Genet. Res., Camb. (1993), **61**, *pp.* 205–224 *With 7 text-figures*

Directional selection and the evolution of sex and recombination

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(*Received 21 July 1992 and in revised form 17 November 1992*)

Summary

Models of the evolutionary advantages of sex and genetic recombination due to directional selection on a quantitative trait are analysed. The models assume that the trait is controlled by many additive genes. A nor-optimal selection function is used, in which the optimum either moves steadily in one direction, follows an autocorrelated linear Markov process or a random walk, or varies cyclically. The consequences for population mean fitness of a reduction in genetic variance, due to a shift from sexual to asexual reproduction are examined. It is shown that a large reduction in mean fitness can result from such a shift in the case of a steadily moving optimum, under light conditions. The conditions are much more stringent with a cyclical or randomly varying environment, especially if the autocorrelation for a random environment is small. The conditions for spread of a rare modifier affecting the rate of genetic recombination are also examined, and the strength of selection on such a modifier determined. Again, the case of a steadily moving optimum is most favourable for the evolution of increased recombination. The selection pressure on a recombination modifier when a trait is subject to strong truncation selection is calculated, and shown to be large enough to account for observed increases in recombination associated with artificial selection. Theoretical and empirical evidence relevant to evaluating the importance of this model for the evolution of sex and recombination is discussed.

1. Introduction

Mather (1943) first pointed out that a quantitative trait under partly directional and partly stabilizing selection is under two opposing pressures of selection on the amount of genetic variation in the trait. In a constant environment, where the mean of the trait rapidly approaches the optimal value, increased genetic variability lowers the mean fitness of the population because of the production of phenotypically more extreme individuals with lower fitnesses. If the environment is changing, so that the mean deviates from the optimum, the population mean fitness may be increased by increasing the genetic variance of the trait, allowing a faster rate of tracking of the optimum.

Mather recognized that a major effect of selection on the genetic variance of a trait is mediated by linkage disequilibrium between alleles at different loci controlling the trait. In a constant environment, the reduction in phenotypic variance in a heritable trait caused by stabilizing selection results in the generation of negative linkage disequilibrium, such that 'plus' alleles affecting the trait tend to be associated in the population with 'minus' alleles at other loci. If the loci affecting the trait are approximately additive in their effects, these associations result in a negative covariance between the effects of different loci, which reduces the genetic variance below that expected in the absence of such covariance (Bulmer, 1974). The lower the frequency of genetic recombination between loci, the larger this effect.

It follows that mean fitness in a constant environment is likely to be largest when recombination is absent, and so asexual reproduction or genes causing zero recombination should be favoured in a constant environment [see Felsenstein (1974) for a justification of the use of mean fitness in this context]. Conversely, if the environment is changing sufficiently fast, breakdown of this negative linkage disequilibrium by increased genetic recombination should be favoured, as the resulting increase in genetic variance will allow a more rapid response to the resulting pressure of directional selection. These notions are at the core of Mather's influential concept of a conflict between immediate fitness and genetic flexibility. This concept has usually been interpreted in terms of group selection rather than selection on genotypes or individuals (e.g.

Stebbins, 1950, chap. 5). Presumably for this reason, the role of increased genetic variance in promoting the response to selection has been neglected in more modern theories of the evolution of sex and recombination (Michod & Levin, 1988).

In the past few years, there has been a revival of interest in the notion that selection on quantitative traits may lead to a selective advantage to sex and recombination in temporally varying environments (Maynard Smith, 1980, 1988; Bergman & Feldman, 1990, 1992; Korol, Preygel & Preygel, 1990; Crow, 1992). This was preceded by the development by Bulmer (1974, 1985) and Lande (1975) of detailed models of the effects of selection on the genetic variance of quantitative traits, and the realization that significant effects of linkage disequilibrium may be detectable at the level of the genetic variance for the trait even though pairwise associations between loci are very weak.

There is now no doubt that the type of process outlined above is mechanistically sound, and capable of generating advantages to sex and recombination at both the genotypic and population level (Maynard Smith, 1988). In addition, there is evidence from artificial selection experiments that a response to directional selection on a trait is frequently associated with a correlated increase in recombination rate (Flexon & Rodell, 1982; Korol *et al.* 1990; Gorodetskii, Zhuchenko & Korol, 1991), suggesting that this idea may have an empirical basis. A similar process of the break-down by recombination of negative linkage disequilibrium generated by selection underlies the selective advantage to increased recombination when there are synergistic interactions among the fitness effects of deleterious alleles maintained by mutation (Feldman, Christiansen & Brooks, 1980; Kondrashov, 1988; Charlesworth, 1990).

This paper is concerned with the theoretical analysis of the conditions with respect to the intensity of selection and rate of change of the environment that are required to produce advantages to sex and recombination. Four modes of selection, respectively involving a steady shift in the optimum, a randomly varying optimum, a cyclically varying optimum, and directional truncation selection, will be considered here. The effects of sexual versus asexual recombination on the mean fitness of the population, and the direction and strength of selection on rare modifiers of the rate of recombination, will be analysed. The first topic is relevant to the question of whether or not sexual populations can be invulnerable to invasion by asexual variants that evade the reproductive cost of sex (Maynard Smith, 1978). The second is relevant to the question of the contribution of selection in varying environments to the maintenance of non-zero levels of genetic recombination, and to the promotion of differences in rates of recombination between populations or taxa.

2. Nor-optimal selection with a steadily moving optimum

(i) *Relation of population mean fitness to genetic variance*

Consider an additively inherited quantitative trait subject to a nor-optimal selection scheme with a moving optimum, such that in generation n the fitness of an individual with trait value z is $w_z = \exp -(z-\theta_n)^2/(2\omega^2)$, where θ_n is the optimal value in generation n and $1/\omega^2$ measures the strength of selection. Let the additive genetic variance in the trait be V_G and the environmental variance be V_E. Following Turelli (1984), let $V_S = V_E + \omega^2$ be a composite of the environmental variance and the inverse of the strength of selection. In what follows, all trait values will be assumed to be measured on a scale on which $V_E = 1$. From the standard selection equation (Bulmer, 1985, p. 151), we obtain the following expression for the change in population mean

$$\bar{z}_n = (1-k)\,\bar{z}_{n-1} + k\theta_{n-1}, \tag{1}$$

where $k = V_G/(V_G + V_S)$.

If the optimum changes at a steady rate $\Delta\theta$ per generation, this gives

$$\bar{z}_n - \theta_n = (1-k)(\bar{z}_{n-1} - \theta_{n-1}) - \Delta\theta \tag{2}$$

and so the difference between the population mean and the optimum equilibrates at

$$\theta - \bar{z} = \Delta\theta/k. \tag{3}$$

This result has been obtained independently by Lynch, Gabriel & Wood [1991, eqn (15)].

The mean fitness of the population (Latter, 1970) is given by

$$\bar{w} = \frac{\omega}{\sqrt{(V_G + V_S)}} \exp -\frac{(\bar{z}-\theta)^2}{2(V_G + V_S)}. \tag{4}$$

With a steadily moving optimum, the population mean fitness thus equilibrates at a value given by

$$\ln \hat{w} = \ln \omega - \tfrac{1}{2}\ln (V_G + V_S) - \frac{(\Delta\theta)^2 (V_G + V_S)}{2V_G^2}. \tag{5}$$

A related formula is given by Lynch & Lande [1992, eqn (9)].

The effect of a change in additive variance on mean fitness can be determined by examining the derivative of this function with respect to V_G. It is convenient to scale V_G and $(\Delta\theta)^2$ relative to V_S. Let $V = V_G/V_S$ and $\phi = (\Delta\theta)^2/V_S$. Turelli (1984) suggested that a typical value of V_S relative to V_E is between 10 and 20. Even if the heritability of the character is extremely high, this implies that it is safe to assume that V is small, so that second-order terms in V can be neglected, to a good approximation. We then have

$$\ln \hat{w} \approx \ln \omega - \tfrac{1}{2}\ln V_S - \tfrac{1}{2}V - \phi/2V^2. \tag{6}$$

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The condition for $\partial \hat{w}/\partial V > 0$ is

$$2\phi > V^3, \tag{7a}$$

or

$$2(\Delta\theta)^2 > V_G^3/V_S^2. \tag{7b}$$

If V_S is taken to be approximately $20V_G$, in accordance with the assumptions just discussed, then the condition for a mean fitness advantage to an increase in V_G is roughly

$$|\Delta\theta| > 0{\cdot}035\sigma_G \tag{7c}$$

where σ_G is the additive genetic standard deviation and $|\Delta\theta|$ is the absolute value of the rate of change of optimum.

This shows that the absolute value of the rate of change of optimum must be of the order of $\frac{1}{28}$th of the genetic standard deviation or more, for the advantage of an enhanced speed of tracking of the environment to overcome the disadvantage due to a higher genetic load under stabilizing selection. If this critical value is not reached, then there will be a reduction in mean fitness associated with a small increase in genetic variance.

It is useful to relate these criteria to the genetic load imposed by the combination of stabilizing and directional selection. Following Charlesworth (1984*b*), the first three terms on the right-hand side of equation (6) reflects the reduction in log fitness due to stabilizing selection. The fourth term reflects the additional load due to directional selection. If condition (7*a*) is satisfied, the directional selection load term is greater than $0{\cdot}25V$, e.g. 0·0125 with $V_S = 20V_G$. Since the stabilizing selection load term under this condition is approximately 0·025, this does not represent a massive increase in load. At the level of mean fitness, a modest increase in load due to directional selection is thus sufficient to create an advantage to a small increase in genetic variance.

The above condition is conservative; owing to the nonlinear nature of the dependence of log mean fitness on V in equation (6), a large increase in V will cause an increase in mean fitness under much lighter conditions. If V is multiplied by a factor of $C < 1$, then mean fitness will decrease if $\phi(C+1) > C^2V^3$, a lighter condition than (7*a*). The corresponding directional selection load is $0{\cdot}5C^2V/(C+1)$. Thus, a shift to an asexual mode of reproduction, or to zero recombination, if accompanied by a sufficiently large decrease in genetic variance, will result in a lower equilibrium population mean fitness under quite light conditions. A population founded by a single asexual clone, with no genetic variance, suffers an equilibrium fitness loss of infinity under this model, since it fails to keep up with the moving optimum, and so will rapidly decline in abundance in competition with a genetically variable sexual population (cf. Crow, 1992). This is somewhat unrealistic, as new variability will rapidly be introduced into the asexual population by mutation. An alternative, and more realistic, perspective is to consider competition between sexual and asexual populations which are both in equilibrium with respect to genetic variance. This is the subject of the next section.

(ii) *Relation of population mean fitness to genetic variance and breeding system*

A crude approach to this question is to assume that the number of loci affecting the trait is large, and the effect of each locus is small. With random mating, the additive genetic variance at the start of a generation can be partitioned into the genic variance V_A, composed of the sum of the additive variance terms contributed by each locus, and the linkage disequilibrium covariance term C_L. Under these conditions, changes in genetic variance reflect only changes in C_L (Bulmer, 1985, p. 150). If this is the case, the genetic variance will not change as a result of the change in optimum, since the effect of selection on variance is independent of θ (Bulmer, 1985, p. 151). The equilibrium genetic variances under stabilizing selection alone can then be used to calculate V_G in the above equations.

For convenience, a model of quantitative variability maintained by the interaction between mutation and stabilizing selection will be used here. If recombination is free, the considerations presented by Turelli (1984) suggest that, for most biologically realistic parameter sets, the equilibrium genetic variance under stabilizing selection for a sexual haploid is approximated by the Latter–Bulmer formula

$$V_G \approx 2muV_S, \tag{8a}$$

where m is the number of loci and u is the mutation rate per locus. The variance is twice this for a diploid, for the same mutation rate. This formula should provide a good approximation provided that u is sufficiently small compared with the product of $1/V_S$ and the mean square of the effect of a new mutation on the trait (Turelli, 1984, p. 185).

For a non-recombining or asexual haploid genome, we effectively have a single locus with mutation rate mu. Under this circumstance, the Gaussian model of allelic effects of Kimura (1965), Lande (1975) and Fleming (1979) becomes appropriate, since the 'per locus' mutation rate is likely to be high relative to the product of $1/V_S$ and the mean square of the effect of a new mutation (Turelli, 1984). The equilibrium genetic variance for an asexual or non-recombining haploid is given by

$$V_G \approx \sqrt{(V_m V_S)}, \tag{8b}$$

where V_m is the variance per generation due to new mutations. If the mean square of the allelic effects of new mutations is a^2, $V_m = mua^2$.

A similar result holds for asexual diploids, but V_m is now $2mua^2$, where a^2 is the mean square heterozygous

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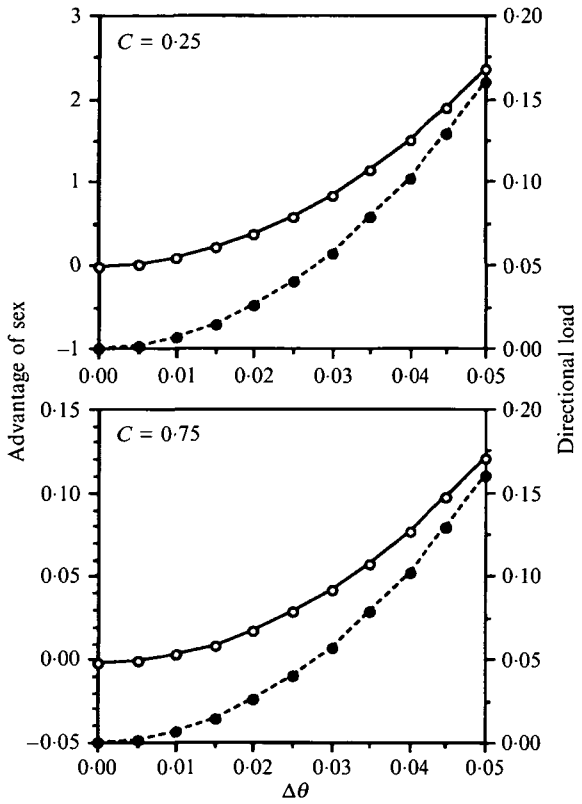

Fig. 1. The selective advantage to asexual reproduction (○) and load due to directional selection (●) as functions of the rate of change of optimum $\Delta\theta$, in the case of a steadily moving optimum. The selective advantage of sexual reproduction is measured by the difference in equilibrium log mean fitness between a high variance (sexually reproducing) population, and a low variance (asexual) population. The directional selection load is expressed as the reduction in log mean fitness of the population below that in an unchanging environment. $\Delta\theta$ is measured on a scale in which the environmental variance of the trait is unity; the genetic variance of the sexual population on this scale is $V_G = 0{\cdot}4$, and $V_S = 20$. The genetic variance of an asexual population is assumed to be a factor of C times that of a sexual population ($C = 0{\cdot}25$ or $0{\cdot}75$). For $\Delta\theta$ close to zero, there is in fact a small disadvantage of sex in both cases ($-0{\cdot}0074$ for $\Delta\theta = 0$ and $C = 0{\cdot}25$; $-0{\cdot}001$ for $\Delta\theta = 0$ and $C = 0{\cdot}75$).

allelic effect of a new mutation. The argument is slightly more subtle for non-recombining but sexual diploids. In this case, segregation of the single chromosome in each generation removes the covariance induced by selection between maternal and paternal genomes [the 'Hardy–Weinberg disequilibrium' covariance: Bulmer (1985, p. 158)]. Thus, only a portion of the reduction in genetic variance created by selection in a given generation is transmitted to the next generation, instead of all of it as assumed in the model leading to equation (8*b*) [cf. Lynch & Gabriel, 1983, equation (6)]. Following Bulmer (1985, p. 159), it seems reasonable to assume that one-half of the reduction in genetic variance is due to Hardy–Weinberg disequilibrium when the number of loci is large, and so only one-half (that due to linkage disequilibrium) is transmitted to the next generation. Using this in the formulation of Lynch & Gabriel (1983) for asexual populations, we obtain the approximate equilibrium genetic variance for a sexual diploid with zero recombination as

$$V_G \approx \sqrt{(2V_m V_S)}. \qquad (8c)$$

For values of *mu* of the order of 0·01, and with V_S of the order of 10–20 times V_E, it is found that formula (8*a*) predicts a higher equilibrium V_G than (8*b*) or (8*c*), because the genetic variance in (8*b*) or (8*c*) increases as the square root of the mutation rate, but (8*a*) is linear in the mutation rate (cf. Turelli, 1984; Table 1). Thus, with mutation and selection parameters that appear to be biologically reasonable, the equilibrium variance for a freely recombining sexual population may be several times that for a population with no recombination. For example, with $mu = 0{\cdot}02$, $a^2 = 0{\cdot}05$, and $V_S = 20V_E$, the equilibrium genetic variance for a freely recombining haploid population is approximately four times that of a haploid population which lacks recombination.

Fig. 1 shows the relation between the difference s between the log mean fitnesses of a freely recombining sexual population and an asexual population and the rate of change of the optimum, for two different magnitudes of the effect of asexuality on the equilibrium genetic variance. [The more exact equation (5) was used in these calculations.] When s is small, this is approximately the same as the selection coefficient against an asexual line that has reached mutational equilibrium, competing against a sexual population, ignoring any inherent reproductive advantage to asexuality. For small values of $|\Delta\theta|$, there is a disadvantage to sexual reproduction, as predicted from the considerations above. For the advantage of the sexual population to overcome a two-fold reproductive cost of sex, we require $s > \ln 2 = 0{\cdot}69$. With a four-fold reduction in genetic variance, this is accomplished when $|\Delta\theta| > 0{\cdot}03$, and the directional selection load for the sexual population is 0·057. When the variance is only reduced by 25%, a sufficiently large advantage to overcome the cost of sex is not achieved in the parameter range studied here. Protection against an asexual sub-population with a two-fold reproductive advantage can thus be gained with weak directional selection if the genetic variance is sufficiently reduced by asexuality.

The results are also relevant to the fate of a dominant gene that suppresses recombination, competing against a recessive allele that allows free recombination, since the respective long-term mean finesses determine the fate of the competing alleles in this case (Felsenstein, 1974). There is no reproductive cost to recombination suppression, so that the conditions for maintenance of non-zero recombination are less stringent than those for maintenance of sexual reproduction [with the qualification that the reduction in variance for diploid populations associ-

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ated with recombination suppression is smaller by a factor of 0·7 than that associated with asexuality: cf. equations (8*b*) and (8*c*)].

(iii) *Selection on a modifier of recombination*

The conditions derived above therefore suggest that there will be selection maintaining non-zero recombination when the rate of change of optimum $|\Delta\theta|$ exceeds a threshold level, which must be higher for a diploid than a haploid population. If $|\Delta\theta|$ is below this threshold, the advantage of reducing genetic variance created by stabilizing selection causes a pressure of selection for reduced recombination. This leaves open the question of what genome-wide frequency of recombination is favoured if there is indeed selection for non-zero recombination. This can be investigated by examining the fate of genic modifiers with minor effects on recombination, and attempting to determine nature of the ESS frequency of recombination that is sufficient to ensure immunity to invasion by such modifiers.

The case of selection on a rare modifier allele affecting recombination in a haploid population will be considered in most detail in this section, using an approach similar to that of Charlesworth (1990) for the case of mutation-selection balance. As in that case, the effect of selection on a modifier of recombination depends on the asymptotic differences in mean and variance between carriers of the modifier and the general population. Let these quantities be $\delta\bar{z}$ and δV_G respectively. The derivation of general equations for these is given in the Appendix, using a modification of Bulmer's (1985, p. 158) infinite locus model for selection on a quantitative character [equations (A 2) and (A 9)]. Given values of $\delta\bar{z}$ and δV_G, the selection coefficient s on a modifier can be calculated from the deviation of log mean fitness of the modifier population from that of the general population, $\delta \ln \bar{w}$ [equation (A 11*a*)]. This measures the asymptotic rate of change in allele frequency x of the modifier while rare, $\Delta x/x$. In general, $\delta\bar{z}$ and δV_G are complicated functions of the numbers of chromosomes, their map lengths, and the position of the modifier within the chromosome on which it is carried (cf. Charlesworth, 1990).

Some useful insights into the nature of selection on recombination can, however, be obtained without taking these details into account. Equations (A 11*a*, *b*) shows that the direction of selection on the modifier depends only on the direction of its effect on variance, and on the sign of a quantity that depends on the current variance, the magnitude of rate of change of the optimum, and on the harmonic mean of the frequency of recombination between the modifier and the selected loci, ρ_H. With nor-optimal selection, a modifier which has the same direction of effect on recombination on all pairs of loci that it affects will be associated with a change in variance of the same sign as its effect on recombination [see Appendix, section (ii)].

Using equation (A 11*b*), it is easily seen that a population with free recombination ($\rho_H = 0{\cdot}5 \gg V$) will be invulnerable to invasion by a modifier that reduces recombination by a small amount if and only if

$$3\phi > V^2. \tag{9a}$$

This can be compared with the corresponding sufficient condition for immunity to invasion by a factor that completely suppresses recombination [equation (7*a*)]. Since the right-hand side of (9) involves V^2 instead of V^3, the condition is considerably more stringent than that for a suppressor of recombination. If V is 1/20, equation (9*a*) yields the condition comparable to (7*c*)

$$|\Delta\theta| > 0{\cdot}129\sigma_G. \tag{9b}$$

Equation (7) also gives the condition for the spread of a recessive modifier that introduces some recombination into a non-recombining population (in which V is expected to be considerably smaller than with free recombination, reducing the stringency of the condition). This can be compared with the condition derived from equation (A 11*b*) for a dominant modifier increasing recombination away from zero ($\rho_H = 0$):

$$\phi(2+V) > V^3. \tag{9c}$$

For small V, the two conditions are nearly identical, indicating that dominance has little effect on the condition for invasion of a non-recombining population by a modifier that increases recombination.

These results indicate that non-zero recombination can be favoured under conditions under which free recombination is disfavoured, suggesting the existence of an ESS such that the mean recombination frequency for a pair of trait loci is considerably less than one-half. Some general notion of the nature of the ESS can be obtained from equations (9) by noting that, with the assumptions made here, V depends only weakly on the recombination frequencies, unless these are mostly close to zero. From equation (A 3*c*), we obtain the expression

$$V \approx \tilde{V}_A\left(1-\left(\frac{1}{r_H}-1\right)\tilde{V}_A+\left(\frac{1}{r_H}-1\right)^2\tilde{V}_A^2-\ldots\right), \tag{10}$$

where $\tilde{V}_A = V_A/V_S$. From the considerations presented above, second-order terms in $\tilde{V}_A$ are likely to be small. Hence, unless r_H is of the same order as $1/\tilde{V}_A$ (i.e. recombination is near zero), r_H has only a second-order effect on V. Over a wide range of recombination frequencies, V in equations (A 11*a*, *b*) can thus be treated as independent of recombination and equated to $\tilde{V}_A$, to a first-order approximation. Thus, the modifier will be neutral when

$$\rho_H \approx \frac{2}{1+\tilde{V}_A^2/\phi}. \tag{11}$$

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While the value of ρ_H is in general dependent on the position of the modifier on the chromosome on which it is located, equation (27) of Charlesworth (1990) shows that this dependence is logarithmic, and hence rather weak, if the number of trait loci is large. This means that ρ_H provides an index of the average amount of recombination in the genome under a wide range of conditions. Numerical examples show that ρ_H is very close to the harmonic mean recombination frequency between a random pair of loci, under the assumption of no interference and a random distribution of loci over chromosomes. Note that $\rho_H \leqslant 0{\cdot}5$, and that the ESS value of ρ_H is zero unless the condition of equation (7) is satisfied by the value of V given by equation (8*b*).

This analysis provides no insight into the strength of selection propelling the population towards the ESS. As discussed above, this depends on the linkage relations of the modifier with the genes controlling the trait under selection. Two limiting cases that yield simple results can be analysed: a single chromosome with a low rate of recombination, and free recombination between the modifier and the trait loci. The latter case provides a good approximation to the strength of selection on a modifier in a genome with several chromosomes, where most loci affected by the modifier are unlinked to it. These cases will be considered in the following two sections.

(iv) *A single chromosome with a low rate of recombination*

In this case, the products of recombination frequencies in equation (A 9) can be neglected in comparison with the recombination frequencies themselves (this is also the case if there is complete interference). Following Charlesworth (1990), it seems reasonable to consider the case when the modifier affects the map length l of the chromosome (in Morgans) by an amount $\delta l = \epsilon l$, where ϵ is the proportional effect of the modifier on map length. If loci are uniformly affected by the modifier, we have $\delta r_{ij} \approx \epsilon r_{ij}$ when recombination rates are low. Assume that a proportion p of loci are located to the left of the modifier's position on the chromosome, and a proportion $p = 1-p$ to the right (Charlesworth, 1990, p. 209). With a uniform distribution of loci along the chromosome (Charlesworth, 1990, p. 210), substitution into equation (A 9) and integration yield the following relation

$$E = E_1\left\{\frac{1}{\rho_i + r_{ij}}\right\} + E_2\left\{\frac{1}{\rho_i + \rho_j}\right\}$$

$$\approx (\ln m - p\ln p - q\ln q)/l. \qquad (12a)$$

Using equation (A 9), we find

$$\delta V \approx \frac{\epsilon E V^2}{1+2VE}. \qquad (12b)$$

Substituting these relations into equation (A 11*a*),

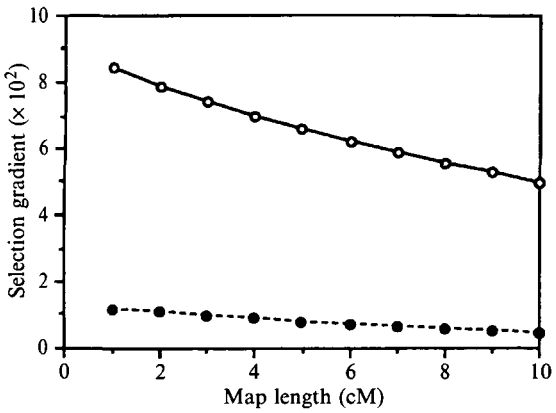

Fig. 2. The selection gradient ($\times 10^2$) for the proportional effect ϵ on map length of a modifier of recombination, expressed as a function of map length, in the case of a steadily moving optimum and a short map length [see equation (13)]. The case when $\Delta\theta/\sigma_G = 0{\cdot}10$ (○) and when $\Delta\theta/\sigma_G = 0{\cdot}05$ (●). It is assumed that $V_G = V_E = 1$ and $V_S = 20$.

and using the fact that in this case $1/\rho_H \approx (\ln m)/l$, we obtain

$$s = \delta(\ln \bar{w}) \approx \frac{\epsilon E}{2(2+VE)}\left(\phi\left(\frac{2\ln m}{l} - 1\right) - V^2\right). \qquad (13)$$

This expression indicates that a substantial pressure of selection can act on a modifier that increases the map length of a single chromosome with a short map, provided that the rate of change of the optimum is sufficiently large in relation to the genetic variance. Fig. 2 shows some examples of this for the case of a modifier situated in the middle of the chromosome, expressed in terms of the selection gradient, s/ϵ. (It is useful to note that, with $l \ll 1$ as assumed here, the selection gradient on map length in Morgans is considerably larger than that on ϵ.) While this case gives the greatest intensity of selection, with E in equation (12*a*) equal to $(\ln 2m)/l$, it is easily seen that with large m there is very little effect of the position of the modifier; a terminally located modifier has an E value of $(\ln m)/l$. Even for a moderate rate of change of optimum, as in the lower of the two curves in Fig. 2, there is a selection gradient of the order of 1% for increased recombination.

(v) *Free recombination between the modifier and the trait loci*

If the modifier recombines freely with all the trait loci, and there are J chromosomes of length l, equation (A 9) yields

$$\delta V \approx \frac{2V^2}{J} E_L\left\{\frac{\delta r_{ij}}{r_{ij}(1+r_{ij})}\right\}, \qquad (14a)$$

where E_L denotes the expectation over all pairs of trait loci on the same chromosome. Using the same assumption as before about the effect of the modifier on map distance, with general recombination fre-

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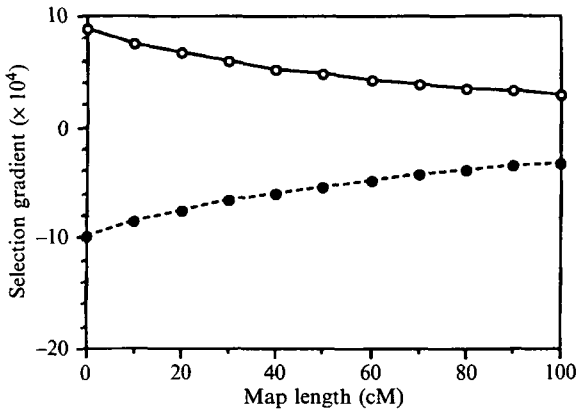

Fig. 3. The selection gradient ($\times 10^4$) on the proportional effect ϵ on map length of a modifier of recombination, expressed as a function of map length, in the case of an unlinked modifier and a single chromosome carrying the trait loci [see equation (15*b*)]. A steadily moving optimum is assumed. The case when $\Delta\theta/\sigma_G = 0{\cdot}15$ (○) and when $\Delta\theta/\sigma_G = 0{\cdot}10$ (●). Other parameters are as in Fig. 2.

quencies we have $\delta r_{ij} < \epsilon r_{ij}$, so that an upper limit to δV is obtained by equating E_L to

$$E'_L = \frac{\epsilon}{l} E\left\{\frac{1}{1+r_{ij}}\right\} \approx \frac{\epsilon}{3l}(l + 2\ln(3-e^{-2l}) - 2\ln 2). \quad (14b)$$

In the case of a short map length for each chromosome, $E_L \approx \epsilon$, and we have

$$s \approx \epsilon(3\phi - V^2)/J. \quad (15a)$$

More generally, the mean value theorem applied to equation (14*a*) with the Haldane (1919) mapping function for no interference gives $E_L \approx E'_L l/(e^l - 1)$. We thus have

$$s \approx \frac{\epsilon(l + 2\ln[\frac{1}{2}(3-e^{-2l})])(3\phi - V^2)}{3J(e^l - 1)}. \quad (15b)$$

The intensity of selection is inversely proportional to the number of chromosomes. For a long map, it is easily seen that this condition produces much weaker selection than equation (15*a*). Fig. 3 shows some numerical example for the case of a single chromosome. (The displayed selection gradients should be divided by the number of chromosomes for cases where there are multiple chromosomes.) The maximum selection gradient for increased recombination is of the order of 10^{-3} in the case of the more rapidly moving optimum; there is selection against increased recombination for the more slowly moving optimum, in contrast to the linked modifier shown in Fig. 2.

3. Nor-optimal selection with a randomly fluctuating optimum

(i) *Relation of population mean fitness to genetic variance*

The above approach can be extended to the case of a randomly fluctuating environment, such that the selective optimum θ_n follows a linear stationary Markov process, with a mean of zero, variance V_θ, and autocorrelation $\tau_i (-1 \leqslant \tau < 1)$ between θ values which are i generations apart i.e. $\theta_n = \tau\theta_{n-1} + \epsilon_{n-1}$, where ϵ_n is a random variable with mean zero and variance V_ϵ. We have $V_\theta = V_\epsilon/(1-\tau)^2$. From standard theory on fluctuating environments (Haldane & Jayakar, 1963; Gillespie, 1973; Karlin & Liberman, 1974), the most appropriate measure of average mean fitness is the expectation of the natural logarithm of mean fitness, i.e.

$$E\left\{\ln\omega - \tfrac{1}{2}\ln(V_G + V_S) - \frac{(\bar{z}-\theta)^2}{2(V_G+V_S)}\right\}. \quad (16)$$

This can be evaluated as follows. Assuming that the genetic variance equilibrates at a constant value V_G, and that V_S is a constant, the following expression is obtained from equation (1) for the mean phenotypic value in a an arbitrary generation n:

$$\bar{z}_n = (1-k)^n \bar{z}_0 + k\sum_{i=1}^{n}(1-k)^{i-1}\theta_{n-i}. \quad (17)$$

The first term on the right-hand side may be neglected for large n. Hence, the asymptotic value of $E\{(\bar{z}-\theta)^2\}$ is given by

$$V\left\{k\sum_{i=1}^{n}(1-k)^{i-1}\theta_{n-i} - \theta_n\right\}$$
$$= V_\theta + k^2 V\left\{\sum_{i=1}^{n}(1-k)^{i-1}\theta_{n-i}\right\}$$
$$-2kE\left\{\theta_n\sum_{i=1}^{n}(1-k)^{i-1}\theta_{n-i}\right\}. \quad (18)$$

Lengthy but straightforward calculations using the properties of the linear stationary process lead to the asymptotic result (valid for $k > 0$)

$$E\{(\bar{z}-\theta)^2\} \approx \frac{2(1-\tau)V_\theta}{(1-\tau[1-k])(2-k)}. \quad (19a)$$

Substituting into equation (16), we obtain

$$E\{\ln\bar{w}\} \approx \ln\omega - \tfrac{1}{2}\ln V_S - \tfrac{1}{2}\ln(1+V)$$
$$-\frac{(1-\tau)V_\theta}{(1-\tau[1-k])(2-k)V_S(1+V)}. \quad (20a)$$

For $V_\theta = 0$, it is obvious that expected log mean fitness decreases with increased genetic variance. With small V, such that $k \approx V$, and with $\tau < 1 - O(V)$, the following expression is obtained, neglecting second-order terms in V:

$$E\{\ln\bar{w}\} \approx \ln\omega - \tfrac{1}{2}\ln V_S - \tfrac{1}{2}\psi + \tfrac{1}{2}V\left(\frac{\psi(1+\tau)}{2(1-\tau)} - 1\right), \quad (20b)$$

where $\psi = V_\theta/V_S$ is the variance in optimum scaled relative to the inverse measure of the strength of selection (ψ is the analogue of ϕ in the case of a steadily moving optimum). Lynch & Lande (1992)

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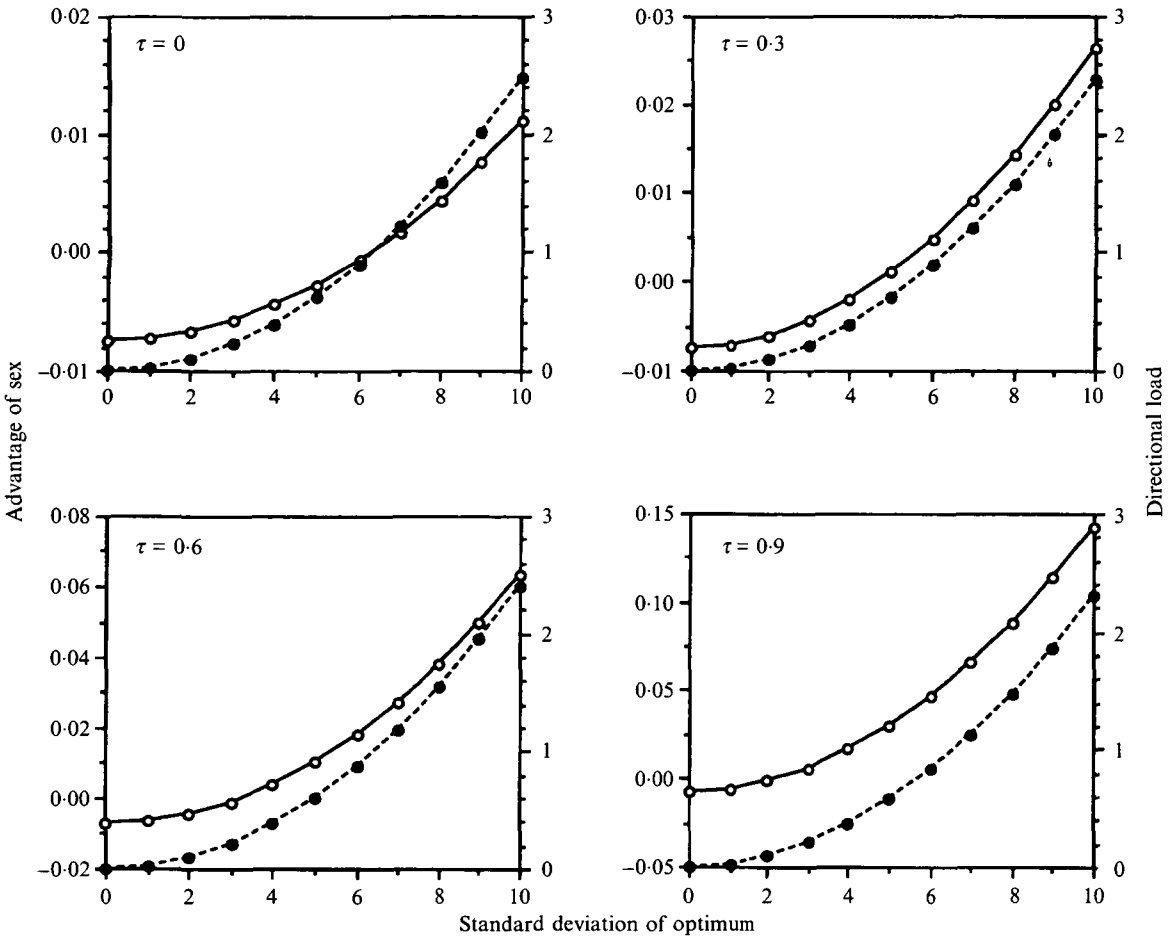

Fig. 4. The selective advantage to asexual reproduction (○) and load due to directional selection (●) as functions of the standard deviation of the optimum, in the case of a randomly varying optimum. Four different cases with different autocorrelations of the optimum are displayed. In each case, the C value for the asexual population is 0·25. Other parameters are as in Fig. 1.

have derived a related expression for the case of zero autocorrelation [their equation (9)].

It is obvious from this expression that the condition for expected log fitness to increase with increased genetic variance is

$$\frac{\psi(1+\tau)}{2(1-\tau)} > 1. \qquad (21a)$$

The stringency of this condition decreases with τ, and is clearly impossible to satisfy when $\tau = -1$. When there is no autocorrelation in the fitness optimum, the condition for expected log mean fitness to increase with increased genetic variance is simply $\psi > 2$. For $\tau = 0{\cdot}5$, it becomes $\psi > 0{\cdot}67$, and for $\tau = 0{\cdot}9$, $\psi > 0{\cdot}11$. As with the case of steadily moving optimum, it is useful to relate condition (21) to the load created by the directional selection imposed by the shifting optimum. This load is enshrined in the terms involving ψ in equations (20). Since the above considerations imply that ψ must be of the order of at least 0·1 for an advantage to increased genetic variance (unless τ is very close to 1), and the dominant directional selection term in equation (20b) is $0{\cdot}5\psi$, the increased load due to directional selection must be of the order of 0·05 or more for there to be an advantage to increased genetic variance. With a low autocorrelation, an extremely high directional load is required. Fig. 4 shows some numerical examples of the magnitude of the difference in expected log mean fitness between sexual (high variance) and asexual (low variance) populations for various sets of parameter values, assuming that the genetic variance of the asexual population is one-quarter that of the sexual. In contrast to the case of a steadily moving optimum, the advantage of sex never approaches a value sufficient to overcome the two-fold reproductive cost of sex. A high autocorrelation is most favourable to sexual reproduction.

If $\tau = 1$, the process is no longer stationary, but is a random walk, with V_θ increasing indefinitely with time, at rate V_ϵ per generation. Applying equation (2) to this case, it is evident that asymptotically we have

$$E\{(\bar{z}-\theta)^2\}(1-[1-k]^2) = V_\epsilon,$$

i.e.

$$E\{(\bar{z}-\theta)^2\} \approx V_\epsilon/2V \qquad (19b)$$

and

$$E\{\ln \bar{w}\} \approx \ln\omega - \tfrac{1}{2}\ln V_S + \tfrac{1}{2}V - V_\epsilon/4VV_S. \qquad (20c)$$

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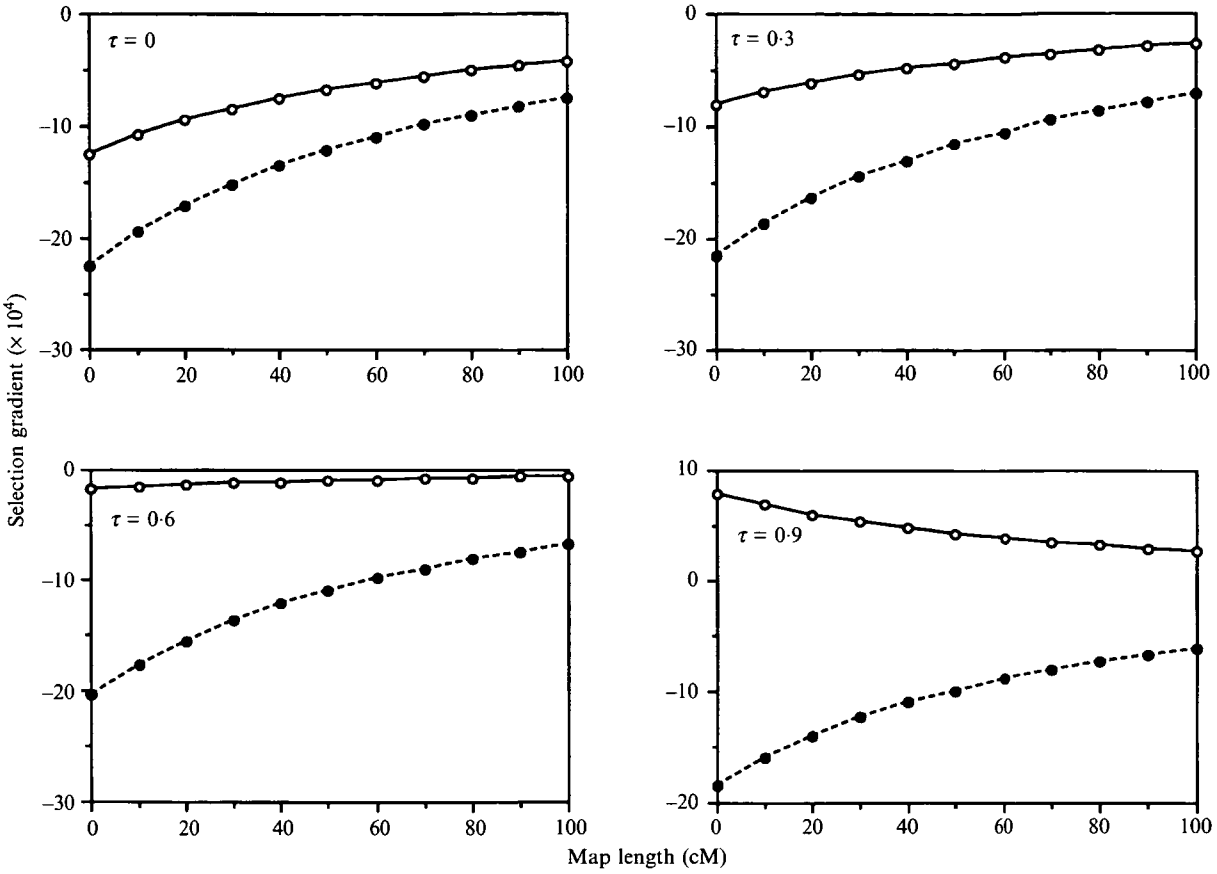

Fig. 5. The selection gradient ($\times 10^4$) on the proportional effect ϵ on map length of a modifier of recombination, expressed as a function of map length, in the case of an unlinked modifier and a single chromosome carrying the trait loci [see equation (26)]. The case when $\psi = 0{\cdot}5$ ($\sigma_\theta/\sigma_G = 3{\cdot}16$) (○) and when $\psi = 0{\cdot}1$ ($\sigma_\theta/\sigma_G = 1{\cdot}41$) (●). Other parameters are as in Fig. 2.

This implies that increased genetic variance is favoured when

$$V_e/2V_S > V^2, \tag{21b}$$

which is a much lighter condition than (21*a*), with plausible parameter values.

(ii) *Selection on a modifier of recombination*

The conditions for increase in frequency of a rare modifier of recombination in the case of a randomly varying environment can be analysed in a similar way to Section 2(iii) above. For the reason given in Section (iv) of the Appendix, only the case of a modifier that is unlinked to the trait loci can be analysed easily. As before, the focus is on the haploid case. The equations for the effect of the modifier on covariances and variance [equations (A 5–9)] are unchanged. In order to calculate the effect of the modifier on the expectation of log mean fitness, $\delta E\{\ln \bar{w}\}$, we note that (ignoring second-order terms in the δs), equation (16) yields

$$E\{\delta \ln \bar{w}\} \approx \frac{1}{2V_S}\delta V E\{(\bar{z}_n-\theta_n)^2\} - 2E\{\delta\bar{z}_n(\bar{z}-\theta_n)]\} - \frac{\delta V}{2}. \tag{22}$$

In order to evaluate this, we need to determine $E\{\delta\bar{z}_n(\bar{z}_n-\theta_n)\}$. The details are given in section (iv) of the Appendix. With small V and $\tau < 1-O(V)$, equations (16) and (A 16) yield the approximate relation

$$E\{\delta \ln \bar{w}\} \approx \frac{\delta V}{2}\left(\frac{\psi(1+\frac{1}{2}\tau)}{1-\frac{1}{2}\tau}-1\right). \tag{23}$$

As before, the value of δV is given by equation (A 9), and has the same sign as the effect of the modifier on recombination, if this is in the same direction for all affected loci. There will then be a disadvantage to a modifier that decreases recombination if

$$\psi\frac{1+\frac{1}{2}\tau}{1-\frac{1}{2}\tau} > 1. \tag{24}$$

It is easily seen that the threshold value of ψ required to satisfy this relation decreases with increasing τ. With $\tau = -1$, a freely recombining population will be immune to invasion by a modifier that reduces recombination if $\psi > 3$; with $\tau = 0$ $\psi > 1$ is required, whereas $\psi > 0{\cdot}33$ is required if τ is close to 1. It is remarkable that this condition is less stringent than the population mean fitness criterion derived above when the autocorrelation is sufficiently low. This suggests that under these circumstances a loosely-

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linked modifier is more likely to gain a selective advantage than an absolutely-linked one, for which the mean fitness criterion is more appropriate. The free recombination criterion becomes more stringent than the mean fitness criterion when $\tau > 0{\cdot}562$.

The strength of selection on the modifier can be calculated by similar methods to those used in Section 3(v). With free recombination between the modifier and the trait loci, and with J chromosomes of map length $l \gg V$, the analogue of equation (15*b*) is

$$s \approx \frac{\epsilon V^2(l+2\ln[\frac{1}{2}(3-e^{-2l})])}{3J(e^l-1)}\left(\frac{\psi(1+\frac{1}{2}\tau)}{1-\frac{1}{2}\tau}-1\right). \tag{26}$$

Some numerical results are shown in Fig. 5. It is seen that, with the parameter sets used here, the autocorrelation must be very high for recombination to be favoured.

4. Nor-optimal selection with a cyclically fluctuating optimum

(i) *Relation of population mean fitness to genetic variance*

A cyclically-varying optimum provides a selective scheme that seems intuitively to be intermediate between the cases of a steadily moving and a randomly fluctuating optimum considered above. A simple model of such a scheme is to write

$$\theta_n = A\cos\left(\frac{2\pi n}{L}\right), \tag{27}$$

where L is the period of the environmental cycle, and A is the amplitude of the oscillation in the optimum. A more general representation of a cyclically varying environment can be obtained from a Fourier series of cosine functions of the form of equation (27); this will not be pursued further here.

Substituting this into equation (17), we obtain the asymptotic result

$$\bar{z}_n \approx kA\sum_{j=0}^{n}(1-k)^{j-1}\cos\left(\frac{2\pi j}{L}\right). \tag{28}$$

If the period of the cycle is sufficiently long relative to the amplitude, then this expression can be approximated by an integral:

$$\bar{z}_N \approx VA\,e^{-(n-1)V}\int_0^{n-1} e^{Vx}\cos\left(\frac{2\pi x}{L}\right)dx$$

$$\approx \frac{2\pi ALV\sin(2\pi(n-1)/L)}{V^2L^2+4\pi^2}. \tag{29}$$

The asymptotic value of $E\{(\bar{z}-\theta)^2\}$ in equation (16) is approximated by the integral of $(\bar{z}-\theta)^2$ over one cycle using equation (29), and dividing by L. For small V, substitution into equation (16) yields the approximation:

$$E\{\ln\bar{w}\} \approx \ln\omega - \tfrac{1}{2}V_S - \tfrac{1}{2}V - \frac{\xi}{4}\left(1 - V + \frac{4\pi^2V^2L^2}{(V^2L^2+4\pi^2)^2}\right), \tag{30}$$

where $\xi = A^2/V_S$, and is the analogue of ϕ and ψ in the previous cases.

For small VL and for very large VL, the condition for expected log fitness to increase with V is $\xi > 2$, which is similar to the condition on ψ in the case of a randomly varying environment with a zero autocorrelation. Since the additional load imposed by directional selection is approximately given by $\frac{1}{4}\xi$ in equation (30), this implies that a very high load is required to confer an advantage to increased genetic variance in this case. Equation (30) also suggests that the period of the cycle has only a small influence on the condition for a mean fitness advantage to increased variance.

The magnitudes of the directional selection loads for populations with different genetic variances, and the expected asymptotic log mean fitness advantages of increased variances, are shown in Table 1. The values are calculated using both the exact equations (16) and (28), and the approximate expression (30). The exact results are obtained by iterating the equations for a minimum of 1000 generations, verifying that the changes in mean have reached a steady state, and then taking the means of log population fitnesses over an entire cycle.

It can be seen from the table that, although the approximate equation is in quite close agreement with the exact values for directional selection loads (except for very short cycles), the effect of changing the genetic variance is usually so small compared with the error in the approximation that the magnitude and direction of the net effect on average log mean fitness are usually not well predicted. As expected, the agreement is best for the lowest amplitude of the environmental cycle that was studied ($\xi = 0{\cdot}31$), but this case does not yield an advantage for increased variance. The exact results suggest that the criterion $\xi < 2$ for an advantage to increased variance is too stringent, and that $\xi > 1$ is a closer approximation. In all cases, the advantage or disadvantage of increased variance is small, even when the directional selection load is very large (e.g. with $\xi = 2{\cdot}81$). As predicted by the approximate formulae, there is little sensitivity to the period length, except for very small or very large cycles. The exact results indicate that the advantage of increased variance tends to increase when the period is very long, presumably because this case converges on the more favourable case of a steadily moving optimum.

(ii) *Selection on a modifier or recombination*

Selection on a recombination modifier can be studied by methods similar to those used previously. Unfortunately, the poor agreement between the analytical approximation and the numerical results in the mean fitness analyses described above suggests that it would

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Table 1. *Effect of genetic variance on mean fitness in a cyclically varying environment*

	Directional selection loads				Advantage of increased V_G		
	High V_G		Low V_G		Mean fitness advantage		Modifier
L	Exact	Approx.	Exact	Approx.	Exact	Approx.	advantage*
			$A = 2{\cdot}5$ ($\xi = 0{\cdot}31$)				
2	0·150	0·077	0·155	0·078	−0.003	−0.006	−0.242
5	0·075	0·077	0·077	0·078	−0.005	−0.006	−0.353
10	0·075	0·077	0·077	0·078	−0.005	−0.006	−0.318
50	0·075	0·077	0·077	0·078	−0.005	−0.006	−0.279
100	0·068	0·083	0·077	0·078	0.012	−0.012	−0.292
200	0·054	0·092	0·077	0·078	0·014	−0.020	−0.336
			$A = 3{\cdot}25$ ($\xi = 0{\cdot}53$)				
2	0·254	0·129	0·261	0·131	+0·000	−0.006	−0.064
5	0·127	0·129	0·131	0·131	−0.004	−0.006	−0.251
10	0·127	0·131	0·130	0·131	−0.003	−0.006	−0.193
50	0·124	0·133	0·131	0·132	−0.001	−0.008	−0.126
100	0·115	0·140	0·130	0·132	0·007	−0.016	−0.148
200	0·091	0·156	0·128	0·135	0·029	−0.029	−0.222
			$A = 5{\cdot}0$ ($\xi = 1{\cdot}25$)				
2	0·600	0·306	0·619	0·311	0·011	−0.003	0.532
5	0·302	0·306	0·309	0·311	0·002	−0.003	0.089
10	0·300	0·307	0·309	0·311	0.002	−0.003	0·227
50	0·293	0·314	0·309	0·311	0·009	−0.010	0·384
100	0·273	0·332	0·307	0·312	0·027	−0.026	0·333
200	0·215	0·369	0·302	0·318	0·079	−0.058	0·215
			$A = 7{\cdot}5$ ($\xi = 2{\cdot}81$)				
2	1·351	1·392	0·689	0·700	0·034	0·003	1·822
5	0·675	0·689	0·696	0·700	0.013	0·003	0·675
10	0·675	0·690	0·696	0·700	0·014	0·002	1·136
50	0·659	0·706	0·695	0·701	0·028	−0.012	1·489
100	0·615	0·747	0·692	0·704	0·070	−0.050	0·615
200	0·484	0·830	0·830	0·716	0·188	−0·122	0·484

In this case, V_S was set to 20. V_G was 0·4 for the high variance (sexual) population, and 0·1 for the low variance (asexual) population. The loads and mean fitness advantage are calculated in terms of the appropriate terms in the expected log mean fitnesses of the populations.

* The advantage to the modifier is that for an unlinked modifier increasing recombination, expressed as a proportion of its effect on variance (scaled relative to V_S), δV.

be unprofitable to obtain such approximations for a modifier in this case. Instead, the case of an unlinked modifier was studied by iteration of equations (28), (29) and (A 12*a*), to determine the steady-state trajectories of $\bar{z}$ and $\delta\bar{z}$. Equation (22) was then used to calculate the mean value of $\delta \ln \bar{w}$ relative to δV. The sign of this quantity gives the criterion for increase in the frequency of a rare modifier increasing recombination. No assumptions about the linkage relations among the trait loci are needed.

The last column of Table 1 shows the result of these calculations. The conditions for an advantage to a modifier increasing recombination are similar to those for an increase in the average mean fitness. The magnitude of selection on the modifier is considerably affected by period, but its sign is never changed. Since δV is independent of environmental variation in the selection regime, this implies that the selection coefficient on the modifier varies in parallel. Selection in favour of increased recombination is strongest when the cycle length is very short or fairly long, but diminishes again for very long periods.

5. Strong directional selection

(i) *Preliminary considerations*

As mentioned in Section 1, there is evidence that artificial selection on quantitative traits can lead to a correlated increase in the frequency of genetic recombination. It is therefore of interest to compare the consequences of strong directional selection with the results obtained up to now, which have assumed relatively weak selection. Since the experiments have

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mostly been carried out on *Drosophila*, diploid inheritance will be assumed here.

The standard model of truncation selection on a metrical trait is used, which assumes that only the proportion p of the population which falls above a threshold value T contributes to the next generation (Falconer, 1989, chap. 11). Assuming a normal distribution of the trait and large population size, the selection intensity i (the difference between the mean of the selected parents and the population mean before selection, expressed as a standardized normal deviate) is equal to $\exp(-\frac{1}{2}z_t^2)/(p\sqrt{2\pi})$. Here, $z_t = (T-\bar{z})/\sigma_p$ is the standardized normal deviate corresponding to T, where σ_P is the phenotypic standard deviation (Falconer, 1989, chap. 11). The change in mean additive genetic value due to one generation of selection is $ih^2\sigma_P$, where h^2 is the heritability, V_G/V_P. The corresponding change in additive genetic variance is

$$\Delta = -i(i-1)\,V_G h^2 \tag{31}$$

(Bulmer, 1985, chap. 9).

The genetic variance in a population subject to artificial selection of this kind rapidly equilibrates to the value given by equation (9.47) of Bulmer (1985). This equilibrium value depends only on the parameters just defined and on the harmonic mean recombination fraction r_H between a random pair of trait loci [cf. equation (A 3*c*)]. If r_H is small, as in the case of organisms such as *Drosophila* with small numbers of chromosomes, the equilibrium genetic variance may be considerably smaller than that in the starting population. It will accordingly be used for the initial value of V_G for the population into which the modifier is introduced.

(ii) *Selection on a recombination modifier with truncation selection*

Using the argument that leads to equation (A 19) for the case of nor-optimal selection and diploidy, we obtain the following recursion relation for the deviation of the additive genetic value at the ith locus in the modifier population from the value for the general population

$$\delta Z_i' = (1-\rho_i)\,\delta Z_i + \frac{\delta(ih^2\sigma_P)}{2m}. \tag{32a}$$

This yields the equilibrium relation

$$\delta\bar{z} \approx \frac{i[1-\frac{1}{2}h^2]\,\delta V_G + V_G\,\delta i}{2\sigma_P\rho_H}. \tag{32b}$$

Using the relation between i and z_t [see Section 5(i) above], we can express δi in terms of the more fundamental variables $\delta\bar{z}$ and δV_G as

$$\delta i \approx \frac{iz_t}{\sigma_P}\left(\delta\bar{z} + \frac{z_t\,\delta V_G}{2\sigma_P}\right). \tag{33b}$$

A further expression in $\delta\bar{z}$ and δV_G can be obtained from the equations for the covariances in the modifier population, equation (A 18). Write E for the expectation over all loci pairs of the sum of the multiplicands of $\delta\Delta$ in equation (A 18), and δE for the expectation of the sum of the multiplicands of Δ. Using equation (31) to evaluate these multiplicands in terms of $\delta\bar{z}$ and δV_G, we obtain the relation

$$(1+aE)\,\delta V_G + bE\delta\bar{z} = -\Delta\delta E, \tag{34}$$

where

$$a = \frac{ih^2}{V_P}((V_E+V_P)(i-z_t)+\tfrac{1}{2}V_G z_t(1+z_t[2i-z_t]))$$

and

$$b = ih^4(1+z_t[2i-z_t]).$$

Combining these relations with equations (33*a*, *b*), we obtain

$$\delta\bar{z} \approx \frac{-c\Delta\delta E}{(1-d)(1+aE)+bcE}, \tag{35a}$$

$$\delta V_G \approx \frac{(1-d)\,\delta\bar{z}}{c}, \tag{35b}$$

where

$$c = \frac{i}{2\sigma_P\rho_H}\left(1+\frac{z_t^2}{2V_P}-\tfrac{1}{2}h^2\right)$$

and

$$d = \frac{iz_t}{2V_P\sigma_P\rho_H}.$$

The selection coefficient on the modifier is given approximately by $-i\delta z_t$ (Falconer, 1989, p. 202), and so we obtain

$$s \approx \frac{i}{\sigma_P}\left(\delta\bar{z} + \frac{z_t\,\delta V_G}{2\sigma_P}\right). \tag{36}$$

These equations enable the selection coefficient on a rare modifier of the rate of recombination to be calculated, providing that the recombination terms E and δE are specified. For the numerical examples considered here, these have been calculated by a modification of equations (26) and (27) of Charlesworth (1990). The modification involves dropping terms involving the selection coefficients on the underlying loci [see Appendix, Section (ii), for a justification of this procedure]. The results obtained in this way should be quantitatively accurate, unless the map length of each chromosome is small. The model assumes that there are J chromosomes, each of map length l, with m loci distributed randomly across the chromosomes. Recombination occurs at equal rates in the two sexes, and there is no interference. The modifier is assumed to affect the map length of each chromosome by an amount ϵl.

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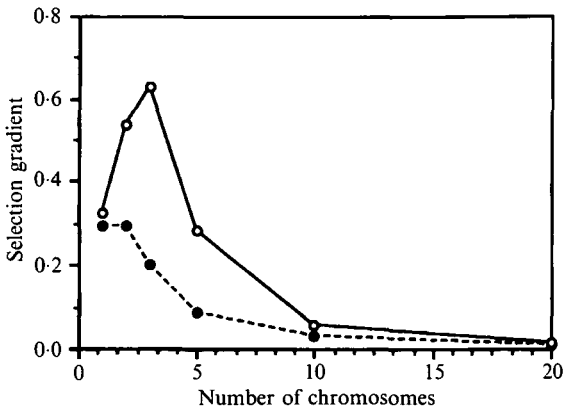

Fig. 6. The selection gradient on the proportional effect ϵ on map length of a modifier of recombination in the case of strong truncation selection (proportion selected 10%, selection intensity 1·8). The modifier is assumed to be located in the middle of one of the chromosomes, and to affect the map length of each chromosome equally. An initial map length of 50 cM for each chromosome (○) and an initial map length of 100 cM (●) are assumed.

Some results are shown in Fig. 6. It can be seen that, especially for the shorter map length, strong truncation selection leads to a large selection pressure in favour of a modifier increasing recombination. The case of an organism like *Drosophila* that has crossing over in only one sex can also be modelled by this method, by writing the frequency of recombination between a pair of loci separated by a given map distance as one-half of the value given by the Haldane mapping function. This yields results that are almost identical to those obtained with equal rates of crossing over in both sexes, but with chromosomes half the map length. The results in fig. 6 with three chromosomes and a map length of 0·5 should thus approximate what is expected for *Drosophila*. The strength of selection on a modifier is clearly very dependent on the number of chromosomes, and falls off rapidly as this increases above five. The peak at three chromosomes for the case of a map length of 50 cM for each chromosome may be artefactual, as the equations for change in covariance become unreliable for low recombination rates and strong selection.

6. Discussion

The results presented above show clearly that a sufficiently strong, sustained pressure of directional selection on a quantitative trait can favour sexual reproduction and genetic recombination, in agreement with results obtained previously, primarily by computer modelling (Maynard Smith, 1980, 1988; Bergman & Feldman, 1990, 1992; Korol *et al.* 1990; Crow, 1992). This does not necessarily imply that this process has been an important factor in the evolution of sex and recombination. The conditions under which it is most likely to operate are reviewed below, and theoretical and empirical evidence relevant to its plausibility is discussed.

(i) *Effects of the selection parameters on selection for sex and recombination*

Inspection of the equations and figures presented above shows that, for each model of nor-optimal selection, there is a threshold level of environmental variability (measured by $|\Delta\theta|$ with a steadily moving optimum, V_θ or V_ϵ with a randomly varying optimum, and A for a cyclical optimum) which must be exceeded for there to be a selective advantage to sexual over asexual reproduction, or for selection for non-zero rates of genetic recombination. If the appropriate threshold is not crossed, then increased genetic variance in the trait under selection leads to a decrease in long-term average population mean fitness, or to selection against modifiers that increase recombination. If there is an ESS mean recombination frequency, its value also increases with the degree of environmental variability.

Of course, it is not necessarily the case that even strong directional selection creates a selection pressure in favour of increased genetic variance (Maynard Smith, 1988; Bergman & Feldman, 1990, 1992). A requirement for this is that selection creates negative linkage disequilibrium (or Hardy–Weinberg disequilibrium, in the case of a non-recombining but sexual population), by reducing the variance of the trait. In the case when fitness is a log-linear function of phenotype, there is no change in variance, and hence no advantage to recombination of segregation (Charlesworth, 1990). More generally, Shnol & Kondrashov (1993) have shown that selection reduces the variance in traits for which the second derivative of the logarithm of fitness with respect to phenotypic value is always negative. The opposite is true if the second derivative is always positive. Only in the former case will there be an advantage to increased recombination under directional selection, using the models developed here. Similar results were obtained by Felsenstein (1965) for a two-locus model.

Under the model of maintenance of variation by mutation, equation (8*a*) implies that the level of genetic variance for a freely-recombining sexual population is proportional to the measure of the inverse of the strength of selection, V_S. Hence, the standardized genetic variance $V = V_G/V_S$ which appears in the selection equations is nearly independent of V_S. It follows from this, and from the fact that the terms involving the various measures of environmental variability are all divided by V_S, that (for a given level of environmental variability) the strength of selection on the quantitative trait has the same relation to selection for sex or recombination as the level of environmental variability. In the case of a randomly varying optimum, selection for sex or recombination is favoured by large values of the

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autocorrelation in optimum, τ. With a cyclical environment, the period has little influence on the direction of selection on sex or recombination, but the magnitude of any advantage seems to be greatest for very short or very long periods (see Table 1).

As noted previously, the degree of environmental variability and the strength of selection on the trait combine to create a genetic load due to directional selection, which bears a similar relation to the nature of selection for sex or recombination as either of the other two parameters considered in isolation. The magnitude of this directional load provides a good guide to the relative plausibility of the various models, as it is unlikely that a selective scenario that requires an extremely high directional load in order to create an advantage to sex or recombination will be compatible with the continued survival of the population (cf. Crow, 1970). On these grounds, the cyclical optimum model and the random optimum model with low autocorrelation appear to be implausible, as they both require extremely high loads in a sexual population to provide even a modest advantage to sex or recombination (see Fig. 4 and Table 1). While the random optimum model with an autocorrelation of 0·9 or so can certainly provide a significant mean fitness advantage to sex, and a strong advantage to non-zero recombination, it cannot generate a sufficient advantage to overcome the two-fold reproductive cost of sex (Maynard Smith, 1978) without an extremely high directional load. The same applies to the case of a cyclical environment. Only the model of a steadily moving optimum [Section 2(ii)] can create such a large advantage to sex with a moderate directional load in the sexual population.

The load created by directional selection is closely related to the time average of the variance in fitness (cf. Crow, 1970, p. 172), as may be seen as follows. From the nor-optimal selection formula and equation (4), the variance of fitness (scaled relative to the square of mean fitness) in a given generation is

$$\frac{V_w}{\bar{w}^2} = \frac{(V_G+V_S)}{\sqrt{[\omega^2(2V_G+2V_E+\omega^2)]}} \times \left\{ \frac{\exp-\left[\frac{(\bar{z}-\theta)^2}{2V_G+2V_E+\omega^2}\right]}{\exp-\left[\frac{(\bar{z}-\theta)^2}{V_G+V_S}\right]} \right\} - 1. \qquad (37a)$$

Provided that $\omega^2 \gg V_G+V_E$, as we have previously assumed, the component of the scaled variance in fitness attributable to the deviation of the mean from the optimum can be can be approximated by

$$V_w^* \approx \exp\left(\frac{V_G+V_E}{V_S}\right) - 1. \qquad (37b)$$

Provided that the expected squared deviation of population mean from the optimum is small, using equation (19*b*) we find that the expectation over time of the scaled genetic component of the variance in fitness is given by

$$E\{V_{Gw}^*\} \approx \frac{VE\{(\bar{z}-\theta)^2\}}{V_S}. \qquad (38)$$

From equations (4), (5) and (20), the directional load is $1/(2V)$ times the right-hand side of this equation. With $V = 1/20$, a directional load that is required to select for sex or increased recombination thus corresponds to a scaled genetic variance in fitness of one-tenth the size of the load. Houle (1992) has reviewed data on fitness components in a variety of different species, usually measured under laboratory conditions. Traits such as fecundity and longevity often show scaled genetic variances as high as 0·2 or so (Houle's Table 1 and Fig. 1), but values that approach 1 are rare. If variance in fitness were due solely to directional selection, such values would easily be compatible with a selective advantage to increased genetic variance, or even to sexual reproduction, with a steadily moving optimum. They are less easy to reconcile with an important role for fluctuating environments, especially as the proportion of the variance in fitness that is due to directional selection is unknown, and seems likely to be small compared with other sources such as mutation (Charlesworth, 1987).

It is fairly easy to see why the case of a steadily moving optimum is most favourable to sex and recombination. Any advantage of an increase in the additive genetic variance must accrue from the fact that it enables the mean of the trait under selection to change more rapidly under directional selection (Mather, 1943; Maynard Smith, 1988). With a steadily moving optimum, there are no reverses to the direction of selection; hence, an increase in additive genetic variance always allows the population mean to approach more closely to the current optimum, thereby reducing the load due to directional selection [equations (3) and (5)]. With a randomly fluctuating optimum, there will be frequent reversals in the direction of selection. Since there is always a lag between the optimum and the mean, an increased speed of response of selection can lead to the change in population mean between one generation and the next being in the opposite direction to that needed to bring the mean closer to the optimum in the succeeding generation. This is particularly true if there is a low autocorrection between the successive values of the optimum; the overall consequence is that there is a considerably weaker advantage to increased genetic variance in a random environment than with a steadily moving optimum.

But equation (21*b*) shows that an optimum that undergoes a random walk (which is equivalent to an autocorrelation of one) can provide an advantage to increased variance under lighter conditions. If $V_S = 20V_G$, the variance in optimum V_ϵ need only be about 0·1 times the additive genetic variance for increased variance to be advantageous. The corresponding

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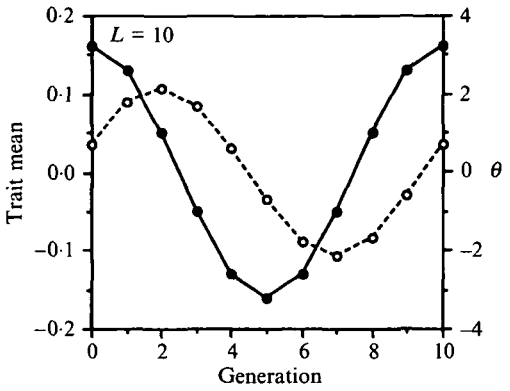

Fig. 7. The cycle of changes of optimum (●) and mean (○) for a population subject to a cyclical environment of period 10. The amplitude A is 3·5. Other parameters are as in Table 1.

directional load is $V_e/4V_G = 0{\cdot}025$. This is approximately one-tenth the value required for a two-fold advantage to sex under this model, assuming that the asexual population has one-quarter the genetic variance of the sexual population. This type of random environmental fluctuation is thus much more favourable to the maintenance of sex or recombination than a stationary process. It is straightforward to show that a combination of a stationary Markov process or random walk and a steadily moving optimum gives a directional load that is simply the sum of the values contributed by each process on its own [cf. equations (5) and (20*c*)]. A slowly moving optimum combined with a random walk about the trend could thus be a possible source of selection for increased recombination or sexual reproduction.

A somewhat unexpected result is that a cyclical optimum behaves like a randomly fluctuating optimum with a low autocorrelation. From equation (30) and Table 1, it may be seen that an advantage to sex or recombination exists only if the directional selection load exceeds approximately one-quarter; this corresponds to the critical value in the case of a random environment with an autocorrelation of one-third. The reason for this relatively weak ability of cyclical selection to promote sex and recombination appears to be that the lag in adjusting the population mean to the optimum causes the cycle of population means to be out of phase by 180° with the cycle of the optimum [see equation (29) and Fig. 7]. Hence, there is always a substantial deviation of the mean from the optimum, even with a large genetic variance.

A. S. Kondrashov (personal communication) has suggested that the degree of fluctuation in the character mean generated by variable selection provides a useful means of assessing the plausibility of these models as an explanation of the evolution of sex and recombination. Statistical analyses of data on evolving fossil lineages suggest that average rates of change of mean are generally very small over long periods of time; a change of population mean of one phenotypic standard deviation every million years represents exceptionally fast evolution (Simpson, 1953; Charlesworth, 1984*a*). While there is frequently evidence for statistically significant variation in rate around the average rate of change, the magnitude of these fluctuations appears usually to be several orders of magnitude smaller than the within-population variance of the character (Charlesworth, 1984*b*), and the associated directional selection loads are correspondingly small (of the order of 10^{-4} at most). Hence, there are severe difficulties in accepting the plausibility of any model which requires geologically fast changes in trait mean.

Kondrashov's model of fluctuating selection assumes truncation selection of individuals who depart too far from the optimal value; he showed that excursions in the optimum and mean of the order of one phenotypic standard deviation are required for there to be an advantage of sex or recombination. The model of a steadily moving optimum of Section 2 requires that the rate of change of the optimum (and hence the mean) be approximately 4% of the genetic standard deviation per generation; even if the direction of selection were to reverse itself every 100 generations or so, there would be excursions of the mean at least of the order of a phenotypic standard deviation. Similarly, with randomly fluctuating selection the variance in trait mean over time can be shown by the methods of Section 3 to be

$$V_{\bar{z}} \approx \frac{V_\theta V(1+\tau)}{2(1-\tau)}. \tag{38}$$

Comparison of this with equation (21) implies that sexual reproduction is favoured if $\sigma_{\bar{z}} > \sigma_G$. This is quite similar to Kondrashov's result for truncation selection.

With the random walk model, the variance in mean increases with time, and so a comparable result cannot be written down. However, it is easily seen from equations (2) and (19*b*) that the variance of the change in mean per generation, $V_{\Delta\bar{z}}$, is approximately equal to $VV_e/2$. From the condition for an advantage to increased variance discussed above, the variance in change in mean must be approximately $0{\cdot}0025V_G$, with $V_S = 20V_G$. Again, although small, this seems much larger than is indicated by the fossil data (Charlesworth, 1984*b*).

The detailed numerical results for the cases of a cyclically varying environment shown in Table 1 indicate that the excursion of the mean over the entire cycle must be at least 10% of the environmental variance in the trait, to generate an advantage to sex and recombination (the case with an amplitude of 5 in Table 1). This is a more modest condition than the others discussed here, but the associated directional selection load is very high.

Finally, the possibility that selection on several quantitative traits may lighten the conditions for an advantage to sex and recombination will be considered

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briefly. The most extreme and simplest case is to assume that multiple, independent quantitative traits are each subject to independent selection regimes. If this is the case, then log fitnesses can be combined additively, and the contributions of each trait to log mean fitness, etc. can be summed. It is easily seen from equations such as (6), (20) and (30) that, while the magnitude of the effect of a given change in variance in each character increases with the number of characters, the conditions for an increase in mean fitness with an increase in variance are unchanged from those derived earlier. There will be a corresponding increase in the directional load. It thus seems that selection on multiple traits does little to alleviate the difficulties discussed above.

(ii) *The intensity of selection on recombination rate*

In addition to the effects of strength of selection on the trait, level of environmental variability and autocorrelation discussed above, the intensity of selection for modifiers that increase the rate of recombination (assuming that the threshold values of the other parameters are exceeded) is mainly affected by the amount of recombination in the initial population, as measured by the number of chromosomes and the map length of each chromosome. As has been found in other models of selection for increased recombination (Charlesworth, 1990), there can be quite strong selection for increasing recombination away from zero (see Fig. 2). However, the intensity of selection is greatly reduced when there are several chromosomes, and when the map length of each is appreciable (see Figs 3, 5). The effect of a change in map length of each chromosome on the average amount of recombination in the genome is inversely proportional to the number of chromosomes (cf. equation (15*b*)], and so almost any selective force affecting map length is bound to diminish as the number of chromosomes is increased. Thus, these results do not necessarily imply that the importance of varying environments relative to other forces acting to increase or decrease recombination falls off with map length and chromosome number.

In the case of a steadily varying environment, selection acting to increase recombination away from zero always operates under lighter conditions than are needed to maintain free recombination [equation (9)], thus implying that an ESS intermediate level of recombination can sometimes be maintained. With a randomly varying environment, and a low autocorrelation ($\tau < 0{\cdot}562$), the condition for mean population fitness to be increased by increased genetic variance is more stringent than the condition for an advantage to a modifier that is unlinked to the trait loci and that increases recombination. Since the mean fitness criterion is equivalent to that for selection for a completely recessive modifier increasing recombination in an initially non-recombining population (Felsenstein, 1974), and since this case implies complete linkage between the modifier and the trait loci, this suggests that there are circumstances in which a loosely-linked, dominant modifier may gain an advantage when a tightly-linked recessive one would be eliminated. Unfortunately, it is not possible with the methods used here to tell whether it is dominance or linkage that is causing the difference, since the modifier analysis assumes some degree of dominance and free recombination [see Appendix, Section (ii)]. On the basis of computations of populations in a cyclically varying environment, Korol *et al.* (1990) report that close linkage of the modifier can sometimes lead to selection against recombination under conditions when a more loosely linked modifier can spread, which agrees with this finding.

Finally, it is worth noting that the present results show clearly that stabilizing selection in a near-constant environment selects quite strongly for a reduction in recombination to zero; the magnitude of the selection coefficients on modifiers affecting the recombination rate in a constant environment can be calculated from equations (13) and (15) with $\phi = 0$. With a 'standard' value of V of 0·05 [see Section 2(i)], equation (15) shows that a modifier reducing the map length of each chromosome by ϵl in a population with J chromosomes experiences a selective advantage of $0{\cdot}0025\epsilon/J$. Thus, despite the very low levels of linkage disequilibrium maintained among the underlying trait loci by stabilizing selection (Bulmer, 1985, p. 159), the cumulative effect is quite significant, and could contribute to the pressure for 'congealing of the genome' (Maynard Smith, 1978, chap. 5; Feldman, Christiansen & Brooks, 1980; Feldman & Liberman, 1986; Bergman & Feldman, 1990). This effect of stabilizing selection contrasts with the selection pressure for increased recombination that arises from synergistic fitness interactions among unconditionally deleterious alleles maintained by mutation–selection balance (Feldman, Christiansen & Brooks, 1980; Kondrashov, 1988; Charlesworth, 1990), reflecting the sustained pressure of directional selection against deleterious alleles in this case. The question of whether or not stabilizing selection on a quantitative trait is a by-product of the deleterious fitness effects of mutations affecting the trait (Barton, 1990; Kondrashov & Turelli, 1992) is thus of great significance for the evolution of sex and recombination.

(iii) *Empirical evidence on the importance of variable environments in the evolution of sex and recombination*

These theoretical considerations clearly cannot provide a conclusive answer to the question of whether variable selection on quantitative traits has been an important factor in the evolution of sex and recombination. At present, there is little empirical evidence that bears critically on this question. Com-

parisons of recombination rates or chiasma frequencies between species are not very helpful in discriminating between alternative theories of the evolution of recombination rates (Charlesworth, 1989). There are also difficulties in using comparative data on the eco-correlates of the occurrence of asexuality (Charlesworth, 1989). While theory implies that high autocorrelations and intense directional selection promote increased recombination, which might suggest possible tests of the models, we lack knowledge of these for natural populations. There does seem to be clear evidence that artificial directional selection in *Drosophila* is frequently associated with a correlated increase in recombination rate (Flexon & Rodell, 1982; Korol *et al.* 1990; Gorodetskii, Zhuchenko & Korol, 1991). The theoretical analyses of the effects of intense truncation selection in Section 5 show that it can indeed generate selection for increased recombination that is strong enough to account for significant increases in recombination rates over the time-scale of selection experiments. This provides some modest empirical support for the models considered here. Comparisons of recombination rates between natural populations that have been subjected to varying levels of directional selection in the recent past might help to provide tests of the models.

This work was supported in part by NSF grant BSR-8817976. I thank Deborah Charlesworth, Alex Kondrashov and Martin Morgan for helpful discussions, and A. Burt and an anonymous reviewer for commenting on the manuscript. Austin Burt suggested the analyses of fitness variance and of the case of a random walk in fitness optimum. Michael Lynch and Marc Feldman kindly provided me with copies of their recent papers.

Appendix

(i) *Change in mean caused by a modifier of recombination in a haploid population with a constant rate of movement of the optimum*

The modifier allele is assumed to be rare, and so a haploid genome carrying the modifier experiences recombination only with a haploid genome from the general population. Selection is assumed to precede recombination within each generation. It is assumed that the trait is controlled by m interchangeable loci. The change in mean additive genetic value caused by selection within a generation can thus be assumed to the same at each locus, and equal to the overall change in mean divided by m. Consider a locus i that recombines with the modifier at rate ρ_i. Let the additive genetic value with respect to this locus in a given generation for the general population be Z_i before selection, Z_i^* after selection, and Z_i' in the next generation. The deviations of the values of Z_i and other variables for the modifier population from their values for the general population are denoted by the prefix δ. We thus obtain

$$Z_i'+\delta Z_i' = (1-\rho_i)(Z_i^*+\delta Z_i^*)+\rho_i Z_i^*. \qquad (A\ 1a)$$

Using equation (1), and neglecting second-order terms in the differences in mean and variance between the modifier and general populations, it is easily seen that, for generation n, with n taken sufficiently large that the effect of the modifier on variance has equilibrated (see Section (ii) below], equation (A 1*a*) reduces to

$$Z_i'+\delta Z_i' \approx Z_i+(1-\rho_i)\,\delta Z_i + \frac{1-\rho_i}{m}\times(\delta k(\theta_{n-1}-\bar{z}_{n-1})-k\delta\bar{z}_{n-1})). \qquad (A\ 1b)$$

Using equation (1), we have

$$\delta Z_i' \approx (1-\rho_i)\,\delta Z_i+\frac{1-\rho_i}{m}(-k\delta\bar{z}_{n-1}+\delta k(\theta_{n-1}-\bar{z}_{n-1})). \qquad (A\ 2a)$$

Using equation (3), this gives the equilibrium relation

$$m\rho_i\,\delta Z_i \approx (1-\rho_i)\left(-k\delta\bar{z}+\frac{\Delta\theta\delta V}{k}\right). \qquad (A\ 2b)$$

For small k ($V \ll 1$), we have $k \approx V$. In this case, and summing over all loci, we obtain the following approximation for the equilibrium value of $\delta\bar{z}$

$$\delta\bar{z} \approx \frac{(1-\rho_H)\,\Delta\theta\delta V}{(\rho_H+[1-\rho_H]\,V)\,V}, \qquad (A\ 2c)$$

where ρ_H is the harmonic mean of the ρ_i over all loci.

(ii) *Change in variance caused by a modifier of recombination in a haploid population*

Relations which yield δV can be obtained as follows, using the approach of Bulmer (1985, p. 150). In contrast to the case of mutation-selection balance studied by Charlesworth (1990), no correction for linkage disequilibrium induced by allele frequency changes is made here. This is because the changes in the selective optimum in general induce variation in the selection coefficients at the trait loci, so that this correction is hard to apply. If the number of trait loci is large, and selection is weak, so that the selection coefficients are small, the corrections for induced linkage disequilibrium are important only for very tight linkage. The expressions derived below should therefore provide good approximations, unless there is very tight linkage for a typical pair of loci.

Let the covariance between loci i and j for the general population in a given generation be C_{ij}; we have $C_L = 2\sum C_{ij}$. The values after selection and in the next generation are indicated in a similar way to that employed for the means. Since the only source of covariance in the haploid model is linkage disequilibrium between loci, the change in variance Δ induced by selection in any one generation is divided equally among the $m(m-1)$ covariance terms in C_L. For the general population, we thus have the recurrence relation

$$C_{ij}' = (1-r_{ij})\left(C_{ij}+\frac{\Delta}{m(m-1)}\right), \qquad (A\ 3a)$$

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where r_{ij} is the recombination frequency between loci i and j for the general population.

This yields the equilibrium expression

$$C_{ij} = \left(\frac{1}{r_{ij}} - 1\right)\frac{\Delta}{m(m-1)}. \quad \text{(A 3}b\text{)}$$

Summing over all loci, we have

$$C_L = \left(\frac{1}{r_H} - 1\right)\Delta, \quad \text{(A 3}c\text{)}$$

where r_H is the harmonic mean recombination frequency between all pairs of loci.

In order to calculate the δC_{ij}, we need to consider two possible locations of the modifier with respect to the pair of trait loci under consideration (Charlesworth, 1990, pp. 218–219). In the first case, the modifier lies outside the pair. This generates the recurrence relationship

$$C'_{ij} + \delta C'_{ij} = (1 - r_{ij} - \delta r_{ij})([1-\rho_i][C^*_{ij} + \delta C^*_{ij}] + \rho_i C^*_{ij}) \quad \text{(A 4)}$$

where δr_{ij} is the effect of the modifier on the frequency of recombination between the two trait loci. Using equations (A 3a, b), and ignoring second-order terms in the δs, we obtain the equilibrium expression

$$\delta C_{ij} \approx \frac{1}{m(m-1)(\rho_i + [1-\rho_i]r_{ij})} \times \left((1-\rho_i - [1-\rho_i]r_{ij})\,\delta\Delta - \frac{\Delta\delta r_{ij}}{r_{ij}}\right). \quad \text{(A 5)}$$

For nor-optimal selection and $V \ll 1$, we have

$$\Delta = -\frac{V_G^2}{V_G + V_S} \approx -V^2 V_S \quad \text{(A 6}a\text{)}$$

and

$$\delta\Delta \approx -\frac{V_G(V_G + 2V_S)}{V_G + V_S}\delta V_G \approx -2V_G\,\delta V. \quad \text{(A 6}b\text{)}$$

In the second case, the modifier lies in between the two trait loci. In this case, we have

$$C'_{ij} + \delta C'_{ij} = (1-\rho_i)(1-\rho_j)(C^*_{ij} + \delta C^*_{ij}) + \rho_i\rho_j C^*_{ij} \quad \text{(A 7)}$$

which yields the equilibrium relation

$$\delta C_{ij} = \frac{1}{m(m-1)(1-[1-\rho_i][1-\rho_j])} \times \left((1-\rho_i)(1-\rho_j)\,\delta\Delta - \frac{\Delta\delta r_{ij}}{r_{ij}}\right). \quad \text{(A 8)}$$

Equations (A 5) and (A 8) determine the value of the deviation of the variance of the modifier population from that for the general population, $\delta V_G = 2\Sigma\delta C_{ij}$. Write E_1 and E_2 for expectations over sets of locus pairs for which the modifier is located outside and between the loci, respectively. Explicit approximate expressions for these can be obtained by methods similar to that used to derive equation (26) of Charlesworth (1990); the details will be omitted here. We have

$$\delta V_G \approx E_1\left\{\frac{1}{\rho_i + [1-\rho_i]r_{ij}} \times \left((1-\rho_i - [1-\rho_i]r_{ij})\,\delta\Delta - \frac{\Delta\delta r_{ij}}{r_{ij}}\right)\right\} + E_2\left\{\frac{1}{1-[1-\rho_i][1-\rho_j]} \times \left((1-\rho_i)(1-\rho_j)\,\delta\Delta - \frac{\Delta\delta r_{ij}}{r_{ij}}\right)\right\}. \quad \text{(A 9)}$$

Using equations (A 6) and the fact that the multiplicands of $\delta\Delta$ in equation (A 9) are positive, it is easily seen that a modifier that has the same direction of effect on all the recombination frequencies r_{ij} that it affects (i.e. such that either $\delta r_{ij} \leqslant 0$ or $\delta r_{ij} \geqslant 0$ for all pairs of loci) will be associated with a change in variance of the same sign as its effect on recombination.

(iii) *Selection on a modifier of recombination in a haploid population with a constant rate of movement of the optimum*

Using equation (4), and assuming that $V \ll 1$, the deviation of the logarithm of the mean fitness of the modifier population is approximated by

$$\delta(\ln\bar{w}) \approx \frac{\delta V}{2}\left(\frac{(\Delta\theta)^2}{V^2 V_S} - 1\right) + \frac{\delta\bar{z}(\Delta\theta)}{VV_S}. \quad \text{(A 10)}$$

Substituting from equation (A 2c), we have

$$\delta(\ln\bar{w}) \approx \frac{\delta V}{2}\left(\frac{\phi}{V^2}\left[\frac{2(1-\rho_H)}{(\rho_H + [1-\rho_H]V)} + 1\right] - 1\right). \quad \text{(A 11}a\text{)}$$

The magnitude of the selection coefficient on the modifier can be obtained by evaluating the expectations in equation (A 9). In general, this will require numerical work. However, using the above result on the relation between the sign of the effect of a modifier on recombination and its effect on variance leads to the conclusion that an increase in genetic variance caused by a modifier that increases the frequency of recombination between all the loci that it affects will be favoured if and only if

$$\frac{2\phi(1-\rho_H)}{\rho_H + [1-\rho_H]V} + \phi > V^2. \quad \text{(A 11}b\text{)}$$

(iv) *Effect of a recombination modifier on fitness in a randomly fluctuating environment*

In order to obtain homogeneous equations in this case, it is necessary to assume that there is either free

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recombination between the modifier and the selected loci in the initial population. This guarantees that the deviations of additive genetic value for the modifier genotype are the same at each locus, and equal to $\delta\bar{z}/m$. From equations (A 1) and (A 2*a*), we find that the asymptotic deviation of the additive genetic value at locus i for the modifier population from that for the general population in generation n is given approximately by

$$\delta\bar{z}_n \approx \delta k \sum_{j=1}^{n} \tfrac{1}{2}^j (1-k)^{j-1}(\theta_{n-j}-\bar{z}_{n-j}). \qquad \text{(A 12}a\text{)}$$

Using equation (13), this becomes

$$\delta\bar{z}_n \approx \delta k \sum_{j=1}^{n} \tfrac{1}{2}^j (1-k)^{j-1} \times\left(\theta_{n-j}-[1-k]^j\bar{z}_0-k\sum_{l=1}^{j}[1-k]^{l-1}\theta_{j-l}\right). \qquad \text{(A 12}b\text{)}$$

Using equation (17), we obtain

$$E\{\delta\bar{z}_n(\bar{z}_n-\theta_n)\} \approx kE\left\{\delta\bar{z}_n\sum_{j=1}^{n}(1-k)^{j-1}\theta_{n-j}\right\}-E\{\delta\bar{z}_n\theta_n\}. \qquad \text{(A 13)}$$

Let A be the first expectation on the right-hand side of equation (A 13). A can be approximated as follows, assuming (without loss of generality) that $E\{\theta_n\}=0$.

$$A \approx k\delta k E\left\{\sum_{j=1}^{n}\tfrac{1}{2}^j(1-k)^{j-1}\left(\theta_{n-j}-k\sum_{l=1}^{j}(1-k)^{l-1}\theta_{j-l}\right)\times\left(\sum_{p=1}^{n}(1-k)^{p-1}\theta_{n-p}\right)\right\}$$

$$\approx k\delta k\left(\frac{V_\theta}{2(1-\frac{1}{2}[1-k]^2)}+\frac{\tau V_\theta}{(1-\frac{1}{2}[1-k]^2)(1-\tau[1-k])}-kC\right), \qquad \text{(A 14)}$$

where

$$C = kE\left\{\sum_{j=1}^{n}\sum_{l=1}^{j}\tfrac{1}{2}^j(1-k)^{j+l-2}\theta_{j-1}\left(\sum_{p=1}^{n}(1-k)^{p-1}\theta_{n-p}\right)\right\}.$$

A lengthy calculation shows that C is asymptotically equal to zero, provided that $k>0$ and $\tau<1$, and so it may be neglected.

A similar analysis yields the asymptotic value of the second expectation on the right-hand side of equation (A 13) as

$$E\{\delta\bar{z}_n\theta_n\} \approx \frac{\tau(1-\tau)V_\theta\delta k}{2(1-\tau[1-k])(1-\frac{1}{2}\tau[1-k])}. \qquad \text{(A 15)}$$

If V is small and $\tau<1-O(V)$, this expectation is the dominant term in equation (A 13). We thus obtain the approximation

$$E\{\delta\bar{z}_n(\bar{z}_n-\theta_n)\} \approx -\frac{\tau V_\theta\delta V}{2-\tau}. \qquad \text{(A 16)}$$

(v) *Effect of a recombination modifier with diploidy*

In the case of diploidy, the methods of Bulmer (1985, p. 159) and Charlesworth (1990) yield the following replacements for equations (A 4) and (A 7):

$$C'_{ij}+\delta C'_{ij} = (1-r_{ij}-\delta r_{ij})([1-\rho_i][C_{ij}+\delta C_{ij}]+\rho_i C_{ij}) + \frac{\Delta+\delta\Delta}{4m(m-1)} \qquad \text{(A 17}a\text{)}$$

$$C'_{ij}+\delta C'_{ij} = (1-\rho_i)(1-\rho_j) \times(C_{ij}+\delta C_{ij})+\rho_i\rho_j C_{ij}+\frac{\Delta+\delta\Delta}{4m(m-1)}. \qquad \text{(A 17}b\text{)}$$

These lead to the following equivalent of equation (A 9)

$$\delta V_G \approx \tfrac{1}{4}E_1\left\{\frac{1}{\rho_i+[1-\rho_i]r_{ij}}\left(\delta\Delta-\frac{\Delta\delta r_{ij}}{r_{ij}}\right)\right\} +\tfrac{1}{4}E_2\left\{\frac{1}{1-[1-\rho_i][1-\rho_j]}\left(\delta\Delta-\frac{\Delta\delta r_{ij}}{r_{ij}}\right)\right\}. \qquad \text{(A 18)}$$

In the case of a single chromosome with short map length, equation (A 18) yields the result that δV is one-quarter the value for the haploid case with the same value of V [cf. equations (12) and (13)]. Similarly, the expression for the case of an unlinked modifier is approximately one-quarter the corresponding value for the haploid case with the same value of V [cf. equations (14*a*, *b*)].

Following Charlesworth (1990, equation [A 10]), the recursion relation for the effect of the modifier on the trait mean, equivalent to equation (A 1*a*), is

$$Z'_i+\delta Z'_i = Z_i+(1-\rho_i)\delta Z_i+\frac{1}{2m}\delta k(\theta_{n-1}-\bar{z}_{n-1}) +k(\theta_{n-1}-\bar{z}_{n-1}-\delta\bar{z}_{n-1})). \qquad \text{(A 19)}$$

In the case of a steadily moving optimum, this equation leads to the equilibrium equation [equivalent to equation (A 2*c*)]

$$\delta\bar{z} \approx \frac{\Delta\theta\delta V}{V(V+2\rho_H)}. \qquad \text{(A 20)}$$

This can be used in conjunction with equation (A 18) to obtain the following expression for the selection coefficient on a modifier, similar to that of equation (A 11*a*).

$$\delta(\ln\bar{w}) \approx \frac{\delta V}{2}\left(\frac{\phi}{V^2}\left[\frac{2}{(V+2\rho_H)}+1\right]-1\right). \qquad \text{(A 21)}$$

The conditions for a selective advantage to recombination are similar to those with haploidy, e.g. with free recombination, the condition for immunity to invasion by an unlinked modifier is again approximately $3\phi > V^2$. With very tight linkage, such that ρ_H is smaller in magnitude than V, the condition for a selective advantage to a modifier increasing recom-

https://doi.org/10.1017/S0016672300031372 Published online by Cambridge University Press

bination is more stringent than with haploidy. The smaller value of δV with diploidy implies that the selection coefficient on a recombination modifier is lower than with haploidy.

The case of a randomly fluctuating environment with an unlinked modifier can be analysed similarly, using equation (A 19) to obtain the equivalent of equation (A 15). Comparison of equation (A 19) with (A 1*b*) with $\rho_t = 0{\cdot}5$ shows that the recursion is the same as in the haploid case. The selection coefficient on a modifier is thus one-quarter that given by equation (26), for the same value of V.

References

Barton, N. H. (1990). Pleiotropic models of quantitative variation. *Genetics* **124**, 773–782.

Bergman, A. & Feldman, M. W. (1990). More on selection for and against recombination. *Theoretical Population Biology* **38**, 68–92.

Bergman, A. & Feldman, M. W. (1992). Recombination dynamics and the fitness landscape. *Physica* **56**, 57–67.

Bulmer, M. G. (1974). Linkage disequilibrium and genetic variability. *Genetical Research* **23**, 281–289.

Bulmer, M. G. (1985). *The Mathematical Theory of Quantitative Genetics*, 2nd edn. Oxford, U.K.: Oxford University Press.

Charlesworth, B. (1984*a*). Some quantitative methods for studying evolutionary patterns in single characters. *Paleobiology* **10**, 308–318.

Charlesworth, B. (1984*b*). The cost of phenotypic evolution. *Paleobiology* **10**, 319–327.

Charlesworth, B. (1987). The heritability of fitness. In *Sexual Selection: Testing the Alternatives* (ed. J. W. Bradbury and M. B. Andersson), pp. 21–40. Chichester, U.K.: John Wiley.

Charlesworth, B. (1989). The evolution of sex and recombination. *Trends in Ecology and Evolution* **4**, 264–267.

Charlesworth, B. (1990). Mutation-selection balance and the evolutionary advantage of sex and recombination. *Genetical Research* **55**, 199–221.

Crow, J. F. (1970). Genetic loads and the cost of natural selection. In *Mathematical Topics in Population Genetics* (ed. K. Kojima), pp. 128–177. Berlin: Springer-Verlag.

Crow, J. F. (1992). An advantage of sexual reproduction in a rapidly changing environment. *Journal of Heredity* (May–June), 169–173.

Falconer, D. S. (1989). *Introduction to Quantitative Genetics*, 3rd edn. London: Longman.

Feldman, M. W. & Liberman, U. (1986). An evolutionary reduction principle for genetic modifiers. *Proceedings of the National Academy of Sciences USA* **83**, 4824–4827.

Feldman, M. W., Christiansen, F. B. & Brooks, L. D. (1980). Evolution of recombination in a constant environment. *Proceedings of the National Academy of Sciences USA* **77**, 4824–4827.

Felsenstein, J. (1965). The effect of linkage on directional selection. *Genetics* **52**, 349–363.

Felsenstein, J. (1974). The evolutionary advantage of recombination. *Genetics* **78**, 737–756.

Fleming, W. H. (1979). Equilibrium distribution of continuous polygenic traits. *Society for Industrial and Applied Mathematics Journal of Applied Mathematics* **36**, 149–168.

Flexon, P. B. & Rodell, C. F. (1982). Genetic recombination and directional selection for DDT resistance in *Drosophila melanogaster*. *Nature* **298**, 672–674.

Gillespie, J. H. (1973). Polymorphism in random environments. *Theoretical Population Biology* **4**, 193–195.

Gorodetskii, V. P., Zhuchenko, A. A. & Korol, A. B. (1991). Efficiency of feedback selection for recombination in Drosophila. *Soviet Genetics* **26**, 1261–1269.

Haldane, J. B. S. (1919). The combination of linkage values and the calculation of distance between loci of linked factors. *Journal of Genetics* **8**, 299–309.

Haldane, J. B. S. & Jayakar, S. D. (1963). Polymorphism due to selection of varying direction. *Journal of Genetics* **58**, 237–242.

Houle, D. (1992). Comparing evolvability and variability of quantitative traits. *Genetics* **130**, 195–204.

Karlin, S. & Liberman, U. (1974). Random temporal variation in selection intensities: case of large population size. *Theoretical Population Biology* **5**, 59–103.

Kimura, M. (1965). A stochastic model concerning the maintenance of genetic variability in quantitative characters. *Proceedings of the National Academy of Sciences USA* **54**, 731–736.

Kondrashov, A. S. (1988). Deleterious mutations and the evolution of sexual reproduction. *Nature* **336**, 435–440.

Kondrashov, A. S. & Turelli, M. (1992). Deleterious mutations, apparent stabilizing selection and the maintenance of quantitative genetic variation. *Genetics* **132**, 603–618.

Korol, A., Preygel, I. & Preygel, S. (1990). *Variability of Crossing Over in Higher Organisms* (in Russian). Kishinev, Moldova: Shtiintsa.

Lande, R. (1975). The maintenance of genetic variability by mutation in a polygenic character. *Genetical Research* **26**, 221–235.

Latter, B. H. D. (1970). Selection in finite populations with multiple alleles. II. Centripetal selection, mutation and isoallelic variation. *Genetics* **66**, 165–186.

Lynch, M. & Gabriel, W. (1983). Phenotypic evolution and parthenogenesis. *American Naturalist* **122**, 745–764.

Lynch, M. & Lande, R. (1992). Evolution and extinction in response to environmental change. In *Biotic Interaction and Global Change* (ed. P. Kareiva, J. G. Kingsolver and R. B. Huey), pp. 234–250. Sunderland, Mass.: Sinauer.

Lynch, M., Gabriel, W. & Wood, A. M. (1991). Adaptive and demographic responses of plankton populations to environmental change. *Limnology and Oceanography* **36**, 1301–1312.

Mather, K. (1943). Polygenic inheritance and natural selection. *Biological Reviews* **18**, 32–64.

Maynard Smith, J. (1978). *The Evolution of Sex*. Cambridge, U.K.: Cambridge University Press.

Maynard Smith, J. (1980). Selection for recombination in a polygenic model. *Genetical Research* **35**, 269–277.

Maynard Smith, J. (1988). Selection for recombination in a polygenic model – the mechanism. *Genetical Research* **51**, 59–63.

Michod, R. E. & Levin, B. R. (1988). *The Evolution of Sex*. Sunderland, Mass.: Sinauer.

Shnol, E. & Kondrashov, A. S. (1993). The effect of selection on the phenotypic variance. *Genetics* (in the press).

Simpson, G. G. (1953). *The Major Features of Evolution*. New York: Columbia University Press.

Stebbins, G. L. (1950). *Variation and Evolution in Plants*. New York: Columbia University Press:

Turelli, M. (1984). Heritable variation via mutation-selection balance: Lerch's zeta meets the abdominal bristle. *Theoretical Population Biology* **25**, 138–193.