Limited Dispersal, Deleterious Mutations and the Evolution of Sex

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ABSTRACT

This study presents a mathematical model that allows for some offspring to be dispersed at random, while others stay close to their mothers. A single genetic locus is assumed to control fertility, and this locus is subject to the occurrence of deleterious mutations. It is shown that, at equilibrium, the frequency of deleterious mutations in the population is inversely related to the rate of dispersal. This is because dispersal of offspring leads to enhanced competition among adults. The results also show that sexual reproduction can lead to a decrease in the equilibrium frequency of deleterious mutations. The reason for this relationship is that sex involves the dispersal of genetic material, and thus, like the dispersal of offspring, sex enhances competition among adults. The model is described using the example of a hemaphroditic plant population. However, the results should apply to animal populations as well.

DEBATE on the evolution of sex continues apace, but there is still no general agreement about the probable solution to the mystery (MAYNARD SMITH 1978; BELL 1982; MICHOOD and LEVIN 1988; HAMILTON et al. 1990; KONDRASTOV 1993). One approach to the evolution-of-sex problem that has received considerable attention has to do with deleterious mutations. A number of researchers have claimed that sex can help to ameliorate the effects of deleterious mutations either by facilitating the repair of these mutations (BERNSTEIN et al. 1988; MICHOOD 1993) or by producing a decrease in the frequency of deleterious mutations in the population (MULLER 1964; HAIGH 1978; KONDRASTOV 1988; LYNCH et al. 1993). However, theoretical studies of deleterious mutations and the evolution of sex have generally assumed a homogeneous environment, and few models have considered the effects of population structure (KONDRASTOV 1993).

In this study, a model of an hermaphroditic plant population is presented. The model allows for recurrent deleterious mutations. It is assumed that the population is structured in a very simple way, so that each individual lives on a site that is capable of supporting, at most, one adult plant. The results show that, if some seeds are not dispersed from the seed-parent's site, then the equilibrium frequency of deleterious mutations (and thus the equilibrium genetic load) is higher than it would have been in a homogeneous population. Furthermore, the equilibrium level of genetic load decreases monotonically as the rate of dispersal increases.

The results also show that sexual reproduction acts in a manner similar to seed dispersal. In particular, genetic load decreases as the rate of sexual reproduction increases. This is because sex involves the dispersal of pollen grains. The relation between sex and genetic load that is described here may help to explain the maintenance of sex despite its apparent cost.

The mechanism described here that produces a reduced load for sexual species can operate in a one-locus model. Thus, the mechanism has nothing to do with genetic recombination. This suggests that the model presented here is very different from most evolution-of-sex models, which typically depend on recombination (FELSENSTEIN 1974). However, the model is not entirely unique in this respect. Other evolution-of-sex models that do not depend on recombination include studies by KIRKPATRICK and JENKINS (1989) and by ESHEL and WEINSHALL (1987).

It is possible to provide an intuitive account that explains the results presented in this study. However, this will be deferred until the model and the results have been fully described.

THE MODEL

Consider a diploid population of annual seed-bearing plants. Each adult is assumed to produce a very large (effectively infinite) number of seeds, and a proportion $\phi$ of these seeds is produced by sexual means, while the rest are produced asexually (where $0 < \phi < 1$). The value of $\phi$ is the same for all individuals. If $\phi > 0$ (so that some seeds are produced sexually), then each adult produces pollen, in addition to seeds. The number of pollen grains that an individual produces is assumed to be proportional to the number of seeds produced by that individual. Furthermore, the coefficient of proportionality is assumed to be large enough so that there are always sufficient pollen grains available for all fertilizations. When adults generate seeds sexually, they produce an ovule, and this is fertilized by a pollen grain that is randomly selected from the total collection of all pollen grains produced in the popula-
tion during that generation. Standard Mendelian segregation is involved in the production of both ovules and pollen. When an adult produces a seed asexually, it simply copies its genetic material into the seed, so that the seed and its parent are genetically identical, except for new mutations.

Consider a single genetic locus in which the wild-type allele is designated by B. Assume that this allele is subject to unidirectional mutation, which occurs at a rate of $\mu$ per generation (where $0 < \mu < 1$). The probability that a new mutation will be incorporated into a seed is independent of whether the seed has been produced sexually or asexually. Mutant alleles are deleterious, and for simplicity it is assumed that all mutant alleles have the same effect upon fertility. Mutant alleles are designated by b.

There are three possible genotypes, BB, Bb, and bb. The frequencies of these three genotypes among the adults during a given generation are represented by $x$, $y$ and $z$, respectively (with $x + y + z = 1$). Assume that the number of seeds (and pollen grains) produced by adults with the BB, Bb and bb genotypes are proportional to 1, $(1 - s_1)$ and $(1 - s_2)$, respectively. As $b$ is deleterious, let us assume $0 < s_1 \leq s_2 < 1$.

Assume that the habitat in which the plant species lives contains an infinite number of sites, each of which can support the development of, at most, one adult. Each juvenile survives to adulthood with probability $\gamma$ and $\nu$, and thus, the population is infinite in size. Of the seeds produced by any given adult, a proportion $\alpha$ survives to adulthood is unlikely. Note that the environment is harsh and/or disturbed, in that the probability of survival to adulthood is low. If $s_1$ is sufficiently large in comparison to $\mu$, and if $m$, $\phi$ and $\alpha$ are not all equal to zero, then we can show that the equilibrium frequency of $b$ will be low at the equilibrium that will be attained if we start with a pure $B$ population (i.e., with $x = 1$ and $y = z = 0$). Unless specified otherwise, all references to equilibrium henceforth in this paper will refer to this low-frequency-of-$b$ equilibrium.

Let $\bar{x}$, $\bar{y}$ and $\bar{z}$ represent the equilibrium frequencies of BB, Bb, and bb adults, respectively. If $m$, $\phi$ and $\alpha$ are not all equal to zero, and if $s_1$ is sufficiently large in comparison to $\mu$, then $\bar{y}$ is guaranteed to be small, and we can write the following:

$$\bar{y} \approx \frac{4\mu(1 - s_1 + ms_1)}{[2s_1(1 - s_1)(\alpha + m(1 - \alpha)(2 - m)] + 2ms_1^2 + \phi s_1(1 - \alpha)(1 - s_1)(1 - 2m + m^2)} \tag{1}$$

The derivation of Equation 1 is explained in the APPENDIX. Let $\bar{y}$ represent the right side of Equation 1. It is straightforward to show that Equation 1 is accurate, except for terms of order $\bar{y}^2$ and terms of order $\mu^2$ (see the APPENDIX). Throughout this paper, $\approx$ will mean the same thing (i.e., that the equation in question is accurate, except for terms of order $\bar{y}^2$ and terms of order $\mu^2$). It is also straightforward to show that, so long as $\bar{y}^2$ and $\mu^2$ are very small, $\bar{y}$ is a locally stable equilibrium that will be approached from any very low initial frequency of $b$ (this is demonstrated in the APPENDIX).

Say that $\bar{w}$ represents a quantity that is proportional to the average fertility of population members. In particular, let $\bar{w} = x + y(1 - s_1) + z(1 - s_2)$. If we assume that $m$, $\phi$ and $\alpha$ are not all equal to zero, and that $s_1$ is sufficiently large to ensure that $\bar{y}$ is small, then it is possible to write a simple but reasonably accurate approximation for $\bar{w}$. This is because, when these assumptions are met, we have $\bar{z} = O(\bar{y}^2)$, and thus we can write

$$\bar{w} \approx 1 - \bar{y} \bar{s}_1. \tag{2}$$

Given our assumptions of a low mutation rate and a single locus under selection, we can expect that values of $\bar{w}$ close to 1.0 (which is the maximum possible value of $\bar{w}$) will be typical. Because of this, it is convenient to work in terms of genetic load ($L$), rather than $\bar{w}$. Load is defined as $L = (w_{\text{max}} - \bar{w})/w_{\text{max}}$, where $w_{\text{max}}$ is the
maximum possible value of $\bar{v}$. In the present model we have $u_{\text{max}} = 1$, and so we can define load as follows:

$$L = 1 - \bar{v}$$

(3)

Note that, in this traditional definition, load constitutes a comparison of the mean fitness to the fitness of the best-possible genotype, regardless of whether this best-possible genotype is actually present in the population.

**Load when all seeds are dispersed or when $\alpha \approx 1$:** Let us now turn to the question of how the various parameters affect genetic load. To begin, consider the case where $m = 1$, so that the distribution of the different sorts of seeds is the same on each site. In this case, we can use Equation 1 to show that at equilibrium

$$L \approx 2\mu.$$  

(4)

This is a classic result, which is due to J. B. S. Haldane (1937). This same equilibrium value of $L$ holds in the limit as $\alpha \rightarrow 1$ for any positive value of $m$. This makes sense, as a high value of $\alpha$ has a similar effect to setting $m = 1$, in that, before germination, most sites have the same distribution of the different seed types. This is because, when $\alpha$ is close to 1.0, there is no adult on most sites, and the seeds on these empty sites are drawn at random from the total pool of seeds produced by all adults. Note that when $m = 1$ (or in the limit as $\alpha \rightarrow 1$), the equilibrium value of $L$ is independent of $\delta$, the proportion of sexually produced seeds. In other words, when seeds are highly dispersed, the degree of sexuality has virtually no effect on mean fitness, and the same is true when nearly all juveniles die before they achieve adulthood.

**Load under asexuality when no genetic material is dispersed:** Next, let us consider the case where $\phi = 0$, $m = 0$ and $\alpha = 0$. This is the case where no genetic material is dispersed in the form of seeds, and none is dispersed in the form of reproductively successful pollen grains. This case was excluded when Equation 1 was derived. Using the equations in the APPENDIX, it is straightforward to show that, when $\phi = 0$, $m = 0$ and $\alpha = 0$, the value of $z$ rises inexorably until equilibrium is achieved at $x = 0$, $y = 0$ and $z = 1$. In other words, the frequency of $b$ increases until it goes to fixation. To be more precise, if $z'$ is the frequency of $bb$ adults in the next generation, then, when $\phi = 0$, $m = 0$ and $\alpha = 0$, we have $z' = z + x\mu + y\mu^2$. At equilibrium, only $bb$ individuals are present in the population. Once this equilibrium is achieved, the equilibrium level of load is given by

$$L = s_2.$$  

(5)

This represents the lowest level of fertility possible under the assumptions of the model. This result holds because, if there is no movement of genetic material between sites, then for each site there is only one adult that can provide the genetic material necessary to produce a juvenile on that site during the next generation. This means that there is no competition between adults. In the absence of competition, differential fertility has no power to impede the force of mutation, and eventually $\delta$ approaches fixation, load approaches its maximal value, and fertility approaches its minimum value.

**The effect of migration on load:** For the remainder of this paper, let us exclude the case where $\phi = 0$, $m = 0$ and $\alpha = 0$ and return to our assumption that $s_j$ is sufficiently large to ensure that $z$ is small. Under this assumption, what is the effect of the rate of migration upon the level of load at equilibrium? We have seen that when no genetic material is dispersed by any means, load reaches its maximum possible value (Equation 5). Thus, it is not surprising to learn that, in general, the equilibrium value of $L$ (which will be designated as $L^*$) is a monotonically decreasing function of $m$, the rate of migration. In particular, the relation is as follows:

$$\frac{\partial L}{\partial m} = \frac{-4\mu(1 - \alpha)(1 - s_j)(1 - m)}{2(1 - s_j)[\alpha + m(1 - \alpha)]} \times (\delta - 2m) + 2ms_1 + \phi(1 - \alpha)$$

$$\times (1 - s_j)(1 - 2m + m^2)^2$$

(6)

Examination of this equation reveals that, in the limit as $\alpha$ goes to 1.0, $m$ has no effect on load at equilibrium. This result is already implied by Equation 4. Equation 6 also reveals that $m$ has no effect on the equilibrium level of load in the limit as $s_j$ approaches 1.0.

**The effect of sexual reproduction on load:** How does the rate of sexual reproduction affect genetic load? To answer this question, let us examine the partial derivative of $L$ with respect to $\phi$. This is given by

$$\frac{\partial L}{\partial \phi} = \frac{-4\mu(1 - \alpha)}{2(1 - s_j)[\alpha + m(1 - \alpha)]} \times (\delta - 2m) + 2ms_1 + \phi(1 - \alpha)$$

$$\times (1 - s_j)(1 - 2m + m^2)^2$$

(7)

Examination of this equation reveals that, in the limit as $\alpha$ goes to 1.0, $\phi$ has no effect on load, and the same is true as $m$ goes to 1.0 and as $s_j$ goes to 1.0. However, for any other choice of parameters, an increase in $\phi$ will decrease load. Thus, it appears that increasing the rate of sexual reproduction is good for the genetic health of the population, except when seeds are very well dispersed, when juvenile viability is very low, or when a single deleterious mutation results in a nearly total loss of fertility. This result makes sense in light of the intuitive explanation of Equation 5. Dispersal of genetic material between sites enhances competition between adults. The production of pollen is one way that genetic material can be dispersed, and it is most
effective when $\phi$ is large, so that most seeds are produced after incorporation of genetic material donated by a pollen grain.

The effect of mutation rate on load: The partial derivative of $\hat{L}$ with respect to the rate of mutation ($\mu$) is given by

$$\frac{\partial \hat{L}}{\partial \mu} \approx \frac{4(1 - s_i + ms_i)}{[2(1 - s_i)[2 + m(1 - \alpha) - \mu]} \times (2 - m) + 2ms_i + \phi(1 - \alpha) \times (1 - s_i)(1 - 2m + m^2)$$  \tag{8}

Examination of Equation 8 reveals, as one would expect, that the equilibrium level of load always increases with $\mu$. In fact, we can make an even stronger statement in light of Equation 8. In particular, $\hat{L}$ is approximately proportional to $\mu$. This must be so because $\mu$ does not appear on the right side of Equation 8.

The effect of juvenile viability on load: The relationship between juvenile viability ($\alpha$) and the equilibrium level of load is given by

$$\frac{\partial \hat{L}}{\partial \alpha} \approx \frac{[-4\mu(2 - \phi)(1 - s_i) \times (1 - s_i + ms_i)(1 - m^2)]}{[2(1 - s_i)[2 + m(1 - \alpha)]}$$.  \tag{9}

This equation shows that, so long as $m < 1$, the equilibrium level of genetic load will decrease as $\alpha$ increases. This is not surprising given that (as mentioned previously) a high value of $\alpha$ leads to a high level of competition between adults.

The effect of the strength of fertility selection on load: As we saw earlier, the strength of fertility selection ($s_i$) has virtually no effect on the equilibrium level of genetic load when $m = 1$, or in the limit as $\alpha \to 1$ (see Equation 4). However, when $m < 1$, the value of $s_i$ does have an effect on $\hat{L}$, and the relation is as follows:

$$\frac{\partial \hat{L}}{\partial s_i} \approx \frac{-4\mu(1 - \alpha)(2 - \phi)(1 - m^2)}{[2(1 - s_i)[2 + m(1 - \alpha)]}$$.  \tag{10}

This equation shows that, when $m < 1$, an increase in $s_i$ will result in a decrease in the level of load at equilibrium. In the limit as $s_i$ approaches unity, we have

$$\lim_{s_i \to 1} \hat{L} = 2\mu. \tag{11}$$

Thus, the lowest level of load that can be achieved by manipulating $s_i$ is the same as the lowest level that can be achieved by manipulating $m$ or $\alpha$ (see Equation 4).

The case where $m = 0$ and $\alpha = 0$: Let us now consider the special case where $m = 0$ and $\alpha = 0$, so that there is no movement of seeds between sites, and all juveniles survive to adulthood. In this case, $\hat{L}$ is given by

$$\hat{L} \approx \frac{4\mu}{\phi}. \tag{12}$$

This is a striking result, as it tells us that when there is no movement of seeds between sites, the degree of sexuality can have a very large effect upon load. Furthermore, when $\phi = 1$, half of all the genetic material in seeds comes from outside of the seed-parent’s site, and yet load is twice what it would be if there were complete dispersal of seeds ($m = 1$). Note also, from Equation 12, that when $m = 0$ and $\alpha = 0$, the effect of $\phi$ on load is strongest when $\phi$ is small, but it is substantial even when $\phi$ is large. In particular, in the limit as $\phi$ approaches 1.0, load decreases linearly as $\phi$ increases, and slope in the region of $\phi = 1$ is approximately equal to $-4\mu$. (However, recall that Equation 12 is only valid so long as $\phi$ is not so small that $\hat{y}$ takes on a substantial value.)

Equation 12 was derived under the assumption that $\hat{y}$ is small. As implied by the discussion of Equation 5, this assumption will be violated if $m = 0$, $\alpha = 0$ and $\phi$ is sufficiently small. However, Equation 12 is roughly accurate for a wide variety of biologically reasonable parameter choices. For example, if $\mu = 10^{-5}$, $s_i = 0.02$ and $\phi = 0.1$, then we have $\hat{y} \approx 0.02$. Any error in the estimation of the equilibrium load is on the order of $s_i(\hat{y}^2 + \mu^2)$ (see Equations 2 and 3). Thus, in this case, errors should be on the order of $10^{-5}$.

The case where $m > 0$ and $\alpha > 0$: What is the effect of the magnitude of $\phi$ upon load when $m > 0$ and $\alpha > 0$? This question is addressed by Figure 1. The figure shows that when $s_i$ is small in absolute terms (but large in comparison to $\mu$), the decrease in load brought about by a transition from $\phi = 0$ (asexuality) to $\phi = 1$ (obligate sexuality) is substantial (in excess of 10%) for a wide range of parameters. As the figure shows, this can be so even for high values of $\alpha$ (e.g., $\alpha = 0.75$) or high values of $m$ (e.g., $m = 0.5$).

**DISCUSSION**

The results provide support for the idea that when deleterious mutations occur repeatedly, limited dispersal of offspring leads to an increase in the equilibrium level of genetic load. In other words, when dispersal is limited, the equilibrium frequency of deleterious mutations is higher than when all offspring are dispersed. From one point of view, this result may seem surprising, as previous work shows that population structure appears to have virtually no effect on the long-term fates of mutations that are beneficial, rather than deleterious (Maruyama 1970). On the other hand, deleterious mutations that arise repeatedly will eventually become fixed in a finite population (Crow and Kimura 1970). Thus, from another point of view, it would be...
The accuracy of these curves was checked using the complete production by sexual means. Note that calculated using Equations 1, used. However, when \( j \) is small, these curves are more or less broad range of parameter values. In all cases, generations (each of which can support one adult).

Genetic load, even in a population like the one studied here, which is infinite, but divided into finite subpopulations. The results show that dispersal decreases genetic load. After all, under the assumptions of the model, sex involves outbreeding, and thus it necessarily involves the movement of genetic material (i.e., pollen) from one site to another. Therefore, sex is a type of dispersal. It was also shown that, under the assumptions of the model, the equilibrium level of load \( \bar{L} \) is enhanced by a high mutation rate. Furthermore, if dispersal is limited, \( \bar{L} \) is inversely related to the rate of juvenile survival \( (\alpha) \) and to the strength of fertility selection against heterozygotes \( (s_i) \).

An intuitive explanation for the relation between limited dispersal and genetic load was given after presentation of Equation 5. The basic idea is that dispersal of genetic material (either by seed dispersal or by dispersal of reproductively successful pollen grains) leads to an increase in the genetic diversity of the individuals who compete for each of the sites upon which an individual can grow to adulthood. Genetic diversity generally enhances the effectiveness of natural selection (Fisher 1930; Crow and Kimura 1970). Thus, genetic load is lower when there is more dispersal than when there is less. This intuitive explanation suggests that results similar to those presented here could be generated for a model in which fitness depends on multiple loci, or for a model in which individuals live in populations that contain more than one adult. I have produced preliminary results that show that population subdivision can increase genetic load when an infinite-sized population is subdivided into groups that contain multiple individuals. However, much more must be done before the generality of the results presented here can be accepted. The intuitive explanation also suggests that a process similar to the one documented here would favor the evolution of inbreeding-avoidance mechanisms.

The model presented here incorporates fertility selection. This is both mathematically convenient and biologically plausible for many plants. However, it seems clear that similar results could be produced for a viability-selection model. To see this, let us modify the model by assuming that all breeding adults produce the same number of seeds, and that the germination of these seeds and the survival of the resulting plants to reproductive age depends on the seeds’ genotypes. Clearly, if there is one-way mutation to deleterious alleles, and if there is no movement of genetic material between sites as a result of colonization, migration or sexual reproduction, then deleterious alleles will become fixed in the population as a result of genetic drift on each of the sites (so long as the probability of survival is positive for all genotypes). Clearly, if there is one-way mutation to deleterious alleles, and if there is no movement of genetic material between sites as a result of colonization, migration or sexual reproduction, then deleterious alleles will become fixed in the population as a result of genetic drift on each of the sites (so long as the probability of survival is positive for all genotypes). However, if selection is sufficiently strong and migration or sex helps to keep the population of juveniles on each of the sites genetically heterogeneous, then it will be possible to keep the frequency of deleterious mutations from rising to a high frequency.

The model assumes that mutation is unidirectional. It is unlikely that this assumption will be strictly realized

The analysis focused on how the various parameters of the model affect \( \bar{L} \), which is the equilibrium level of genetic load. The results show that dispersal decreases the equilibrium level of load. What is more intriguing is the observation that if dispersal is limited (i.e., \( m < 1 \)), then the rate of sexual reproduction also reduces the equilibrium level of genetic load. This result makes sense in light of the relation between dispersal and load. After all, under the assumptions of the model, sex involves outbreeding, and thus it necessarily involves the movement of genetic material (i.e., pollen) from one site to another. Therefore, sex is a type of dispersal. It was also shown that, under the assumptions of the model, the equilibrium level of load \( \bar{L} \) is enhanced by a high mutation rate. Furthermore, if dispersal is limited, \( \bar{L} \) is inversely related to the rate of juvenile survival \( (\alpha) \) and to the strength of fertility selection against heterozygotes \( (s_i) \).
in natural populations. However, so long as the frequency of \( b \) is small (as assumed in the estimation of \( \hat{f} \) by \( \hat{y} \)) the effects of back mutation should be negligible. Nevertheless, if a similar model to the one presented here were to be produced for a finite population, incorporation of back-mutation might be worthwhile, as it would prevent permanent fixation of \( b \) when (and if) this disfavored allele drifts to high frequency.

It might also be interesting to study a model in which a continuous range of mutant effects are possible, and in which there is no limit to the effects on fitness that can be produced by the variants at a locus. In such a model, we might expect that genetic drift would lead to a steady increase in genetic load under the conditions used to derive Equation 5, where there is no mechanism for the dispersal of genetic material between sites. So long as there is some limit to fecundity, this should eventually lead to extinction of the population [i.e., a “mutational meltdown” (LYNCH et al. 1993, 1995)].

Unfortunately, it is not at all clear that the effects documented in this study can account for the evolution and maintenance of obligate sexuality and outbreeding in natural populations. It should be recognized that the model is a highly simplified caricature of reality, and thus it must be regarded as an early foray into the study of dispersal and sex in natural populations. The assumed population structure is extremely simple, with a maximum of one breeding adult per site, and a migration scheme that does not take account of how the sites are arranged in space. Furthermore, only a single locus is assumed to control fitness, and this is certainly false in natural populations. Nevertheless, it is reasonable to expect that the results produced will, at least, provide an indication of the qualitative nature of the results that would be obtained from a much more realistic model.

The applicability of the model depends, in part, on parameter values in natural populations. For example, \( \hat{y} \) (the right side of Equation 1) is used extensively in the model, but it is only a reasonably accurate approximation to \( y \) when it is reasonably small. If the per-locus mutation rate is at a biologically reasonable level (say \( 10^{-5} \)), then this presents no problem so long as there is significant dispersal of genetic material among sites (i.e., at least one of the parameters \( \phi, m \) and \( \alpha \) must be substantially in excess of zero) and selection coefficients are of the level typically calculated in mutation-accumulation experiments [such as the classic work of MUKAI (1972)]. In these experiments, selection coefficients of \(-2\%\) are commonly observed. With \( \mu = 10^{-5} \) and \( s = 0.02 \), \( \hat{y} \) takes on a low value even with quite restricted genetic dispersal. For example, with these values, if \( m = 0.05, \phi = 0 \) and \( \alpha = 0 \), then we have \( \hat{y} \approx \hat{y} \approx 0.01 \). However, mutations with very small selection coefficients may well be common (OHTA 1992). If mutations are sufficiently small in effect, the analytic methods used here will not be applicable, though it would not be surprising if a model that incorporated very small effect mutations produced results similar to those presented here.

Clearly, the processes described here can not account for sex and outbreeding if juvenile survival rates are very low, or if seeds are highly dispersed. This is encouraging, as highly dispersed plant species are often partially or fully asexual or selfed (STEBBINS 1958; BAKER 1974). In animals, the picture is less clear. Bell claims that strong dispersers \( (e.g., \) butterflies and dragonflies) tend to be sexual (BEll 1982), and this appears to contradict one of the basic predictions of the theory. However, the strong dispersers cited by Bell have larval stages that are not highly dispersed. Much selection can occur during larval maturation, so the contradiction may be more apparent than real. In animals with internal fertilization, there is also opportunity for selective abortion. The same may be true for many plants (STEPHenson and BERTIN 1983). With some modification, the model could probably be made to apply to these selective-abortion situations. After modification, the group of embryos produced and nurtured by an adult would constitute the group of siblings on a site. Of course, in this case, there would be no migration between “sites.” Thus, the effect of the rate of sexual reproduction upon genetic load could be very large in this instance (see Equation 12).

It is interesting to note that harsh environments \( (xeric \) areas, high altitudes, high levels of disturbance, etc.) are apparently associated with asexuality and/or inbreeding in both plants and in animals, and these environments may well be characterized by low rates of juvenile survival (STEBBINS 1958; BAKER 1974; BELL 1982; LYNCH 1984; but see BIERZYCHUDEK 1985 for a contrary view regarding plants). The apparent association between environmental harshness and asexuality or inbreeding is consistent with the theoretical predictions of the model. In particular, we have seen that the rate of sexual reproduction has little effect on genetic load when the environment is very harsh (as reflected in a low juvenile-survival rate).

The relevance of the results presented here depends on the load experienced at each locus, and on the way in which load at different loci combines. The per-locus load depends on the deleterious mutation rate, as well as on the processes described above. Current estimates of genomic mutation rates in higher plants and animals range from 0.1 to 100, though both the low-end and the high-end of this range seem unlikely (MUKAI et al. 1972; KONDRAshov 1988; CHARLESWORTH et al. 1990; HOUlE et al. 1992; KONDRAshov and TURELLI 1992; KEIGHTLEY 1994). If actual mutation rates are on the low-end of this range, then, under a simple multiplicative model, very substantial decreases in per-locus load might have to be associated with sex to produce a benefit of sufficient size to compensate for the so-called two-
fold cost of sex (Maynard Smith 1978). However, if mutation rates are more substantial, then a modest amelioration of per-locus load levels might be sufficient.

The benefits of sex that are highlighted by the results presented here are largest when dispersal of seeds between sites occurs at a low rate (see Figure 1). With this in mind, it is important to recognize that the convenient assumption that dispersed seeds land on randomly selected sites is unlikely to be met in most natural populations. Rather, it is probably safe to say that seeds will typically be more likely to land on sites close to their seed-parents, rather than on distant sites (Harper 1977). This phenomenon may well make seed dispersal less effective and thus accentuate the benefits of sex. It seems likely that the benefits of sex are further accentuated in real populations because the typical distance of pollen dispersal is probably much greater than the typical distance of seed dispersal.

The results presented in this paper are relevant for determining the outcome of ecological contests between competing species. However, it would be worthwhile to consider whether processes similar to those described here can produce an advantage for sexuality when there is variation in the mode of reproduction within species, rather than between them.

As a final note of caution regarding the applicability of the results, it is important to recognize that, in a complex structured population, differences in levels of genetic load are not the only determinant of the outcome of ecological contests. Analysis relevant to this point will be presented elsewhere.

Evolutionary processes related to the ones described in this paper might be important in a variety of contexts. The results suggest that the effectiveness of natural selection is diminished when dispersal is limited. If this is so, then we should expect that in a finite population limited dispersal would enhance the rate of fixation of very mildly deleterious mutations. Because of the large number of loci that affect fitness, the resulting "drift load" can have a large impact on viability and fertility (Kimura et al. 1963; Tachida 1990; Lande 1994; Kondrashov 1995; Lynch et al. 1995). Similarly, in a population that is both asexual and finite, the results suggest that limited dispersal will tend to accelerate the action of Muller's ratchet and related processes (Muller 1964; Wagner and Gabriel 1990; J. R. Peck, G. Barreau, and S. C. Heath, unpublished results). Finally, as the effectiveness of constant selection pressures is apparently diminished by asexuality when dispersal is limited, it seems plausible to suggest that obligately sexual populations will respond to the transitory selection pressures that can be produced by a fluctuating environment more readily than asexual or partially asexual species.

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LITERATURE CITED


APPENDIX

This appendix presents the derivations of the basic recursions for $x$, $y$, and $z$. In addition, $\hat{y}$ will be derived, and it will be demonstrated that $\hat{y}$ is stable so long as it is small.

Let $p^*$ represent the frequency of $B$ among pollen grains and ovules. It is straightforward to show that $p^*$ is given by

$$p^* = \frac{(1 - \mu)[2x + y(1 - s_i)]}{2\bar{w}}.$$  \hspace{1cm} (A1)

Let us number the three genotypes as 1, 2 and 3 for BB, Bb, and bb, respectively. Say that an adult with genotype $i$ produces seeds with genotypes BB, Bb and bb in frequencies $x_i^*$, $y_i^*$, and $z_i^*$, respectively. These seeds may be produced both by sexual means and by asexual means. Consider a Bb adult (which is the most complicated case). The frequency of BB seeds, BB seeds and bb seeds among this adult's asexually produced seeds is clearly 0, $(1 - \mu)$, and $\mu$, respectively. For sexually produced seeds the corresponding frequencies are $(1 - \mu)p^*/2$, $(1 - \mu)p^* + \mu p^*$, and $\mu p^* + q^*/2$, respectively, where $q^* = (1 - p^*)$. After summation and simplification, we have the following:

$$x_i^* = \frac{\phi p^*(1 - \mu)}{2},$$ \hspace{1cm} (A2)

$$y_i^* = (1 - \mu) - \frac{\phi(1 - \mu(1 + 2p^*))}{2},$$ \hspace{1cm} (A3)

$$z_i^* = \mu + \frac{\phi(q^* (1 + \mu) - 2\mu)}{2}.$$ \hspace{1cm} (A4)

The values of $x_i^*$, $y_i^*$, and $z_i^*$ are derived in a similar manner for $i = 1$ and $i = 3$.

Let $x_n$, $y_n$, and $z_n$ represent, respectively, the frequency of BB, Bb and bb genotypes within the total collection of all seeds produced in the population. Thus, we have $x_n = [x_1^* + y(1 - s_1)x_1^* + z(1 - s_2)x_1^*]/\bar{w}$, $y_n = [y_1^* + y(1 - s_1)y_1^* + z(1 - s_2)y_1^*]/\bar{w}$ and $z_n = [(x_2^* + y(1 - s_1)x_2^* + z(1 - s_2)x_2^*)]/\bar{w}$. Let $x_i^*$, $y_i^*$ and $z_i^*$ represent, respectively, the frequency of BB, Bb and bb seeds on sites occupied by adults with genotype $i$ after seed dispersal. Thus, for example, we have

$$x_i^* = \frac{x_i^* (1 - s_i) (1 - m) + \bar{w}d_{x_i}}{(1 - s_i) (1 - m) + \bar{w}},$$ \hspace{1cm} (A5)

$$y_i^* = \frac{y_i^* (1 - s_i) (1 - m) + \bar{w}d_{y_i}}{(1 - s_i) (1 - m) + \bar{w}},$$ \hspace{1cm} (A6)

and so forth. Note that these equations reflect the fact that after seed dispersal the site of an adult with a relatively low fertility will contain a smaller proportion of seeds that were produced on that site than will be the case on the site of a high-fertility adult.

The final step in the derivation is very simple. The probability that a seed with a given genotype will germinate on a site occupied by an adult with genotype $k$ is just $x_i^*$, $y_i^*$ and $z_i^*$ for seeds with genotypes BB, Bb, and bb, respectively. On sites on which the juvenile has died before producing seeds, these same probabilities are given by $x_n$, $y_n$, and $z_n$, respectively. Thus, if the value of $x$, $y$ and $z$ in the next generation are represented by $x'$, $y'$ and $z'$, then we have

$$x' = (1 - \alpha)(xx^* + yy^* + zz^*) + \alpha x_n,$$ \hspace{1cm} (A7)

$$y' = (1 - \alpha)(xy^* + yy^* + zz^*) + \alpha y_n,$$ \hspace{1cm} (A8)

and

$$z' = (1 - \alpha)(xx^* + yy^* + zz^*) + \alpha z_n.$$ \hspace{1cm} (A9)

We know that $x' = 1 - y' - z'$ and $x = 1 - y - z$, and so these three recursions can be reduced to two.

As stated in the main text, $\hat{y}$ represents the right side of Equation 1, and it gives the approximate value of the equilibrium frequency of $y$ ($\hat{y}$) when $\hat{y}$ is small. The difference between $y$ and $\hat{y}$ is on the order of $(\hat{y}^2 + \mu^2)$. To derive $\hat{y}$, we simply take the value of $y'$ from Equation A8 and solve the equation $y' = y$ for $y$ after setting $z = 0$, $y = (1 - x)$, and ignoring terms that include $\mu^2$, $\hat{y}^2$ or $\mu y$ (we can safely set $z = 0$ because, under the assumptions of the model, $z$ is on the order of $[\hat{y}^2 + \mu^2]$ when $y$ is small). Note that $\hat{y}$ is a unique solution of this equation.

We can show that $\hat{y}$ is stable when it is small by using Equation A8 to approximate $y'$, and setting $z = 0$, $y = (1 - x)$, and ignoring terms that include $\mu^2$, $\hat{y}^2$ or $\mu y$. The system then becomes one-dimensional. We can use the approximate expression for $y'$ to show that when $y = 0$, $y' \approx 2\mu$. It is straightforward to use this same approximate expression for $y'$ to show that $(dy'/dy) > 0$ when $y$ is small. This fact, along with the observation that $y' > 0$ when $y = 0$, implies that the value of $y$ will approach $\hat{y}$ from any initial value so long as $\hat{y}$ is small and the initial frequency of $b$ (and hence, $y$) is also small.