What's wrong with a little sex?

J. R. PECK & D. WAXMAN

Centre for the Study of Evolution and School of Biological Sciences, University of Sussex, UK

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Abstract

In many species, most (or all) offspring are produced by sexual means. However, theory suggests that selection should often favour the evolution of species in which a small fraction of offspring are produced sexually, and the rest are produced asexually. Here, we present the analysis of a model that may help to resolve this paradox. We show that, when heterozygote advantage is in force, members of species in which sex is rare will tend to produce poorly adapted offspring when they mate. This problem should be less severe in species where most offspring are produced by sexual means. As a consequence, once the rate of sexual reproduction becomes sufficiently rare, the benefits of sex may vanish, leading to the evolution of obligate asexuality. Substantial benefits of sexual reproduction may tend to accrue only if a large proportion of offspring are produced sexually. We suggest that similar findings are likely in the case of epistatic interactions between loci.

Introduction

Asexuality has its advantages. For example, in asexual lineages that are derived from sexual species, male reproductive effort is often much reduced, or absent (Bell, 1982; Dudman & Richards, 1997). (Male reproductive effort includes pollen production, production of male individuals, etc.) The diminution of male reproductive effort can lead to a large ecological advantage because it frees up reproductive resources that can be used for the production of offspring (Maynard Smith, 1978).

Asexuality also has its down side. For example, asexuality may make it difficult for a population to evolve in response to changing environmental conditions (Michod & Levin, 1988; Hamilton *et al.*, 1990; Hurst & Peck, 1996). Asexuality can also increase susceptibility to deleterious mutations (Hurst & Peck, 1996; Michod & Levin, 1988).

In some species, offspring are produced both by sexual means and by asexual means. This is true, for example, of a variety of flowering plants (Briggs & Walters, 1984). Theory suggests that, in these species, the amount of male reproductive effort (e.g. pollen production) will increase more or less linearly with the proportion of offspring that are produced sexually. However, most relevant theoretical studies also suggest that most of the benefits of sex accrue if only a small proportion of offspring are produced sexually (Charlesworth *et al.*, 1993; Green & Noakes, 1995; Hurst & Peck, 1996; Peck *et al.*, 1997). As a result, two recent reviews of the evolution-of-sex literature have expressed puzzlement at the evolution of the many species where most (or all) reproduction is sexual (Green & Noakes, 1995; Hurst & Peck, 1996).

Here, we suggest a possible solution to the paradox of species in which most reproduction is sexual. This solution has to do with an advantage of asexuality that is more subtle than the diminution of male reproductive effort. In particular, asexual species can take advantage of nonadditive genetic variation, while sexual species cannot. For example, when heterozygote advantage is in force, an asexual species can achieve an equilibrium where only heterozygotes are present. This is the optimal state under heterozygote advantage. As we shall see, a population where only a small proportion of offspring are produced sexually (and the rest asexually) can reach a state where nearly all individuals are heterozygotes. Such a near-optimal state is inaccessible to obligately sexual species because of the action of segregation. A species where most reproduction is asexual can take advantage

Correspondence: Joel R. Peck, School of Biological Sciences,
The University of Sussex, Brighton BN1 9QG, UK.
Fax: +44 1273 678433;
e-mail: j.r.peck@sussex.ac.uk

of nonadditive genetic variation, but this benefit comes with a price. We shall show that the sexually produced offspring of such a species may be much less fit than the sexually produced offspring in a species where most reproduction is by sexual means. Thus, in a species where sex is rare, the benefits of sex may be very small. To obtain substantial benefits from sex, it may often be necessary to produce most offspring by sexual means.

To illustrate our argument, we use a model of heterozygote advantage. However, we expect that similar considerations will apply in the more complex case of epistasis.

The model and results

We consider a very large and diploid population of hermaphrodites. Each individual is capable of producing offspring by means of 'female effort' (i.e. by providing genetic material plus the essential nutrients and protection necessary for initial development). For each offspring that a given individual produces via female effort, there is a probability ϕ that the offspring will be produced by sexual means, and a probability $1 - \phi$ that the offspring will be produced asexually (where $0 \leq \phi \leq 1$). Thus, if $\phi = 1$, then the species is obligately sexual, and it is obligately asexual if $\phi = 0$.

Assume that fitness is controlled by a single genetic locus with two possible alleles (B_1 and B_2). Say that no mutations occur at this locus. Assume that generations are nonoverlapping, so that adults die shortly after the birth of their offspring. Say that the probability of becoming an adult (i.e. the probability of surviving to reproductive age) for the three possible genotypes ($\mathrm{B}_1\mathrm{B}_1$, $\mathrm{B}_1\mathrm{B}_2$ and $\mathrm{B}_2\mathrm{B}_2$) are given by $1 - s_1$, 1 and $1 - s_2$, respectively (where $0 < s_1 \leq 1$ and $0 < s_2 \leq 1$). This means that the model involves viability selection, and the $\mathrm{B}_1\mathrm{B}_2$ heterozygote is more viable than either of the homozygotes. (Note: the results would be qualitatively unaltered if we did not assume that heterozygotes always survive, but this assumption facilitates exposition.) We shall ignore the special and biologically unrealistic case where $s_1 = s_2$ (i.e. where the $\mathrm{B}_1\mathrm{B}_1$ and $\mathrm{B}_2\mathrm{B}_2$ homozygotes have exactly the same viability). Furthermore, we assume that the alleles are labelled so that $s_1 < s_2$.

It is possible to produce a number of results from the model just specified. We describe the details of these results, and the methods used to derive them, in the Appendix. Here, we give the main results verbally, and we present an illustrative example.

Let p represent the frequency of B_1. Under the assumptions of the model, the frequencies of the three genotypes will always come to a polymorphic equilibrium (so long as the initial value of p satisfies $0 < p < 1$). Thus, a polymorphic equilibrium value of p (i.e. $\hat{p}$) always exists. This value satisfies $0 < \hat{p} < 1$. Furthermore, we can show that, when $\phi = 0$ (obligate asexuality) we have $\hat{p} = 0.5$. This makes sense, since at equilibrium when $\phi = 0$, only the heterozygote is present. Additionally, as any $\phi > 0$ is reduced, $\hat{p}$ becomes closer to 0.5.

Let $\hat{\hat{W}}$ represent the average probability of surviving to reproductive age, once equilibrium is reached. It is straightforward to show that $\hat{\hat{W}}$ is a decreasing function of ϕ. In other words, as the population becomes more sexual, the proportion of offspring that survive to reproductive age becomes smaller.

Although the viability of offspring becomes lower, on average, as the population becomes more sexual, this does not necessarily mean that viability is decreasing for all subgroups among the offspring. In particular, let us divide the offspring into two classes, those that are the result of sexual reproduction, and those that were produced by asexual means. (The frequencies of members of these two classes are ϕ and $1 - \phi$, respectively.) Let $\hat{\hat{W}}_\mathrm{S}$ denote the proportion of sexually produced offspring that survive to reproductive age (at equilibrium). Let $\hat{\hat{W}}_\mathrm{A}$ denote the same quantity for asexually produced offspring.

We can show that $\hat{\hat{W}}_\mathrm{A}$ is always greater than $\hat{\hat{W}}_\mathrm{S}$. Thus, sexually produced offspring are, on average, less viable than asexually produced offspring. However, although $\hat{\hat{W}}_\mathrm{A}$ is always greater than $\hat{\hat{W}}_\mathrm{S}$, this is a statement about averages, and it does not mean that, for any given female, the viability of her sexually produced offspring is expected to be lower than the viability of her asexually produced offspring. For example, consider the offspring produced by a $\mathrm{B}_2\mathrm{B}_2$ homozygote. The expected viability of her sexually produced offspring is actually higher than the viability of her asexually produced offspring. This is because, under our assumption that $s_1 < s_2$, $\mathrm{B}_2\mathrm{B}_2$ is the least viable genotype, and sex can lead to the presence of other genotypes among the offspring.

In the Appendix, we consider how the rate of sexual reproduction (ϕ) affects $\hat{\hat{W}}_\mathrm{S}$ and $\hat{\hat{W}}_\mathrm{A}$. We show that, as ϕ increases, $\hat{\hat{W}}_\mathrm{S}$ also increases, but $\hat{\hat{W}}_\mathrm{A}$ falls (see Fig. 1). Thus, the difference between $\hat{\hat{W}}_\mathrm{A}$ and $\hat{\hat{W}}_\mathrm{S}$ always becomes smaller as ϕ increases, and, similarly, the ratio $\hat{\hat{W}}_\mathrm{A}/\hat{\hat{W}}_\mathrm{S}$ decreases as ϕ increases.

Discussion

The significance of the results presented here depends, in part, on whether or not heterozygote advantage is common. This is a controversial issue, and it has been reviewed elsewhere (Frankel, 1983; Mitton & Grant, 1984; Houle, 1989). However, it is worth noting here that cases of particular loci where the heterozygote seems to confer a higher fitness than either homozygote are well known. Examples occur in humans, rats, insects and plants (Cavalli-Sforza & Bodmer, 1971; Greaves *et al.*, 1977; Mitchell-Olds, 1995; Watt *et al.*, 1996). Furthermore, in most of the known examples, the selective differences between homozygotes and heterozygotes are large. This suggests that there may be many other loci in which heterozygote advantage occurs, but has a modest (and thus difficult to detect) effect.

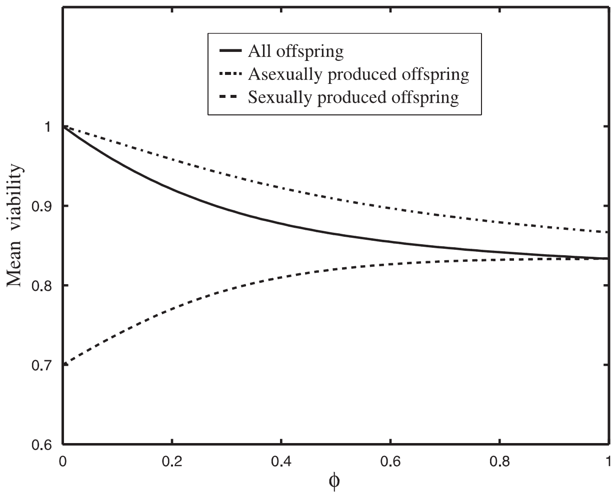

Fig. 1 The proportion of offspring surviving to reproductive age (mean viability) as a function of proportion of offspring produced by sexual means (ϕ). The solid line gives the proportion of all offspring that survive ($\hat{\hat{W}}$), while the dashed line and the line with alternating dots and dashes give the same statistic for sexually and asexually produced offspring (that is, these lines give, respectively, $\hat{\hat{W}}_S$ and $\hat{\hat{W}}_A$). For purposes of this figure, we set $s_1 = 0.2$ and $s_2 = 1$.

In many taxa, a positive association has been found between the overall level of heterozygosity and various indicators of fitness (Mitton & Grant, 1984; Zouros & Foltz, 1987). However, this may not be a result of heterozygote advantage at any particular locus (Houle, 1989). More research is required to clarify the causal relations involved.

Heterozygote advantage may arise when environments vary in time and/or in space (Wallace, 1968; Gillespie, 1991). Furthermore, this can happen even if, at any given time and place, one of the possible homozygotes is the fittest genotype. The advantage to heterozygotes arises because of averaging over environments. Heterozygotes often show greater developmental stability than homozygotes when environments are variable (Mitton & Grant, 1984), and this militates in favour of environmental heterogeneity as a source of heterozygote advantage. In addition, in some cases, there is direct evidence that environmental heterogeneity enhances the relative fitness of heterozygotes (Mitton & Grant, 1984).

Apparent heterozygote advantage can arise if, in one deme, a recessive (or partially recessive) deleterious mutation becomes fixed at a particular locus, while in another deme, another recessive (or partially recessive) deleterious mutation becomes fixed at a locus that is very closely linked to the first locus (Wallace, 1968; Houle, 1989). If these two demes exchange migrants, then there will be apparent heterozygote advantage, at least until recombination creates chromosomes that are free of both of the linked deleterious mutations. If the population is not very large and the loci are very closely linked, then chromosomes that are free of both deleterious recessive mutations might not arise for a long time. Apparent heterozygote advantage of this sort may be very common, particularly in species with structured populations (Frankel, 1983; Houle, 1989; Stuber *et al.*, 1992).

Overall, it seems plausible that, in a wide variety of organisms, there will be at least one or two small regions of the genome where heterozygotes have an advantage. For our purposes, it does not matter much whether heterozygote advantage arises because of the superiority of heterozygotes in a particular environment, or because of environmental heterogeneity, or because of the action of closely linked deleterious mutations. The processes we describe are likely to play a role so long as, for one or more loci, heterozygotes tend to be fitter than homozygotes.

In our model, we assumed that each offspring is produced by sexual means with probability ϕ. However, it is of interest to consider the implications of the results for alleles that affect the rate of sexual reproduction, and that can cause within-population variation in the tendency to reproduce sexually. It seems likely that such alleles exist, as there are a number of organisms that are known to reproduce by both sexual and asexual means, and in some of these, it has been possible to change the rate of sexual reproduction by means of selective breeding (Carson, 1967; Blackman, 1972; Viney, 1996; Parker, 1998).

Imagine a genetic locus that controls the rate of sexual reproduction (ϕ). Alleles at this *sexuality determining locus* will be affected by selective pressures that arise from a number of different sources. For example, sex can have a variety of benefits such as the amelioration of the effects of deleterious mutations, and the facilitation of the incorporation of beneficial mutations (Bell, 1982; Crow, 1988; Kondrashov, 1988; Michod & Levin, 1988; Kondrashov, 1993; Peck, 1993, 1994, 1996; Hurst & Peck, 1996; Peck *et al.*, 1997). Sex can also help in the 'arms race' against coevolving parasites (Hamilton, 1980). These effects should favour alleles that increase the rate of sexual reproduction. On the other hand, an offspring produced by asexual means is guaranteed to have all of the alleles carried by its mother, while a sexually produced offspring will inherit only half of its mother's alleles (by direct descent). This factor favours alleles that reduce the rate of sexual reproduction (Maynard Smith, 1978).

The results presented here suggest another factor that favours alleles that decrease the rate of sexual reproduction. Say that alleles at the sexuality determining locus are in approximate linkage equilibrium with alleles at the selected locus. In this case the average viability of sexually and asexually produced offspring should be the appropriate measures for determining the strength of indirect selection upon alleles at the sexuality determining locus. Our results show that, if viability is determined by a locus that is subject to heterozygote advantage, then, on average, sexually produced offspring will be less fit than asexually produced offspring. This should favour alleles that decrease the rate of sexuality. Furthermore, the effects can be quite strong. To take an extreme example, if $s_1 = 0.990$ and $s_2 = 0.999$, then almost all asexually produced offspring will survive, but about half of sexually produced offspring will die. Furthermore, in this extreme and nongeneric case, the viability of sexually and asexually produced offspring is almost (but not quite) independent of ϕ.

The effects can be even larger if there are multiple loci that are subject to heterozygote advantage, even if each of them is characterized by small selection coefficients. As an example, say that loci combine multiplicatively to determine fitness, and that all loci that determine fitness are approximately in linkage equilibrium. Assume that $\phi \approx 0$, so that very few offspring are produced sexually. Assume further that, at each selected locus, $s_1 = 0.05$ and $s_2 = 0.051$. If there is only one locus under selection, then sexually produced offspring will be about 2.5% less viable than asexually produced offspring. However, if there are 20 selected loci, then sexually produced offspring will be about 40% less viable than asexually produced offspring. If there are 100 such loci, then almost all asexually produced offspring will survive selection, while approximately 92% of sexually produced offspring will perish.

One of the most intriguing aspects of the results is the way that the difference in the viability of sexually and asexually produced offspring changes with ϕ. The average viability of asexually produced offspring is always higher than the average viability of sexually produced offspring, and the difference in viability always increases as ϕ decreases. This means that the strength of the selective pressure produced by heterozygote advantage that favours asexuality becomes stronger as the population becomes more asexual. Furthermore, the low fitness of sexually produced offspring in mostly asexual populations (i.e. in populations with low ϕ-values) may prevent these populations from obtaining the benefits of sex, as sexually produced offspring may be unlikely to produce long-lasting lineages. These factors may often result in the evolutionary instability of intermediate values of ϕ (i.e. instability of mixed sexual and asexual reproduction). In other words, partial asexuality may be a slippery slope for many species. Once the rate of sexual reproduction becomes sufficiently low, the benefits of sex may vanish, leading to the evolution of obligate asexuality.

We believe that the results we have presented for heterozygote advantage can be extended to other types of nonadditive genetic variation. In particular, we expect that the analysis could be extended to epistatic interactions between loci, especially when frequency-dependent selection maintains variance at one or more loci (Barton, 1995; Charlesworth & Barton, 1996). However, confirmation of this expectation must await further analysis.

There is some evidence to suggest that the sorts of transitions in genotype frequencies that we have described here do actually occur in real organisms. Malmberg has shown that, in the bacteriophage T4, epistatic interactions become more common when the rate of sexual reproduction is low (Malmberg, 1977). Evidence that is more directly related to heterozygote advantage comes from the observation that an excess of heterozygotes (in comparison to Hardy–Weinberg expectations) is apparently common in parthenogens (Lokki *et al.*, 1976; Nevo *et al.*, 1984; Gray, 1986). A particularly intriguing example, in this regard, is the diploid cyclic parthenogen, *Daphnia magna*. This species shows an excess of heterozygotes in long-standing populations (Hebert *et al.*, 1982). Because of the occasional bouts of sexual reproduction, the excess of heterozygotes cannot be attributed to hybridization events in the establishment of the species.

The balance of selective forces acting on the mode of reproduction can be expected to differ from one species to another. Thus, for example, sex is less costly when both parents invest heavily in helping offspring to grow to reproductive age (Bell, 1982). In addition, in species that happen to have no segregating alleles that produce heterozygote advantage, and where there are no significant epistatic interactions involving polymorphic loci, there may be little problem with producing a small proportion of offspring sexually, and the rest asexually.

Although the processes we have described here do not preclude the existence of species that produce only a minority of offspring via sexual means, they can be expected to decrease the selective advantage of this sort of lifestyle in many populations. These considerations may help to resolve the mystery concerning the evolution of species in which most reproduction involves sex.

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Appendix

In this Appendix we provide the expressions for quantities referred to in the main text. We then derive these expressions, and provide proofs of our assertions.

We begin with the case where $\phi = 0$ (obligate asexuality). In this case it is straightforward to show that only the B_1B_2 heterozygote is present at equilibrium, and thus, $\hat{\hat{W}} = 1$. For the remainder of the analysis presented in this Appendix, we assume $\phi > 0$.

Let x_1, x_2 and x_3 represent, respectively, the frequencies of the genotypes B_1B_1, B_1B_2 and B_2B_2 among adults. Let us define z as follows:

$$z = x_1 - x_3. \tag{1}$$

Let $\hat{x}_1$, $\hat{x}_2$ and $\hat{x}_3$ represent, respectively, the equilibrium values of x_1, x_2 and x_3, and let $\hat{z}$ represent the quantity $\hat{x}_1 - \hat{x}_3$. Using the assumption that $s_1 < s_2$, we can write $\hat{z}$ explicitly in terms of the parameters of the model as follows:

$$\hat{z} = \frac{(s_2 - s_1)\phi}{(2-\phi)s_1 s_2 + \sqrt{(s_1 + s_2 - s_1 s_2)^2 \phi^2 + 4 s_1^2 s_2^2 (1-\phi)}} \tag{2}$$

We can now write $\hat{x}_1$, $\hat{x}_2$ and $\hat{x}_3$ as

$$\begin{aligned} \hat{x}_1 &= \frac{\hat{z} s_2 (1 - s_1)(1 + \hat{z})}{s_2 - s_1 + \hat{z}(s_1 + s_2 - 2 s_1 s_2)} \\ \hat{x}_2 &= \frac{(s_2 - s_1)(1 - \hat{z}^2)}{s_2 - s_1 + \hat{z}(s_1 + s_2 - 2 s_1 s_2)} \\ \hat{x}_3 &= \frac{\hat{z} s_1 (1 - s_2)(1 - \hat{z})}{s_2 - s_1 + \hat{z}(s_1 + s_2 - 2 s_1 s_2)}. \end{aligned} \tag{3}$$

Let $\hat{p}$ represent the equilibrium value of p (the frequency of B_1). We have

$$\hat{p} = \frac{1}{2}(1 + \hat{z}). \tag{4}$$

In the limit as ϕ goes to zero we have $\hat{z} = 0$, and thus $\hat{z} = ½$. To provide an estimate of $\hat{p}$ when ϕ is small we note that, when $\phi \ll 1$ we have (from eqn 2)

$$\hat{z} = \frac{\phi(s_2 - s_1)}{4 s_1 s_2} + O(\phi^2) \tag{5}$$

and hence

$$\hat{p} = \frac{1}{2} + \frac{\phi(s_2 - s_1)}{8 s_1 s_2} + O(\phi^2). \tag{6}$$

Thus, when ϕ is sufficiently small, $\hat{p}$ is close to ½.

From the assumptions of the model, the equilibrium mean viability, $\hat{\bar{W}}$, is given by

$$\hat{\bar{W}} = \phi \hat{\bar{W}}_{\mathrm{S}} + (1 - \phi)\hat{\bar{W}}_{\mathrm{A}}. \tag{7}$$

The assumptions also lead directly to the following expressions for $\hat{\bar{W}}_{\mathrm{S}}$ and $\hat{\bar{W}}_{\mathrm{A}}$:

$$\hat{\bar{W}}_{\mathrm{S}} = \hat{p}^2(1 - s_1) + 2p(1 - \hat{p}) + (1 - s_2)(1 - \hat{p})^2 \tag{8}$$

and

$$\hat{\bar{W}}_{\mathrm{A}} = (1 - s_1)\hat{x}_1 + \hat{x}_2 + (1 - s_2)\hat{x}_3. \tag{9}$$

One can use eqns 2, 3, 4, 7, 8 and 9 to write $\hat{\bar{W}}$, $\hat{\bar{W}}_{\mathrm{S}}$ and $\hat{\bar{W}}_{\mathrm{A}}$ as explicit functions of the parameters of the model, but the resulting expressions are lengthy, and not obviously useful.

To derive the foregoing quantities, let us begin by defining $q = 1 - p$. We denote the average viability of sexually produced offspring during a particular generation as $\bar{W}_{\mathrm{S}}$, and the average viability for asexually produced offspring is $\bar{W}_{\mathrm{A}}$. The average viability of offspring (without respect to mode of reproduction) is simply $\bar{W}$. From the assumptions of the model, we have

$$\bar{W}_{\mathrm{A}} = (1 - s_1)x_1 + x_2 + (1 - s_2)x_3, \tag{10}$$

$$\bar{W}_{\mathrm{S}} = (1 - s_1)p^2 + 2pq + (1 - s_2)q^2 \tag{11}$$

and $\bar{W} = (1 - \phi)\bar{W}_{\mathrm{A}} + \phi \bar{W}_{\mathrm{S}}$.

Let x_i' represent the value of x_i in the next generation. The assumptions of the model lead to the following expressions:

$$x_1' = \frac{(1 - s_1)[(1 - \phi)x_1 + \phi p^2]}{\bar{W}} \tag{12}$$

$$x_2' = \frac{(1 - \phi)x_2 + 2\phi pq}{\bar{W}} \tag{13}$$

$$x_3' = \frac{(1 - s_2)[(1 - \phi)x_3 + \phi q^2]}{\bar{W}}. \tag{14}$$

Equations 12–14 are not independent, as $x_1 + x_2 + x_3 = 1$. Thus, we need solve only two equations for two quantities. It is convenient to work with the variables x_2 and z. We have

$$\begin{gathered} p = \frac{1 + z}{2}, \quad q = \frac{1 - z}{2}, \\ x_1 = \frac{1 + z - x_2}{2}, \quad x_3 = \frac{1 - z - x_2}{2} \end{gathered} \tag{15}$$

and from this we can write the following expressions:

$$\begin{aligned} \bar{W} = 1 &+ \frac{z(s_2 - s_1)}{2} \\ &- \left(\frac{s_1 + s_2}{2}\right)\left(1 - (1 - \phi)x_2 - \frac{\phi(1 - z^2)}{2}\right), \end{aligned} \tag{16}$$

$$x_2' = \frac{(1 - \phi)x_2 + \frac{\phi}{2}(1 - z^2)}{\bar{W}} \tag{17}$$

and

$$z' = \frac{z\left(1 - \frac{s_1 + s_2}{2}\right) + \left(\frac{s_2 - s_1}{2}\right)\left(1 - (1 - \phi)x_2 - \frac{\phi}{2}(1 - z^2)\right)}{\bar{W}}, \tag{18}$$

where z' is the value of z in the next generation.

Next, we search for $\hat{x}_2$ and $\hat{z}$ by finding values of x_2 and z for which the left and right sides of the preceding two equations are equal. After some algebra, eqns 17 and 18 allow us to write the following expression for $\hat{x}_2$, in which ϕ does not appear:

$$\hat{x}_2 = \frac{(s_2 - s_1)(1 - \hat{z}^2)}{s_2 - s_1 + (s_1 + s_2 - 2 s_1 s_2)\hat{z}} \tag{19}$$

We can use this, along with eqn 18, to get

$$\phi = \frac{4 \hat{z} s_1 s_2 (s_2 - s_1)}{2 \hat{z} s_1 s_2 (s_2 - s_1) + (s_2 - s_1)^2 - (s_1 + s_2)(s_1 + s_2 - 2 s_1 s_2)\hat{z}^2} \tag{20}$$

This leads to a quadratic equation in $\hat{z}$ which has two solutions. However, only one, which is given by eqn 2, corresponds to a valid solution, for which all three equilibrium genotype frequencies are non-negative.

To prove our assertions about the way that the equilibrium values of $\hat{\bar{W}}_{\mathrm{S}}$, $\hat{\bar{W}}_{\mathrm{A}}$ and $\hat{\bar{W}}$ change with ϕ, we must first establish the fact that $\mathrm{d}\hat{z}/\mathrm{d}\phi > 0$. To do this, we use eqn 2 to get

$$\frac{\mathrm{d}\hat{z}}{\mathrm{d}\phi} = \frac{2 \hat{z} s_1 s_2}{\phi \sqrt{(s_1 + s_2 - s_1 s_2)^2 \phi^2 + 4 s_1^2 s_2^2 (1 - \phi)}}. \tag{21}$$

By inspection, we can confirm that this is positive for all values of ϕ that satisfy $0 < \phi < 1$.

Next, we show that $\mathrm{d}\hat{\bar{W}}_\mathrm{S}/\mathrm{d}\phi > 0$. To begin, we note, using eqns 4 and 8, that

$$\hat{\bar{W}}_\mathrm{S} = 1 + \frac{1}{2}\hat{z}(s_2 - s_1) - \frac{1}{4}(s_1 + s_2)(1 + \hat{z}^2), \tag{22}$$

and thus

$$\frac{\mathrm{d}\hat{\bar{W}}_\mathrm{S}}{\mathrm{d}\phi} = \frac{\mathrm{d}\hat{\bar{W}}_\mathrm{S}}{\mathrm{d}\hat{z}}\frac{\mathrm{d}\hat{z}}{\mathrm{d}\phi} = \frac{1}{2}[s_2 - s_1 - \hat{z}(s_1 + s_2)]\frac{\mathrm{d}\hat{z}}{\mathrm{d}\phi}. \tag{23}$$

We know that $\mathrm{d}\hat{z}/\mathrm{d}\phi > 0$, and thus, from eqn 23, we see that $[s_2 - s_1 - \hat{z}(s_1 + s_2)]$ reaches its minimum value when $\phi = 1$. When $\phi = 1$, we have (from eqn 2) $\hat{z} = (s_2 - s_1)/(s_1 + s_2)$, and thus, eqn 23 shows us that $\mathrm{d}\hat{\bar{W}}_\mathrm{S}/\mathrm{d}\phi = 0$ when $\phi = 1$. These observations imply that, for $0 < \phi < 1$, we have $\mathrm{d}\hat{\bar{W}}_\mathrm{S}/\mathrm{d}\phi > 0$.

To show that $\mathrm{d}\hat{\bar{W}}_\mathrm{A}/\mathrm{d}\phi < 0$, we substitute into eqn 9 from eqn 15 to get

$$\hat{\bar{W}}_\mathrm{A} = 1 - \frac{1}{2}(s_1 + s_2)(1 - \hat{x}_2) + \frac{1}{2}\hat{z}(s_2 - s_1) \tag{24}$$

and hence

$$\frac{\mathrm{d}\hat{\bar{W}}_\mathrm{A}}{\mathrm{d}\phi} = \frac{\mathrm{d}\hat{\bar{W}}_\mathrm{A}}{\mathrm{d}\hat{z}}\frac{\mathrm{d}\hat{z}}{\mathrm{d}\phi} = \frac{1}{2}\left(s_2 - s_1 + (s_1 + s_2)\frac{\mathrm{d}\hat{x}_2}{\mathrm{d}\hat{z}}\right)\frac{\mathrm{d}\hat{z}}{\mathrm{d}\phi}. \tag{25}$$

It can be shown, using eqn 19 that $(s_2 - s_1 + (s_1 + s_2)(\mathrm{d}\hat{x}_2/\mathrm{d}\hat{z})) < 0$. Thus, since $\mathrm{d}\hat{z}/\mathrm{d}\phi > 0$ for $0 < \phi < 1$, we have $\mathrm{d}\hat{\bar{W}}_\mathrm{A}/\mathrm{d}\phi < 0$ for $0 < \phi < 1$.

Let us now prove that $\hat{\bar{W}}_\mathrm{S} < \hat{\bar{W}}_\mathrm{A}$ always holds over the interval $0 < \phi < 1$. We have seen that $\mathrm{d}\hat{\bar{W}}_\mathrm{S}/\mathrm{d}\phi > 0$ and that $\mathrm{d}\hat{\bar{W}}_\mathrm{A}/\mathrm{d}\phi < 0$. Thus, we need only show that, in the limit as $\phi \to 1$, we have $\hat{\bar{W}}_\mathrm{S} < \hat{\bar{W}}_\mathrm{A}$. In this limit, we have (from eqns 2, 3, 22 and 24)

$$\hat{\bar{W}}_\mathrm{A} = 1 - \frac{s_1 s_2(s_1 + s_2 - 2s_1 s_2)}{(s_1 + s_2)(s_1 + s_2 - s_1 s_2)} \tag{26}$$

and

$$\hat{\bar{W}}_\mathrm{S} = 1 - \frac{s_1 s_2}{s_1 + s_2}. \tag{27}$$

Thus, in the same limit, we have

$$\frac{\hat{\bar{W}}_\mathrm{A}}{\hat{\bar{W}}_\mathrm{S}} = 1 + \frac{s_1^2 s_2^2}{(s_1 + s_2 - s_1 s_2)^2}. \tag{28}$$

As this ratio is clearly in excess of unity, we have proved that $\hat{\bar{W}}_\mathrm{A} > \hat{\bar{W}}_\mathrm{S}$ always holds for $0 < \phi < 1$.

Finally, we will show that $\mathrm{d}\hat{\bar{W}}/\mathrm{d}\phi < 0$ within the interval $0 < \phi < 1$. Using eqns 3, 7 and 20, we can write $\hat{\bar{W}}$ in terms of s_1, s_2 and $\hat{z}$ as follows:

$$\hat{\bar{W}} = 1 - \frac{2s_1 s_2 \hat{z}}{s_2 - s_1 + (s_1 + s_2)\hat{z}}. \tag{29}$$

Thus,

$$\frac{\mathrm{d}\hat{\bar{W}}}{\mathrm{d}\phi} = \frac{-2s_1 s_2(s_2 - s_1)}{[s_2 - s_1 + (s_1 + s_2)\hat{z}]^2}\frac{\mathrm{d}\hat{z}}{\mathrm{d}\phi}. \tag{30}$$

We know that, for $0 < \phi < 1$ we have $\mathrm{d}\hat{z}/\mathrm{d}\phi > 0$, and thus, since $s_1 < s_2$, we have $\mathrm{d}\hat{\bar{W}}/\mathrm{d}\phi < 0$.