^2$	$\frac{1}{4}(f_1+2f_2+f_3)(1-\frac{1}{4}(f_1+2f_2+f_3))\delta^2(1-\varepsilon)^4$
$A_1A_2 \times A_2A_2$	0	$\frac{1}{2}(2-\delta(f_2+f_3))$	$\frac{1}{2}(1-\frac{1}{2}(f_2+f_3))(f_2+f_3)\delta^2$
	1	$\frac{1}{2}(2-\delta(f_2+f_3))(1-\varepsilon)$	$\frac{1}{2}(1-\frac{1}{2}(f_2+f_3))(f_2+f_3)\delta^2(1-\varepsilon)^2$
	2	$\frac{1}{2}(2-\delta(f_2+f_3))(1-\varepsilon)^2$	$\frac{1}{2}(1-\frac{1}{2}(f_2+f_3))(f_2+f_3)\delta^2(1-\varepsilon)^4$
$A_2A_2 \times A_2A_2$	0	$1-f_3\delta$	$(1-f_3)f_3\delta^2$
	1	$(1-f_3\delta)(1-\varepsilon)$	$(1-f_3)f_3\delta^2(1-\varepsilon)^2$
	2	$(1-f_3\delta)(1-\varepsilon)^2$	$(1-f_3)f_3\delta^2(1-\varepsilon)^4$

Table 4. Sib-sib correlations in fitness at equilibrium. (Where no reference is provided, the results have been derived by methods set out in Leach and Mayo 1967)

Selection	Fitness			Mutation ($A_2 \to A_1$)	Correlation
	A_1A_1	A_1A_2	A_2A_2		
Dominant	$1-s$	$1-s$	1	μ	$\frac{1}{2}(1+s)$ (Leach and Mayo 1967)
Intermediate	$1-s$	$1-hs$	1	μ	$\frac{1}{2}(1+hs)$ (Haldane 1949)
Frequency dependent	$1-ps$	1	1	μ	$\frac{1}{4}\left(1+6\left(\frac{\mu}{s}\right)^{1/3}\right)$
Recessive	$1-s$	1	1	μ	$\frac{1}{4}$
Overdominant	$1-s$	1	$1-t$	0	$\left(\frac{s+t-2st}{s+t-st}\right)^2$ (Penrose 1964)
Two identical overdominant genes	$1-s$	1	$1-t$	0	$\left(\frac{s+t-2st}{s+t-st}\right)^2+\frac{\left(\frac{s+t-2st}{s+t-st}\right)^4-\left(\frac{s+t-2st}{s+t-st}\right)^2}{1+2\left(\frac{s+t-st}{(st)^2/(s+t)}\right)^2}$ (Mayo et al. 1982)

high because environmental variability is low (e.g., 0.9 for human serum acid phosphatase; Bishop et al. 1987) as much as because there is high overall variance with a substantial genetic component (final feedlot weight in Table 5).

For a general discussion of estimation problems, see Cheverud (1988), and for a discussion of variance in fertility, see Mayo et al. (1978). In this latter study, it was shown by simulation that long-tailed family (litter) size distributions could be completely determined genetically, yet daughter-dam regression would imply $V_A=0$, at the same time as directional selection was exhausting V_G and leading to an increase in mean litter size. In fact, negative daughter-dam regression for litter size is possible, largely through maternal effects, as shown by Falconer (1965) in mice and Rutledge (1980) in pigs.

Mousseau and Roff (1987), as already mentioned, have shown that in some cases heritabilities of fitness trait

Table 5. Heritability estimates for two traits in beef cattle (Woldehawariat et al. 1977)

Trait	No. estimates summarized	Estimation technique	Lowest estimate	Highest estimate	Mean
Birth weight	7	Regression of offspring on parent	0.21	0.57	0.42
	68	Paternal half-sib correlation	−0.29	1.47	0.45
Final feedlot weight	8	Regression of offspring on parent	0.25	0.84	0.44
	28	Paternal half-sib correlation	−0.44	1.00	0.47

are high in natural populations, and have furthermore done so in a way that largely rules out estimation difficulties. The models presented here provide an explanation for such observations.

Overall we can say, first, that many independent, concordant estimates of a heritability may be necessary before we can conclude that heritability is indeed low or high (as for many traits in economically important animals) and, secondly, that the estimates of heritability obtained for many traits (0–0.25) are compatible with the maintenance of variability by mutation-selection balance, i.e., most such traits, whether called "fitness traits" or not, are unlikely to be neutral in any meaningful sense. It should be noted that Bürger et al. (1989) and Keightley and Hill (1987) report high between-generation variances both under a balance between mutation and stabilizing selection and under directional selection; that is, different fitness patterns can yield substantial variability, making inferences from non-zero variance to fitness patterns hazardous. In this context, it is worth recalling the comment of Fisher (1948) on heritability: "Like so many statistical ratios, it has a numerator and a denominator, and its value depends on both elements; whereas, however, the numerator has a simple genetical meaning, and, if properly determined, should be an accurate estimate of the genetic variance, or the amount of variance of the relevant measurement directly available for utilization by selection, the denominator is the total variance exhibited by the variate as measured, and therefore includes the whole of the variance due to errors of measurement, in the strict sense, and, what in a wider sense also are errors of measurement, namely those due to uncontrolled, but potentially controllable environmental variation. It also, of course, contains the genetic variance, and some genotypic variance not immediately available for selective improvement, though liable to become so in future generations. Obviously, the information contained in the numerator is largely jettisoned when its actual value is forgotten, and it is only reported as a ratio to this hotch-potch of a denominator". Clearly, one should work with covariances and variances, but we have reviewed heritability in detail because most discussion has related to heritability.

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References

Bishop GR, Mayo O, Beckman L (1987) Estimation of genetical parameters for a quantitative trait subject to major gene influences. Hum Hered 37:182–185

Bürger R (1986) Constraints for the evolution of functionally coupled characters: a non-linear analysis of a phenotypic model. Evolution 40:182–193

Bürger R, Wagner G, Stettinger F (1989) How much heritable variation can be maintained in finite populations by mutation-selection balance? Evolution (in press)

Charlesworth B (1987) The heritability of fitness. In: Bradbury JW, Andersson MB (eds) Sexual selection-testing the alternatives. Dahlem workshop reports. Wiley, Chichester, vol. 39, pp 15–26

Cheverud JM (1988) A comparison of genetic and phenotypic correlations. Evolution 42:958–968

Eshel I, Hamilton WD (1984) Parent-offspring correlation in fitness under fluctuating selection. Proc R Soc London Ser B222:1–14

Ewens WJ (1979) Mathematical population genetics. Springer, Berlin Heidelberg New York (Biomathematics 9)

Falconer DS (1965) Maternal effects and selection response. In: Genetics today, Proc 11th Int Congr Genet. Pergamon Press, New York, pp 763–774

Falconer DS (1981) Introduction to quantitative genetics, 2nd edn. Longman, London

Fisher RA (1918) The correlation between relatives on the supposition of Mendelian inheritance. Trans R Soc Edinburgh 52:399–433

Fisher RA (1930) The genetical theory of natural selection. Clarendon Press, Oxford

Fisher RA (1948) Limits to intensive production in animals. Br Agric Bull 4:217–218

Gimelfarb A (1985) Is offspring-parent regression affected by assortative mating of parents? Genet Res 47:71–75

Haldane JBS (1949) Parental and fraternal correlations for fitness. Ann Eugen 14:288–292

Haldane JBS, Jayakar SD (1967) Polymorphism due to selection depending on the composition of a population. J Genet 58:318–323

Keightley PD, Hill WG (1987) Directional selection and variation in finite populations. Genetics 117:573–582

Knoppien P (1985) Rare male mating advantage: a review. Biol Rev 60:81–117

Leach CR, Mayo O (1967) On the effects of natural selection on correlations between relatives. Ann Hum Genet 30:379–385

Mayo O, Eckert SR, Waego HN (1982) Models of gene effects for a quantitative trait in man. In: Malhotra KC, Amitabha B

(eds) Proc Indian Stat Inst Golden Jubilee Int Conf, vol 1, pp 479–489

Mayo O, Bishop GR, Hancock TW (1978) The detection of genetical influences on human familty size. Hum Hered 28:270–279

Merritt RG (1988) Inheritance of culm number in two uniculm × multiculm wheat crosses. Euphytica 38:105–111

Mousseau TA, Roff DA (1987) Natural selection and the heritability of fitness components. Heredity 59:181–197

Penrose LS (1964) Some formal consequences of genes in stable equilibrium. Ann Hum Genet 28:159–166

Purvis IW, Atkins KD, Piper LR (1987) Genetic parameters for reproductive traits. In: McGuirk BJ (ed) Merino improvement programs in Australia. Australian Wool Corporation, Melbourne, pp 229–242

Rutledge JJ (1980) Fraternity size and swine reproduction. II. Genetical consequences. J Anim Sci 51:871–874

Tachida H, Cockerham CC (1987) Quantitative genetic variation in an ecological setting. Theor Popul Biol 32:393–429

Wade MJ, McCauley DE (1980) Group selection: the phenotypic and genotypic differentiation of small populations. Evolution 34:799–812

Woldehawariat G, Talamantes MA, Petty RR Jr, Cartwright TC (1977) A summary of genetic and environmental statistics for growth and conformation characters of young beef cattle, 2nd edn. Texas Agric Exp Stn Tech Rep No. 103. Texas A & M University, College Station

Wright S (1935) The analysis of variance and the correlations between relatives with respect to deviations from an optimum. J Genet 30:243–256

Yokoyama S (1987) Social selection in human populations: protected polymorphism of deleterious alleles with incomplete penetrance. Genet Epidemiol 4:223–231

Zonta LA, Jayakar SD (1988) Models of fluctuating selection for a quantitative trait. In: Jong G de (ed) Population genetics and evolution. Springer, Berlin Heidelberg New York, pp 102–108