

Effect of assortative mating on genetic change due to selection

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Received December 20, 1985; Accepted January 20, 1986

Communicated by D. Van Vleck

Summary. Two mathematical models (A and B) were used to study joint effects of selection and assortative mating on genetic change. Computer simulation was used to verify and extend the results. In each model, the genotype was additive with equal effects at each of N loci and the environmental distribution was $N(0, \sigma^2)$. In Model A, each locus had two alleles; in Model B, allelic effects at each locus followed a normal distribution. Using Model A, genetic change with assortative or random mating of selected parents was evaluated for combinations of number of loci ($N = 1, 2, 3$), heritability in base population ($H^{[0]} = 0.2, 0.5, 0.8$), allelic frequency in base population ($p = 0.1, 0.5$), and proportion selected ($\alpha = 0.20, 0.85$). Using Model B, genetic change with or without assortative mating was calculated for combinations of N (1, 2, 3, 5, 10, 100), $H^{[0]}$ (0.2, 0.5, 0.8) and α (0.20, 0.85). Response to selection under both mating systems in a finite population was estimated using Model A from 200 replications of a computer simulation; this was done for all combinations of N (1, 2, 3, 5, 10) and α (0.20, 0.85), with $H^{[0]} = 0.5$ and $p = 0.1$. Results obtained with both models indicate that the effect of assortative mating on genetic change increases with $H^{[0]}$ and α , and decreases with p . With Model A, the relationship between N and the effect of assortative mating on genetic change was not clear; with Model B, however, the advantage of assortative over random mating increased with N , as expected. Simulation results were in agreement with theory of Model A. This study indicates that selection with assortative mating can have a sizable (10 to 20%) long-term advantage over selection with random mating of parents when $H^{[0]}$ is high, p is low and α is large.

Key words: Assortative mating – Selection

Introduction

Mating individuals such that the phenotypes of mates are correlated is assortative mating. Theoretical considerations (e.g., Fisher 1918; Wright 1921; Crow and Felsenstein 1968; Bulmer 1980) and experimental results (Breese 1956; Wilson, Kyle and Bell 1965; Mwenya and Gianola 1984) indicate that positive assortative mating (hereafter referred to as assortative mating), in the absence of selection, can increase the additive genetic variance of a quantitative trait.

Several workers have examined, either theoretically or experimentally, assortative mating as a means of increasing response to selection for quantitative traits. Baker (1973) considered a trait determined by many loci with two alleles per locus. Allelic effects and frequencies were the same at all loci, gene action was additive, and genotypic and phenotypic values were taken as normally distributed. He calculated the additive genetic variance following a single generation of assortative or random mating of selected individuals. The extra additive variance due to assortative mating increased with heritability and gene frequency in the base population, and with the proportion of individuals selected as parents. Lande (1977) studied an additive character influenced by many genes with a wide range of possible allelic effects at each locus. Assuming that the regression of allelic effects on phenotype was linear, and a constant mutation rate, it was shown that the equilibrium genetic variance was independent of the mating system and of the linkage map.

In experiments with *Drosophila* (McBride and Robertson 1963) and *Tribolium* (Wilson et al. 1965; Mwenya et al. 1983) consistently larger responses to selection were obtained with assortative than with random mating during selection. Where tested, dif-

ferences were not statistically significant, suggesting that although assortative mating may increase response to selection, its effect is probably small. Similar results have been obtained using computer simulation (Rico et al. 1973; De Lange 1974; Fernando et al. 1983). The results of De Lange (1974) and of Fernando et al. (1983) were in agreement with the theoretical developments of Baker (1973), viz, a higher proportion selected increased the advantage of assortative mating over random mating of parents. However, the opposite was observed in the *Tribolium* experiment of Mwenya et al. (1983).

The objective of this study was to examine the joint effects of gene frequency and heritability in the base population, number of loci and proportion selected, on long term response to truncation selection with assortative mating of parents.

Model A: two alleles per locus

A trait influenced by a pair of additive genes at each of N independently segregating loci was considered. At each locus, allelic effects were either 0 or 1, with frequencies in the base population of $(1 - p)$ and p , respectively. The genotypic value was the sum of allelic effects, and the phenotypic value was the sum of the genotypic value and of an independent environmental effect distributed normally, with mean 0 and variance σ^2 . A population of infinite size was assumed.

Genotypic frequencies at Generation 0 were calculated assuming the population was in linkage equilibrium. For example, with 2 loci, the frequency of the double heterozygote was $[2p(1 - p)]^2$.

The truncation point (t_0) corresponding to a proportion selected was calculated as follows. Let g_i be the frequency and μ_i be the value of the i^{th} genotype ($i = 1, \dots, 3^N$). Given that an individual has the i^{th} genotype, the probability that its phenotypic value is larger than t_0 is

$$1 - \Phi[(t_0 - \mu_i)/\sigma] = \Phi[(\mu_i - t_0)/\sigma]. \quad (1)$$

where $\Phi[\cdot]$ is the standard normal distribution function. The probability that an individual has the i^{th} genotype and a phenotypic value larger than t_0 is

$$g_i \cdot \Phi[(\mu_i - t_0)/\sigma]. \quad (2)$$

Thus, if a proportion α is to be selected by truncation on phenotypic values, t_0 must satisfy

$$\sum_i g_i \cdot \Phi[(\mu_i - t_0)/\sigma] - \alpha = 0. \quad (3)$$

This nonlinear equation was solved for t_0 using the bisection method (Forsythe et al. 1977).

The frequency of the i^{th} genotype after selection is then

$$g_i^{[n+1]} = g_i^{[n]} \Phi[(\mu_i - t_0^{[n]})/\sigma]/\alpha, \quad (4)$$

where the superscript n is generation number; superscripts are suppressed when the generation number is obvious from the context.

Assortative mating was introduced by mating individuals selected as parents at random within three arbitrary intervals of phenotypic values: (t_0, t_1) , (t_1, t_2) and (t_2, t_3) , where

t_0 = truncation points for selection,

$t_k = t_0 + (t_3 - t_0)k/3$, $k = 1, 2$; and

$t_3 = 2N + 3\sigma$.

Because individuals within an interval are more similar than those in different intervals, a positive correlation between the phenotypic values of mates is induced, i.e., assortative mating. Note that because the largest genetic value is $2N$ and the environmental effects are $N(0, \sigma^2)$, phenotypic values of selected individuals are less than t_3 with probability close to 1.

Let

Q_{ik} = probability that an individual's phenotypic value is between t_{k-1} and t_k ($k = 1, 2, 3$), given that it has genotype i ;

P_{ik} = probability that an individual has genotype i and that its phenotypic value is between t_{k-1} and t_k ;

R_{ijk} = probability that an individual of genotype i mates with an individual of genotype j , given that each has its phenotypic value in interval k (i.e., between t_{k-1} and t_k).

The marginal probability that the phenotypic value of an individual lies in interval k is then $P_{.k} = \sum_i P_{ik}$.

Because of the mating system, the probability that the two mates are in interval k is also $P_{.k}$; this is so because once an individual is in the interval, the mate is also in the interval with probability 1. The mating frequency between genotypes i and j is then

$$R_{ij} = \sum_{k=1}^3 R_{ijk} \cdot P_{.k}. \quad (5)$$

Further, because mating is at random within interval k ,

$$R_{ijk} = \frac{P_{ik}}{P_{.k}} \cdot \frac{P_{jk}}{P_{.k}}. \quad (6)$$

Also, from standard probability theory, $P_{ik} = g_i \cdot Q_{ik}$, and

$$Q_{ik} = \frac{\Phi[(t_k - \mu_i)/\sigma] - \Phi[(t_{k-1} - \mu_i)/\sigma]}{\sum_{k=1}^3 \{\Phi[(t_k - \mu_i)/\sigma] - \Phi[(t_{k-1} - \mu_i)/\sigma]\}}. \quad (7)$$

The genotypic frequencies in the next generation can be obtained following laws of Mendelian inheritance.

At each generation, ($n = 0, 1, \dots$), the genetic mean ($\mu^{[n]}$), the theoretical ($H_T^{[n]}$) and the realized ($H_R^{[n]}$)

heritabilities, and the correlation between phenotypic values of selected mates ($\rho^{[n]}$) were calculated. The formulae used were:

$$\mu^{[n]} = \sum_i g_i^{[n]} \mu_i, \quad (8)$$

$$H_I^{[n]} = V_A^{[n]} / V_P^{[n]}, \quad (9)$$

where

$$V_A^{[n]} = \sum_i g_i^{[n]} \mu_i^2 - (\mu^{[n]})^2 \quad (10)$$

is the additive genetic variance and

$$V_P^{[n]} = V_A^{[n]} + \sigma^2 \quad (11)$$

is the phenotypic variance. Further, because the expected phenotypic value at generation n is also $\mu^{[n]}$, realized heritability was calculated as

$$H_R^{[n]} = (\mu^{[n+1]} - \mu^{[n]}) / (\mu_S^{[n]} - \mu^{[n]}), \quad (12)$$

where $\mu_S^{[n]}$ is the phenotypic mean of the selected parents. The calculation of $\mu_S^{[n]}$ and of $\rho^{[n]}$ are given in an Appendix. Calculations were made for all combinations of N (1, 2, 3), $H^{[0]}$ (0.2, 0.5, 0.8), p (0.1, 0.5), and α (0.20, 0.85), for assortative and random mating.

Model B: infinite number of alleles per locus

This model is essentially that of Lande (1977), except that the effects of mutation and linkage were ignored, and attention was restricted to truncation selection. Lande (1977) considered a multifactorial trait under completely additive genetic action; multiple alleles with a wide range of effects were considered at each locus. Lande (1977) further assumed that the regression of allelic effects at each locus on phenotypic values was linear.

Let Z^M and Z^F be the phenotypic values of the male and female parents of an individual in generation n , and let $X_i^M, X_j^M, X_i^F, X_j^F$ be the allelic effects at loci i and j ($i = 1, \dots, N$) in gametes produced by these parents. Also, let

$$C_{ij}^{[n]} = \text{Cov}(X_i^M, X_j^M) = \text{Cov}(X_i^F, X_j^F),$$

$$C'_{ij}^{[n]} = \text{Cov}(X_i^M, X_j^F) = \text{Cov}(X_i^F, X_j^M).$$

and

$$C_{iZ}^{[n]} = \text{Cov}(X_i^0, Z^0)$$

where Z^0 and X_i^0 are, respectively, the phenotypic value and an allelic effect at locus i in an offspring born in generation n . Following Lande (1977), and letting $\varepsilon \sim N(0, \sigma^2)$ be an environmental deviate distributed independently of the allelic effects,

$$\begin{aligned} C_{iZ}^{[n]} &= \text{Cov}\left[X_i^0, \sum_{i=1}^N (X_i^M + X_i^F) + \varepsilon\right] \\ &= C_{ii}^{[n]} + C'_{ii}^{[n]} + (N-1)(C_{ij}^{[n]} + C'_{ij}^{[n]}) \end{aligned} \quad (13)$$

because X_i^0 is either X_i^M or X_i^F . The genotypic and phenotypic variances are

$$V_A^{[n]} = 2N C_{iZ}^{[n]} \quad (14)$$

and

$$V_P^{[n]} = V_A^{[n]} + \sigma^2 \quad (15)$$

respectively. Lande (1977) showed that with selection and assortative mating,

$$C'_{ij}^{[n]} = (1 - k^{[n-1]}) \rho C_{iZ}^{[n-1]} C_{jZ}^{[n-1]} / V_P^{[n-1]} \quad (16)$$

where ρ is the correlation between phenotypic values of mates,

$$k^{[n-1]} = 1 - V_{PS}^{[n-1]} / V_P^{[n-1]} \quad (17)$$

and $V_{PS}^{[n-1]}$ is the phenotypic variance among selected parents. Also,

$$C_{ii}^{[n]} = C_{ii}^{[n-1]} - k^{[n-1]} C_{iZ}^{[n-1]} C_{iZ}^{[n-1]} / V_P^{[n-1]} \quad (18a)$$

and

$$\begin{aligned} C_{ij}^{[n]} &= C_{ij}^{[n-1]} / 2 + C'_{ij}^{[n-1]} / 2 \\ &\quad - k^{[n-1]} C_{iZ}^{[n-1]} C_{jZ}^{[n-1]} / V_P^{[n-1]}; \quad i \neq j \end{aligned} \quad (18b)$$

Because Lande (1977) assumed that the regression of allelic effects on phenotypic values was linear, it is possible to derive his results directly from normal distribution theory. Let \mathbf{u}_1 and \mathbf{u}_2 be jointly normally distributed vectors with

$$\text{Var}(\mathbf{u}_1) = \Sigma_1, \quad \text{Var}(\mathbf{u}_2) = \Sigma_2$$

and

$$\text{Cov}(\mathbf{u}_1, \mathbf{u}_2) = \Sigma_{12}.$$

Pearson (1903) showed for the multivariate normal case that if by some process such as selection, the covariance matrix of \mathbf{u}_2 is altered from Σ_2 to Σ_2^* , the covariance matrix of \mathbf{u}_1 becomes

$$\Sigma_1^* = \Sigma_1 - \Sigma_{12} \Sigma_2^{-1} (\Sigma_2 - \Sigma_2^*) \Sigma_2^{-1} \Sigma_{21}. \quad (19)$$

We use this result to derive the covariance structure of allelic effects when the variance-covariance matrix of Z^M and Z^F is altered by selection and assortative mating.

With random mating

$$\begin{aligned} \text{Cov}(X_i^M, X_j^F) &= \text{Cov}(X_i^M, Z^F) = \text{Cov}(X_i^F, Z^M) \\ &= \text{Cov}(Z^M, Z^F) = 0. \end{aligned}$$

Further, in the absence of selection $C_{ii}^{[n]} = C_{ii}^{[n-1]}$. Hence,

$$\text{Var} \begin{bmatrix} X_i^M \\ X_j^F \end{bmatrix} = \begin{bmatrix} C_{ii}^{[n-1]} & 0 \\ 0 & C_{jj}^{[n-1]} \end{bmatrix}, \quad (20a)$$

$$\text{Var} \begin{bmatrix} Z^M \\ Z^F \end{bmatrix} = \begin{bmatrix} V_P^{[n-1]} & 0 \\ 0 & V_P^{[n-1]} \end{bmatrix}, \quad (20b)$$

and

$$\text{Cov} \left(\begin{bmatrix} X_i^M \\ X_j^F \end{bmatrix}, [Z^M, Z^F] \right) = \begin{bmatrix} C_{iZ}^{[n-1]} & 0 \\ 0 & C_{jZ}^{[n-1]} \end{bmatrix}. \quad (20c)$$

With selection operating on phenotypic values and with assortative mating of selected parents, write

$$\text{Var} \begin{bmatrix} Z^M \\ Z^F \end{bmatrix} = \begin{bmatrix} V_{PS}^{[n-1]} & V_{PS}^{[n-1]} \varrho \\ V_{PS}^{[n-1]} \varrho & V_{PS}^{[n-1]} \end{bmatrix}. \quad (21)$$

Following Pearson (1903), the covariance matrix of X_i^M and X_j^F after selection and assortative mating can be obtained by using [20a] to [20c] and [21] in [19] so that

$$\begin{aligned} \text{Var} \begin{bmatrix} X_i^M \\ X_j^F \end{bmatrix} | \text{selection and assortative mating} \\ = \begin{bmatrix} C_{ii}^{[n-1]} & 0 \\ 0 & C_{jj}^{[n-1]} \end{bmatrix} - \begin{bmatrix} C_{iZ}^{[n-1]}/V_P^{[n-1]} & 0 \\ 0 & C_{jZ}^{[n-1]}/V_P^{[n-1]} \end{bmatrix} \\ \begin{bmatrix} V_P^{[n-1]} - V_{PS}^{[n-1]} & -V_{PS}^{[n-1]} \varrho \\ -V_{PS}^{[n-1]} \varrho & V_P^{[n-1]} - V_{PS}^{[n-1]} \end{bmatrix} \\ \begin{bmatrix} C_{iZ}^{[n-1]}/V_P^{[n-1]} & 0 \\ 0 & C_{jZ}^{[n-1]}/V_P^{[n-1]} \end{bmatrix} \end{aligned} \quad (22)$$

From [22], the covariance between X_i^M and X_j^F after selection and assortative mating is

$$\begin{aligned} C_{ij}^{[n]} &= \frac{C_{iZ}^{[n-1]} C_{jZ}^{[n-1]} V_{PS}^{[n-1]}}{V_P^{[n-1]} V_P^{[n-1]}} \varrho \\ &= (1 - k^{[n-1]}) \varrho C_{iZ}^{[n-1]} C_{jZ}^{[n-1]} / V_P^{[n-1]}. \end{aligned} \quad (23)$$

Similarly, without selection and regardless of the mating system (Crow and Kimura 1970, p 151), the covariance between allelic effects at different loci in the same gamete is

$$\text{Var} \begin{bmatrix} X_i^l \\ X_j^l \end{bmatrix} = \begin{bmatrix} C_{ii}^{[n-1]} & (C_{ij}^{[n-1]} + C_{ij}'^{[n-1]})/2 \\ (C_{ij}^{[n-1]} + C_{ij}'^{[n-1]})/2 & C_{jj}^{[n-1]} \end{bmatrix} \quad (24)$$

$$\text{Cov} \left(\begin{bmatrix} X_i^l \\ X_j^l \end{bmatrix}, Z^l \right) = \begin{bmatrix} C_{iZ}^{[n-1]} \\ C_{jZ}^{[n-1]} \end{bmatrix} \quad (25)$$

for $l = M, F$. Further, in the absence of selection

$$\text{Var}(Z^l) = V_P^{[n-1]}, \quad (26)$$

but with selection

$$\text{Var}(Z^l) = V_{PS}^{[n-1]}. \quad (27)$$

Thus, by using (24)–(27) in (19), the covariance matrix of X_i^l and X_j^l after selection is

$$\begin{aligned} \text{Var} \begin{bmatrix} X_i \\ X_j \end{bmatrix} | \text{selection} \\ = \begin{bmatrix} C_{ii}^{[n-1]} & (C_{ij}^{[n-1]} + C_{ij}'^{[n-1]})/2 \\ (C_{ij}^{[n-1]} + C_{ij}'^{[n-1]})/2 & C_{jj}^{[n-1]} \end{bmatrix} \end{aligned}$$

$$\begin{aligned} - \frac{1}{V_P^{[n-1]}} \begin{bmatrix} C_{iZ}^{[n-1]} \\ C_{jZ}^{[n-1]} \end{bmatrix} \\ (V_P^{[n-1]} - V_{PS}^{[n-1]}) [C_{iZ}^{[n-1]} C_{jZ}^{[n-1]}] \frac{1}{V_P^{[n-1]}}. \end{aligned} \quad (28)$$

The elements of (28) are explicitly (29a)

$$\text{Var}(X_i^l) = C_{ii}^{[n]} = C_{ii}^{[n-1]} - C_{iZ}^{[n-1]} C_{iZ}^{[n-1]} k^{[n-1]} / V_P^{[n-1]},$$

with $\text{Var}(X_j^l) = C_{jj}^{[n]}$ having a similar form, and

$$\begin{aligned} \text{Cov}(X_i^l, X_j^l) &= C_{ij}^{[n]} = C_{ij}^{[n-1]}/2 \\ &+ C_{ij}'^{[n-1]}/2 - C_{iZ}^{[n-1]} C_{jZ}^{[n-1]} k^{[n-1]} / V_P^{[n-1]} \end{aligned} \quad (29b)$$

When a proportion α of individuals is selected from a normal distribution by truncation on phenotypic values,

$$k = - \frac{\varphi(X)}{\alpha} [X - \varphi(X)/\alpha] \quad (30)$$

where $\varphi(\cdot)$ is the standard normal density function and

$$X = \Phi^{-1}(\alpha). \quad (31)$$

Equations (14) to (18b) were used recursively to calculate the theoretical heritability and the expected response to selection under random or assortative mating. Theoretical heritability was calculated as

$$H_T^{[n]} = V_A^{[n]} / V_P^{[n]}, \quad (32)$$

with $V_A^{[n]}$ and $V_P^{[n]}$ as in (14) and (15). Expected response was

$$\sum_{i=2}^n r_i \quad (33)$$

where

$$r_i = H_T^{[i-1]} (V_P^{[i-1]})^{1/2} \cdot \varphi(X)/\alpha. \quad (34)$$

These calculations were made for combinations of number of loci ($N = 1, 2, 3, 5, 10, 100$), heritability in the base population ($H^{[0]} = 0.2, 0.5, 0.8$) and proportion selected ($\alpha = 0.20, 0.85$), under random or assortative mating with $\varrho = 0.8$.

Computer simulation

A Monte Carlo experiment examined the robustness of the theory developed for Model A when applied to a finite population. Breeding populations of 40 males and 40 females with N independently segregating loci were simulated in a computer using a program written in the Pascal language. Allelic effects at each locus were 0 or 1, and gene action was completely additive. Mating of parents was at random or assortative. Re-

sponse to mass selection, with and without assortative mating, was estimated from 200 replicates of the simulation for combinations of N (1, 2, 3, 5, 10) and α (0.20, 0.85), with $H_T^{[0]}=0.5$ and an initial gene frequency of 0.10.

Table 1. Largest difference between the genetic means of the assortative and random mating lines (Model A) as a percentage of the selection limit, and the generation at which it occurred (in parentheses)^a

No. of loci	Heritability in base population	Proportion selected			
		0.20		0.85	
		$p^b = 0.1$	$p = 0.5$	$p = 0.1$	$p = 0.5$
1	0.2	1.50 (3)	0	5.50 (16)	1.50 (8)
1	0.5	2.00 (2)	0.50 (2)	8.00 (14)	3.00 (4)
1	0.8	0	0	10.50 (14)	6.00 (5)
2	0.2	1.50 (4)	0.25 (2)	6.75 (20)	1.50 (11)
2	0.5	2.50 (3)	0.25 (2)	11.00 (17)	3.52 (8)
2	0.8	4.00 (3)	0.25 (2)	14.00 (16)	5.75 (7)
3	0.2	1.50 (5)	0.17 (2)	6.50 (24)	1.50 (14)
3	0.5	2.33 (4)	0.50 (2)	10.67 (19)	3.33 (9)
3	0.8	3.33 (4)	1.00 (2)	13.17 (19)	5.67 (9)

^a Entries are $\Delta = \{[\mu(\text{Assortative}) - \mu(\text{Random})]/2N\} 100$, see text

^b p = gene frequency in base population

Results

Model A: two alleles per locus

The largest differences (Δ) between the genetic means of the assortative and random mating lines, relative to the selection limit ($2N$), and the generations at which they were realized (\bar{t}) are given in Table 1. The value of Δ ranged from 0% (e.g., $N = 1$, $H_T^{[0]} = 0.8$, $\alpha = 0.20$, $p = 0.1$) to 14% at $\bar{t} = 16$ for $N = 2$, $H_T^{[0]} = 0.8$, $\alpha = 0.85$, $p = 0.1$.

In general, Δ increased as $H_T^{[0]}$ and α increased and as p decreased. However, there were exceptions to this pattern. For example, with $N = 1$, $\alpha = 0.20$ and $p = 0.1$ or 0.5, increasing $H_T^{[0]}$ from 0.5 to 0.8 decreased Δ to 0%; this was because with $H_T^{[0]} = 0.8$, the selection limit was reached in generation 2, the first generation at which differences between the assortative and random mating lines can be observed. Similarly, increasing $H_T^{[0]}$ had no effect on Δ for $N = 2$, $\alpha = 0.20$ and $p = 0.5$.

As α increased, so did Δ , without exception. When gene frequency increased from 0.1 to 0.5, Δ decreased; however, with $N = 1$, $H_T^{[0]} = 0.8$ and $\alpha = 0.20$, increasing p did not affect Δ .

The relationship between N and Δ was not clear. For example, N had no effect on Δ when $H_T^{[0]} = 0.2$, $\alpha = 0.20$ and $p = 0.1$, or when $H_T^{[0]} = 0.2$, $\alpha = 0.85$ and $p = 0.5$. In these two instances, however, \bar{t} increased as N increased. With $H_T^{[0]} = 0.5$, $\alpha = 0.20$ and $p = 0.5$, Δ was

Table 2. Evolution of parameters with selection under assortative or random mating of parents (Model A; $N = 3$, $H_T^{[0]} = 0.5$, $p = 0.1$, $\alpha = 0.85$)^a

Generation	Assortative mating				Random mating ^b			
	t_0	H_T	H_R	μ	t_0	H_T	H_R	μ
0	-0.46	0.500	0.363	0.60	-0.46	0.500	0.363	0.60
1	-0.41	0.548	0.404	0.70	-0.40	0.524	0.403	0.70
2	-0.35	0.590	0.446	0.81	-0.32	0.550	0.443	0.81
3	-0.28	0.627	0.489	0.93	-0.24	0.575	0.484	0.93
4	-0.19	0.661	0.532	1.08	-0.13	0.598	0.523	1.07
5	-0.09	0.690	0.573	1.24	-0.01	0.619	0.559	1.23
6	0.04	0.715	0.612	1.43	0.13	0.638	0.591	1.40
7	0.19	0.736	0.646	1.64	0.29	0.654	0.616	1.59
8	0.37	0.752	0.675	1.87	0.47	0.668	0.638	1.80
9	0.56	0.766	0.699	2.12	0.66	0.680	0.656	2.01
10	0.79	0.775	0.720	2.39	0.87	0.689	0.671	2.24
11	1.04	0.781	0.737	2.67	1.09	0.695	0.683	2.48
12	1.32	0.774	0.751	2.97	1.33	0.699	0.691	2.72
13	1.62	0.761	0.760	3.29	1.57	0.700	0.697	2.98
14	1.96	0.741	0.766	3.61	1.83	0.699	0.700	3.23
15	2.31	0.711	0.768	3.93	2.10	0.695	0.701	3.49
16	2.69	0.669	0.763	4.25	2.37	0.687	0.699	3.75
17	3.09	0.612	0.751	4.56	2.65	0.676	0.693	4.00
18	3.49	0.536	0.729	4.86	2.93	0.661	0.684	4.25
19	3.87	0.439	0.694	5.13	3.21	0.640	0.671	4.49
20	4.23	0.329	0.640	5.37	3.48	0.614	0.653	4.73

^a See text for definition of symbols

^b t_0 : truncation point; H_T : theoretical heritability; H_R : realized heritability; μ : genetic mean

Table 3. Evolution of genetic parameters with selection under assortative or random mating (Model A; $N = 3$, $H_T^{[0]} = 0.5$, $p = 0.1$, $\alpha = 0.20$)^a

Generation	Assortative mating ^b				Random mating ^b			
	t_0	H_T	H_R	μ	t_0	H_T	H_R	μ
0	1.45	0.500	0.574	0.60	1.45	0.500	0.574	0.60
1	2.49	0.629	0.655	1.48	2.49	0.623	0.646	1.48
2	3.69	0.673	0.684	2.61	3.65	0.661	0.668	2.59
3	4.94	0.664	0.663	3.86	4.83	0.650	0.645	3.77
4	5.99	0.562	0.517	5.03	5.85	0.566	0.538	4.89
5	6.52	0.254	0.159	5.80	6.46	0.321	0.222	5.71
6	6.61	0.030	0.015	5.98	6.60	0.050	0.026	5.97
7	6.62	0.002	0.001	6.00	6.62	0.004	0.002	6.00

^{a, b} See Table 2**Table 4.** Largest relative difference (LRD, %) between means of the assortative and random mating lines, and the generation at which it was observed (Model B)

No. of loci	Heritability in population	Proportion selected			
		0.20		0.85	
		LRD	Generation	LRD	Generation
1	0.2	1.72	2	4.78	2
1	0.5	4.18	2	10.44	2
1	0.8	6.29	2	14.30	3
2	0.2	1.78	3	5.91	6
2	0.5	4.18	2	12.45	5
2	0.8	6.29	2	16.59	5
3	0.2	1.89	4	6.58	8
3	0.5	4.18	2	13.65	6
3	0.8	6.29	2	18.05	6
5	0.2	2.01	5	7.24	11
5	0.5	4.18	2	14.95	8
5	0.8	6.29	2	19.60	8
10	0.2	2.13	7	7.88	16
10	0.5	4.25	3	16.27	11
10	0.8	6.29	2	21.22	11
100	0.2	2.30 ^a	20 ^a	8.68 ^a	20 ^a
100	0.5	4.32	3	18.33 ^a	20 ^a
100	0.8	6.29	2	23.89 ^a	20 ^a

^a Relative difference was increasing after 20 generations. LRD is the largest value of $[(\mu_A - \mu_R)/\mu_R]$. 100 in each combination of number of loci, heritability and population selected

lowest (0.25%) when $N = 2$ and had the same value when $N = 1$ or $N = 3$ (0.50%). Finally, Δ was an increasing function of N with $H_T^{[0]} = 0.8$, $\alpha = 0.20$, $p = 0.5$, but a decreasing one when $H_T^{[0]} = 0.8$, $\alpha = 0.85$ and $p = 0.5$.

Where defined (i.e., when $\Delta \neq 0$), \bar{t} ranged from 2 to 24 generations. In general, \bar{t} increased as N and α increased and as $H_T^{[0]}$ and p decreased.

Genetic parameters in the assortative (A) and random (R) mating lines, by generation, are given in Table 2 for $N = 3$, $H_T^{[0]} = 0.5$, $p = 0.1$ and $\alpha = 0.85$. In each line, H_T and H_R increased as selection proceeded, and then started to decline between generations 11 and 15; H_T peaked before H_R in the A and R lines. In the R line, H_T and H_R at Generation 20 were still larger than in the base population. This illustrates that, at least in the short-term, selection may actually increase the ratio of additive genetic to phenotypic variance. Until Generation 16, H_T was larger in the A than in the R line but, thereafter, this parameter decreased rapidly in the A line. Realized heritability was larger in the A line until Generation 19; however, H_R did not subsequently decrease as rapidly as it was observed in the case of H_T . Large differences were observed between H_T and H_R ; for example, $H_R^{[0]}$ was smaller than $H_T^{[0]}$ in the A and R lines.

As shown in Table 2, the difference between the genetic means of the A and R lines was small in the early generations (0.17% of the selection limit by generation 5). However, this relative difference increased thereafter and peaked at generation 19 ($\Delta = 10.67\%$).

Values of H_T , H_R and μ in the A and R lines for $N = 3$, $H_T^{[0]} = 0.5$, $p = 0.1$ and $\alpha = 0.20$ are given in Table 3; note that aside from the fact that selection was more intense, this corresponds to the combination of parameter values used to generate results of Table 2. In the case of Table 3, H_T and H_R peaked at Generation 2 and decreased thereafter irrespective of the line; heritability was negligible by Generation 6. At the onset, $H_R^{[0]}$ was 14.8% larger than $H_T^{[0]}$; this difference decreased as selection continued and, from Generation 3, H_T became larger than H_R . There was a slight advantage of assortative over random mating as indicated by the values of the genetic means. This difference decreased, however, as the two populations approached the selection limit, which was attained at Generation 7.

Model B: infinite number of alleles per locus

The largest difference between genetic means of the A and R lines was expressed as a percentage of the genetic mean in the R line. The largest relative difference (LRD) and the generation at which it was observed (t) are given in Table 4 for several combinations of N , heritability in the base population ($H^{[0]}$) and proportion selected (α). Without exception, LRD increased with heritability and α , and it ranged between about 1.7 to 24%. In general, LRD increased with N . However, with $H^{[0]}=0.5$ and $\alpha=0.20$, LRD was 4.18% for $N=1, 2, 3$ and 5; LRD was 6.29% and invariant to N for $H^{[0]}=0.8$ and $\alpha=0.2$, in which case $\bar{t}=2$.

In general \bar{t} increased (or remained the same) as α and N increased, but decreased as heritability increased. The exception was with $N=1$ and $\alpha=0.85$, where \bar{t} increased from 2 to 3 as heritability changed from 0.2 or 0.5 to 0.8. Effects of heritability and of α on \bar{t} were amplified as the number of loci increased.

Table 5 presents the relative difference between genetic means of the A and R lines at Generations 2, 10 and 20 as a function of number of loci (N), heritability ($H^{[0]}$) and α . At generation 2, this relative difference, always in favor of the A line, was invariant to N and it increased with heritability and with α . The range of

values at Generation 2 was from 1.72% (all N , $H^{[0]}=0.2$, $\alpha=0.20$) to 13.85% (all N , $H^{[0]}=0.8$, $\alpha=0.85$). At Generation 10, the relative difference ranged between -1.28% ($N=1$, $H^{[0]}=0.8$, $\alpha=0.20$) to 22.81% ($N=100$, $H^{[0]}=0.8$, $\alpha=0.85$). At this generation, the effect of heritability on the relative difference depended on N and on α . For $N>1$, the relative difference increased with heritability, but with $N=1$ and $\alpha=0.20$, it decreased. However, with $N=1$ and $\alpha=0.85$, the largest difference was at $H^{[0]}=0.5$. The relative difference in Generation 10 increased with N and α without exception. At Generation 20, the relative difference ranged from -2.92% ($N=1$, $H^{[0]}=0.8$, $\alpha=0.20$) to 23.89% ($N=100$, $H^{[0]}=0.8$, $\alpha=0.85$). The relative difference at Generation 20 increased with heritability for $N>2$. With 1 or 2 loci, the effect of heritability depended on N and on α , but the relative difference increased with N and α . With one exception ($N=1$, $H^{[0]}=0.8$, $\alpha=0.20$) assortative mating of parents always led to faster genetic change due to selection than random mating.

Computer simulation

The correlation between phenotypic values of mates in the A line was about 0.8 with $\alpha=0.20$, and about 0.95 with $\alpha=0.85$. The largest difference between the A and

Table 5. Relative difference^a (%) between genetic means of the assortative and random mating lines at Generations 2, 10 and 20 (Model B)

No. of loci	Heritability in base population	Proportion selected					
		0.20			0.85		
		Generation			Generation		
		2	10	20	2	10	20
1	0.2	1.72	0.79	0.28	4.78	3.54	2.24
1	0.5	4.18	0.42	0.73	10.44	5.28	1.10
1	0.8	6.29	-1.28	-2.92	13.85	4.44	2.99
2	0.2	1.72	1.49	1.02	4.78	5.76	4.81
2	0.5	4.18	2.24	0.91	10.44	11.01	7.04
2	0.8	6.29	2.38	0.20	13.85	13.81	6.56
3	0.2	1.72	1.74	1.38	4.78	6.56	5.95
3	0.5	4.18	2.89	1.78	10.44	13.13	10.16
3	0.8	6.29	3.42	1.64	13.85	16.93	11.67
5	0.2	1.72	1.96	1.72	4.78	7.23	6.98
5	0.5	4.18	3.41	2.62	10.44	14.88	13.17
5	0.8	6.29	4.21	2.98	13.85	19.39	16.40
10	0.2	1.72	2.12	2.02	4.78	7.74	7.84
10	0.5	4.18	3.81	3.36	10.44	16.23	15.75
10	0.8	6.29	4.77	4.06	13.85	21.20	20.25
100	0.2	1.72	2.27	2.30	4.78	8.22	8.68
100	0.5	4.18	4.17	4.08	10.44	17.46	18.33
100	0.8	6.29	5.25	5.06	13.85	22.81	23.89

^a See Table 4 for computation of this difference

Table 6. Largest difference between means of the assortative and random mating lines, as a percentage of the selection limit (Δ), and generation at which it was observed^a (computer simulation)

No. of loci	Proportion selected			
	0.10		0.85	
	Δ	Generation	Δ	Generation
1	2.50 \pm 1.19	2	0	—
2	3.00 \pm 1.00	3	8.75 \pm 2.09	15
3	2.17 \pm 0.86	4	13.17 \pm 1.54	19
5	1.20 \pm 0.66	7	12.40 \pm 1.28	23
10	2.50 \pm 0.60	7	13.75 \pm 0.94	32

^a Entries are averages of 200 replicates; standard error calculated from the Monte Carlo sampling variance. See Table 1 for calculation of Δ

R lines relative to the selection limit (Δ), and the generation at which it was observed are given in Table 6 for several combinations of N and α . As with Model A, larger differences in favor of the A line were observed with $\alpha = 0.85$ than with $\alpha = 0.20$, except in one case ($N = 1$, $\alpha = 0.85$). However, the relationship between Δ and N was unclear, especially with $\alpha = 0.20$. The generation at which Δ was observed also increased with α and with N , except when the latter varied between 5 and 10 with $\alpha = 0.20$.

Discussion

Selection response is a function of the heritability of the trait and of the selection differential. The latter depends on the proportion selected and on the phenotypic variance. Positive assortative mating increases the additive genetic variance (e.g., Bulmer 1980) so the heritability and the selection differential are increased by this system of mating. Thus, response to selection is expected to increase.

The results of this study were in agreement with the above theory. Because in Models A and B, populations of infinite size were considered, the same selection limit was reached with each mating system. Before reaching the selection limit, however, the mean of the assortative mating line was larger than that of the random mating line in almost all cases examined.

In the absence of selection, the effect of assortative mating on additive genetic variance increases with heritability in the base population (Bulmer 1980). A similar relationship should be expected between heritability in the base population and the effect of assortative mating on selection response. This was examined with Models A and B; in general, the advantage of

assortative mating over random mating was an increasing function of heritability in the base population.

Model A was used to examine the impact of initial gene frequency (p) on the effect of assortative mating on response to selection. The advantage of assortative mating over random mating was larger with $p = 0.1$ than with $p = 0.5$. When gene frequency is 0.5, additive genetic variance is maximum (Falconer 1981). Starting from $p = 0.1$, selection increases additive genetic variance and heritability until the gene frequency reaches 0.5; thereafter, further selection decreases heritability. The effect of assortative mating on selection response would then be expected to be larger with $p = 0.1$ than with $p = 0.5$. This is so because until the gene frequency reaches 0.5, both selection and assortative mating work towards increasing additive genetic variance. When $p = 0.5$, selection and assortative mating would have opposite effects on additive variance.

Selection eventually reduces the additive genetic variance due to an increase (or decrease) in gene frequency, and to negative covariances between alleles. Hence, assortative mating is expected to have a larger effect at low selection intensities. Results obtained with both models and with the computer simulation confirmed this theory.

When the number of loci (N) is large, selection can proceed for a longer period of time before the genetic variance is depleted. Thus, even small effects of assortative mating on response to selection per generation can accumulate in the long term to yield a relatively large impact. Further, positive assortative mating induces a positive correlation between alleles, which is evident from equations (16) and (18b); this explains the increase in additive genetic variance resulting from assortative mating. For a trait determined by N loci, $4N^2 - 2N$ covariances between allelic effects contribute to additive genetic variance. Therefore, the effect of assortative mating on response to selection would be expected to increase with N . This was verified using Model B. Results from Model A and from computer simulation, however, did not show a clear relationship between N and the effect of assortative mating on selection response.

Under the assumption of multivariate normality of additive genetic and phenotypic values, theoretical heritability (H_T) is identical to realized heritability (H_R). In Model A, where the trait was determined by a few loci with 2 alleles per locus, large differences between H_T and H_R were observed (see Tables 2 and 3). The magnitude and direction of the differences depended on the point of truncation (t_0) relative to the range of the genotypic values. When the point of truncation was outside the range of the genotypic values, H_T was larger than H_R . Realized heritability was largest when t_0 was within the range of

the genotypic values. For example, with $N = 3$, $H_T^{[0]} = 0.5$, $p = 0.1$ and $\alpha = 0.85$, t_0 was negative in the assortative and random mating lines from generation 0 to 5, and H_R was lower than H_T (Table 2). With $N = 3$, $H_T^{[0]} = 0.5$, $p = 0.1$ and $\alpha = 0.20$, $H_T^{[n]}$ was less than $H_R^{[n]}$ for $N < 3$ (Table 3). Thus, when a trait is determined by a few loci, theoretical heritability may not be useful for predicting expected response to selection.

Although Δ was larger with $\alpha = 0.85$ than with $\alpha = 0.20$ (Table 1), the results in Tables 2 and 3 indicate that when $p = 0.1$, the advantage of assortative mating over random mating at generation 2 was larger with $\alpha = 0.20$ than with $\alpha = 0.85$. In the experiment of Mwenya et al. (1983), where response to selection for pupa weight in *Tribolium castaneum* was studied for 2 generations, the advantage of assortative over random mating was larger with $\alpha = 0.20$ than with $\alpha = 0.50$; this is consistent with the results discussed above.

Overall, except for the effect of N , results from Models A and B were in agreement. The main differences between these two models was in the number of alleles per locus: 2 alleles in Model A, and an infinite number in Model B. These correspond to two extreme genetic systems with respect to number of alleles, and the true state of nature for most quantitative traits is perhaps intermediate.

Results of this study indicate that assortative mating can be used to enhance long-term response to selection. The effect of assortative mating was largest when heritability and proportion selected were high, and when the initial gene frequency was low. It should be kept in mind, however, that while assortative mating can alter the curve of response to selection, it is incapable of changing the ultimate limit, at least under additive gene action and infinite population size. In the context of natural selection, the results also suggest that given a trait related to fitness, and two populations competing for scarce resources, an assortative mated population could potentially displace a randomly mated one.

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Appendix

Calculation of the phenotypic mean of selected parents and of the correlation between mates (Model A)

Mean of selected parents

Let X be the phenotypic value of an individual in the unselected population. The phenotypic mean of individuals selected as parents in a truncation scheme is

$$\mu_S^{[n]} = E(X | t_0^{[n]} < X < t_3^{[n]}) = \sum_{i=1}^{3^N} E(X | t_0^{[n]} < X < t_3^{[n]}; X \in i).$$

$$\text{Prob}(X \in i | t_0^{[n]} < X < t_3^{[n]}), \quad (\text{A.1})$$

where i indexes the genotypic value of an individual. If X has genotype i then

$$X = \mu_i + Z\sigma \quad (\text{A.2})$$

where $Z \sim N(0, 1)$. Hence

$$E(X | t_0^{[n]} < X < t_3^{[n]}; X \in i) = E(\mu_i + Z\sigma | a_i < Z < b_i) \quad (\text{A.3})$$

where

$$a_i = (t_0^{[n]} - \mu_i)/\sigma \quad \text{and} \quad b_i = (t_3^{[n]} - \mu_i)/\sigma. \quad (\text{A.4})$$

From properties of the truncated normal distribution

$$E(Z | a_i < Z < b_i) = \frac{\phi(a_i) - \phi(b_i)}{\Phi(b_i) - \Phi(a_i)}, \quad (\text{A.5})$$

where $\phi(\cdot)$ and $\Phi(\cdot)$ are the standard normal density and distribution functions, respectively. Now, the second term in (A.1) is

$$\begin{aligned} \text{Prob}(X \in i | t_0^{[n]} < X < t_3^{[n]}) &= \\ \text{Prob}(X \in i | t_0^{[n]} < X) &= g_i^{[n+1]}. \end{aligned} \quad (\text{A.6})$$

Using (A.5) in (A.3), and then (A.3) and (A.6) in (A.1) gives the mean of the selected parents.

Correlation between mates

Let U and V be the phenotypic value of males and females selected as parents, respectively, and let X and Y be the phenotypic values of unselected males and females. Because $\text{Var}(U) = \text{Var}(V)$,

$$\rho = \text{Cor}(U, V) = \text{Cov}(U, V) / \text{Var}(U). \quad (\text{A.7})$$

Without sexual dimorphism, $E(U) = E(V)$ so

$$\text{Cov}(U, V) = E(UV) - E^2(U). \quad (\text{A.8})$$

Now

$$E(UV) = \sum_{k=1}^3 E[XY | t_{k-1}^{[n]} < (X, Y) < t_k^{[n]}] \cdot \text{Prob}[t_{k-1}^{[n]} < (X, Y) < t_k^{[n]}] \quad (\text{A.9})$$

and

$$E[XY | t_{k-1}^{[n]} < (X, Y) < t_k^{[n]}] = E(X | t_{k-1}^{[n]} < X < t_k^{[n]}) \cdot E(Y | t_{k-1}^{[n]} < X < t_k^{[n]}) \quad (\text{A.10})$$

because mating occurs at random within phenotypic classes. Further, since X and Y have identical distributions

$$E[XY | t_{k-1}^{[n]} < (X, Y) < t_k^{[n]}] = E^2(X | t_{k-1}^{[n]} < X < t_k^{[n]}), \quad (\text{A.11})$$

and

$$E(X | t_{k-1}^{[n]} < X < t_k^{[n]}) = \sum_{i=1}^{3^N} E(X | t_{k-1}^{[n]} < X < t_k^{[n]}; X \in i) \cdot \text{Prob}(X \in i | t_{k-1}^{[n]} < X < t_k^{[n]}). \quad (\text{A.12})$$

From [A.3], [A.4] and [A.5]

$$E(X | t_{k-1}^{[n]} < X < t_k^{[n]}; X \in i) = \mu_i + \sigma \{ \phi[(\mu_i - t_{k-1}^{[n]})/\sigma] - \phi[(\mu_i - t_k^{[n]})/\sigma] \} / \{ \Phi[(t_k^{[n]} - \mu_i)/\sigma] - \Phi[(t_{k-1}^{[n]} - \mu_i)/\sigma] \}. \quad (\text{A.13})$$

Also

$$\text{Prob}(X \in i | t_{k-1}^{[n]} < X < t_k^{[n]}) = P_{ik}^{[n]} / P_k^{[n]} \quad (\text{A.14})$$

and

$$E(U) = E(X | t_0 < X < t_3) \quad (\text{A.15})$$

which is calculated using (A.1)–(A.6).

The $\text{Cov}(U, V)$ can then be calculated using (A.9)–(A.15). Finally,

$$\text{Var}(U) = E(U^2) - E^2(U), \quad (\text{A.16})$$

where

$$E(U^2) = E(X^2 | t_0^{[n]} < X < t_3^{[n]}) \quad (\text{A.17})$$

and

$$E(X^2 | t_0^{[n]} < X < t_3^{[n]}) = \sum_{i=1}^{3^N} E(X^2 | t_0 < X < t_3^{[n]}; X \in i) \cdot \text{Prob}(X \in i | t_0^{[n]} < X < t_3^{[n]}). \quad (\text{A.18})$$

Further

$$E(X^2 | t_0^{[n]} < X < t_3^{[n]}; X \in i) = E[\mu_i^2 + 2\mu_i Z\sigma + Z^2\sigma^2 | a_i < Z < b_i], \quad (\text{A.19})$$

and

$$E(Z^2 | a_i < Z < b_i) = 1 + \frac{a_i \phi(a_i) - b_i \phi(b_i)}{\Phi(b_i) - \Phi(a_i)}. \quad (\text{A.20})$$

Formulae (A.17)–(A.20), and (A.15) can then be used to compute $\text{Var}(U)$. The correlation between phenotypic values of mates is then calculated with (A.7).