WHY IS A TWO-ENVIRONMENT SYSTEM NOT RICH ENOUGH TO EXPLAIN THE EVOLUTION OF SEX?

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Most of the quantitative study of natural selection concentrates on sexual populations; the conditions under which the sexual system itself has been selected have remained less obvious. The problem of the evolution of sex is expressed acutely in the context of polygynous populations, in which paternal care is almost negligible. Even if courtship and mating are ignored, the fertility of these populations will be half that of similar-sized populations consisting solely of parthenogenetic females.

Williams (1975) termed this drop in individual productivity the "cost of meiosis." This cost is customarily approximated by two ("twofold disadvantage of sex," Maynard Smith 1978), although it may be any number greater than one, depending on the specific population. (a may be regarded as the ratio of the self genes transferred to the next generation by an average parthenogenetic female to those transferred by an average sexual female. Hence, a is the ratio of the fitness of an asexual individual to that of a sexual one.)

This apparent disadvantage of sex at the individual level led to the development of the group-selection approach, which tried to explain sex on the grounds of its benefit to the population as a whole (see Fisher 1930; Muller 1932; Crow and Kimura 1965).

Even when the controversial mechanism of group selection is invoked (Williams 1966), however, sexual reproduction reduces, rather than raises, the average fitness of a sexual population under constant selection pressure at equilibrium. Thus, rather than accelerating evolution, sex apparently slows it, at least when the rate of evolution is measured in terms of the adaptation to a given environment (see Maynard Smith 1968; Eshel and Feldman 1970; Eshel 1971, 1972). This is all the more true with regard to individual selection in such an environment; it seems to be selectively disadvantageous to break up coadapted genotypes of a surviving adult by recombination (see Feldman 1972; Feldman et al. 1980).

Consequently, many models that assume a changing (or rather fluctuating) environment were devised (Williams 1975; Maynard Smith 1978; Hutson and Law 1981). In most of them the problem of the existence of sex was identified, either
explicitly or implicitly, with that of recombination. Hence, the characterization of
a mechanism by which recombination compensates for the cost of meiosis was
pursued. The application of such an approach, however, would be incomparably
more difficult than solving the original problem, since the advantage of recom-
bination in a sexual population is in no way obvious (see, e.g., Feldman 1972).
This may be one explanation for the unreasonable, intense selection in popu-
lations with low fertility (where sex seems especially prevalent) indicated
by many of these models. Another difficulty in some of the models was
overspecificity, as implied by restrictive assumptions about the environment’s
parameters.

Another group of theories that has recently received attention invokes the biotic
environment as the driving force behind the maintenance of sex in populations.
Biotic interactions, such as those with competitors, predators, or parasites, are
characterized by greater uncertainty and dynamism than changes in temperature
or other physical features (see Levin 1975; Clarke 1976; Jaenike 1978; Hamilton
1980). Thus, “asexual populations, when compared with their sexual relatives,
tend to occur . . . in disturbed rather than undisturbed habitats—each of these
trends is consistent with the notion of relatively greater ‘biological accommoda-
tion’ ” (Glesener and Tilman 1978, p. 661).

The suggested mechanism relies on antagonistic interactions among different
species. Species A is a component of the environment of its antagonists; thus, it
acts as a selective force on them. In addition, the force generated by frequent
phenotypes of A will be stronger than that of rare ones. Thus, species A causes its
biotic environment to deteriorate more for its frequent phenotypes than for its rare
phenotypes (see Law and Lewis 1983).

On this basis, several explanations have been proposed for the advantage of sex
in an environment characterized by intensive biotic interactions: frequency-
dependent selection by parasites (Jaenike 1978); pathogen transmission between
relatives to select for sexual recombination (Tooby 1982; Rice 1983); and a zero-
fitness model (Treisman 1976).

Several genetic models were constructed by Hamilton and others (see Hamilton
approach assumed antagonistic coevolution between a parasite and its host or a
predator and its prey. Such an interaction can generate a cyclic process of
frequency-dependent selection.

In their examination of symmetrical environments, Hamilton et al. concluded
that “the most hopeful patterns of symmetry in fitness coefficients force condi-
tions where stability implies that alleles must have LGMF [‘cost of sex’ here] of
unity” (1981, p. 369), meaning that sex can never be beneficial in these cases.
(However, they proposed some special asymmetrical models that can give sex an
advantage. This issue is further discussed in the Appendix.) Their results seemed
to reconfirm the common assumption that recombination, and not reshuffling of
gametes, is the main effect of sex. “In the light of such examples it appears
unlikely that a viable one-locus model for maintenance of sex based on fluctuation
of environment can be devised. This at least gives some cool comfort with regard
to traditional views of sex. If a one-locus model had proved plausibly ‘sufficient,’
an equally important problem would be left outstanding, that of explaining the near universality of crossing-over.” (Hamilton et al. 1981, p. 371.)

This line of thought rests on an implicit assumption that the simple case of two alleles segregating in one locus reflects the general case of \( n \) such alleles. A similar assumption has guided most of those who have considered the problem of sex: “Of course, each feature could exist in more than two states, but I do not think this would make any essential difference to the conclusions” (Maynard Smith 1978, p. 90). Surprisingly, it makes a great difference.

In this paper I present a simple and natural model of \( n \) hostile environments (which could be interpreted as \( n \) parasites), wherein each different environment has a corresponding allele that immunizes its carrier against it. The population considered is divided into sexual and asexual individuals from all possible genotypes, differing only in their reproduction pattern (possibly defined by a different locus).

I show that for \( n = 2 \) a sexual variant can never evolve, whatever choice of parameters is made. I then show that for \( n \geq 3 \) an evolution of sex is possible with an essentially smaller cost of evolution than shown previously. To be more precise, the extinction of 70% of the population will be required if the catastrophes (the hostile environments) occur in a cyclic order. Nevertheless, it seems that an advantage to sex can also be obtained in a more moderate case, one in which the population experiences a few generations of peace between one catastrophe and the next. If a slight heterozygote selection in those “peaceful generations” is also assumed, sex is found to be beneficial when as little as \( \% \) of the population perishes in each catastrophic generation. This value is much smaller than that required in previous models.

A SIMPLE SYMMETRICAL MODEL

I now examine the conclusion that sex cannot be advantageous when a single-locus model is considered and a “natural” (i.e., symmetrical) selection pressure is assumed (Hamilton et al. 1981). To this end, a deterministic model of a single locus, \( n \) alleles, and a symmetrical selection is employed. We start with the case \( n = 2 \), considered by Hamilton et al.

The Case for Two Alleles

Assume a diploid sexual population with two segregating alleles denoted \( A \) and \( B \), and two “seasons” denoted \( A \) and \( B \), wherein the corresponding allele \( A \) or \( B \) is beneficial. The seasons are completely symmetrical with regard to their effect on the two different homozygotes, and appear alternately.

More specifically, assume the fitness coefficients of table 1, for \( \gamma \), \( \beta \), and \( \delta \) nonnegative real numbers. (Henceforth, \( \delta = 1 \) is assumed, without any loss of generality.)

The further assumption that \( \gamma \gg 1 \gg \beta \) would imply a tremendous advantage to each homozygote in one season against a tremendous disadvantage in the other season, and the heterozygote seems always inferior to one of them. This interpretation suggests, at least at first sight, an advantage to those individuals who
can produce offspring genetically different from themselves, namely, sexual individuals.

However, in their analysis of a similar model, with the restriction \( \gamma = \beta^{-1} \), Hamilton et al. (1981) found that the sexual population cannot compete with the asexual one, as long as the asexual population has any positive advantage, however small (i.e., as long as the cost of sex is greater than one). It can be shown quite easily that the same result generally holds for any \( \gamma \) and \( \beta \) (for the deterministic case).

Let \( p \) denote the frequency of allele \( A \) at the beginning of season \( A \), and \( q = 1 - p \) denote the frequency of allele \( B \) at that time. After one generation (of season \( A \)), the frequencies of alleles \( A \) and \( B \) will be \( f_A(p) \) and \( 1 - f_A(p) \), respectively, where \( f_A \) is the transformation corresponding to the changes in allele frequency resulting from viability selection in a sexual population, with the selection coefficients as given in the first column of table 1. Then, \( f_A(p) = (\gamma p^2 + pq)/(\gamma p^2 + 2pq + \beta q^2) \) for \( p \) and \( q \) defined as above. The same is true for \( f_B(p) = (\beta p^2 + pq)/(\beta p^2 + 2pq + \gamma q^2) \). To find necessary and sufficient conditions for a stable polymorphism, we seek an internal fixed point of the transformation \( f_B \circ f_A \), or \( f_{B-A} \).

Applying considerations of symmetry, such a point satisfies the equation \( q = f_A(p) \). Accordingly, at equilibrium, the frequency of allele \( A \) at the end of season \( A \) equals that of allele \( B \) at the end of season \( B \). Hence,

\[
q = (\gamma p^2 + pq)/(\gamma p^2 + 2pq + \beta q^2).
\]

Substituting \( x = p/q \),

\[
F(x) = \gamma x^3 + x^2 - x - \beta = 0 \quad \gamma, \beta > 0.
\]

Since \( F(0) = -\beta < 0 \) and \( \lim(x \to \infty)F(x) = \infty \), this equation has at least one root in the range \( x > 0 \), whose uniqueness can easily be verified. The root corresponds to the frequency \( p \) at equilibrium if this polymorphism is stable, which is the case if the condition \( f'_{B-A}(0) > 1 \) is satisfied (owing to the symmetry). Thus,

\[
\frac{df_{B-A}(x)}{dx} \bigg|_{x=0} = \frac{df_B(x)}{dx} \bigg|_{x=0} = \frac{1}{\gamma} > 1.
\]
We have, then, the very simple condition $\gamma \beta < 1$, which can be interpreted as a superiority of heterozygotes in geometric mean over the generations. This condition is necessary for the maintenance of a stable polymorphism in a sexual population.

Given this condition, the sexual population is not stable against an asexual invading mutant, at least not when it occurs in the heterozygote $AB$. To illustrate, let $\epsilon > 0$ be the initial frequency of such a mutant clone. To test how one cycle of selection ($B \circ A$) influences its frequency (neglecting the possibility of an appearance of new mutant individuals within the sexual population), let

$$\epsilon' = \frac{\epsilon}{1 + \epsilon \bar{w}_A(p) \bar{w}_B(p)},$$

where $\bar{w}_A(x) [\bar{w}_B(x)]$ measures the mean fitness of the sexual population (relative to that of the asexual one) at season $A [B]$ for frequency $x$ of the beneficial allele, and $p$ is the equilibrium frequency of the beneficial allele.

Equation (4) implies the assumption that both populations have the same fertility, which is like assuming the cost of sex is one. Because $\bar{w}_A(p) = \bar{w}_B(p)$ is symmetrical at equilibrium, we obtain a necessary condition for sexual advantage,

$$\bar{w}_A(p) > 1. \quad (5)$$

However, $q = (\gamma p^2 + pq)/\bar{w}_A(p)$ if and only if

$$\bar{w}_A(p) - 1 = (1/q)(\gamma p^2 + pq - q) = (1/q)(\gamma p^2 - q^2).$$

Hence,

$$\bar{w}_A(p) - 1 > 0 \quad \text{if and only if} \quad \gamma p^2 - q^2 > 0 \quad \text{if and only if} \quad \gamma x^2 > 1, \quad x = p/q,$$

which yields

$$F(x) = x(\gamma x^2 - 1) + x^2 - x - \beta > 0 + 1/\gamma - \beta = (1/\gamma)(1 - \gamma \beta) > 0, \quad (7)$$

in contradiction to $F(x) = 0$. Hence, condition (5) cannot be true for any $\gamma, \beta > 0$. Thus, sex will never be advantageous for any value of the fitness coefficients in a symmetrical model, however low the cost. Notice that in the special case of $\beta = 0$, $\gamma \beta < 1$ is true for all $\gamma$; that is, a stable polymorphism always exists in an inferior sexual population.

Thus, the results of Hamilton et al. (1981) seem to have been confirmed somewhat more rigorously, for the symmetrical case. (For the analysis of some different one-locus and two-segregating-alleles models, partly treated by Hamilton et al., see the Appendix.)

The Case for Three Alleles

Before a multilocus model is considered, the initial assumption that taking $n$ equal to two has not caused any loss of generality should be questioned. Assume, for simplicity, $\beta = 0$, which guarantees the existence of a stable polymorphism for all values of $\gamma$. Next, consider three alleles, $A, B, C$, and three seasons, $A, B, C$,
interpreted as before. We assume the fitness coefficients of table 2 (an immediate
generalization of table 1 for $\beta = 0$).

In the present model, unlike the two-allele case, each genotype has zero fitness
at some time. In other words, an asexual clone will never survive long enough to
be noticed, regardless of its means of reproduction. However, sex enables the
population to overcome this infinite series of catastrophes. For example, consider
an individual of the genotype $AA$. One possible way for it to attain a great-
grandson of the same genotype is demonstrated in table 3. It turns out, then, that
the case of two segregating alleles is rather exceptional, whereas the results for
three segregating alleles (or, as shown later, any number greater than two) is
qualitatively different. Although the consideration of two alleles shows sex to be
disadvantageous whatever its cost, when more than two are considered, it is
beneficial at any price (at least when every genotype comes close to zero fitness at
some time).

Note that there is no magic in the number two in this context. It arises here
because we are dealing with diploid creatures. The results above can be general-
ized to the $n$-ploidy case without much difficulty, to yield a resistant genotype
$A_1A_2\ldots A_n$ to $n$ alternating seasons (if each season, once again, has one and only
one corresponding resistant allele), whose asexual mutant will probably be advan-
tageous (or, at any rate, always viable). The number of alternating seasons to yield
an absolute superiority for sex should be not less than $n + 1$.

A MULTISEASONAL MODEL

The example for three alleles discussed above is characterized by two special
features: the population is exposed to a "catastrophe" every generation; and the
catastrophes appear in a regular fixed cycle. The model presented in this section,
in addition to generalizing the number of seasons, also weakens these (and other)
assumptions.

Consider a single locus with $n$ segregating alleles, denoted $A_1, \ldots, A_n$. Let $S =
\{S_i\}_{i=0}^? be such that $S_0$ is a neutral season ("rest season"), meaning either that
no selection operates on the locus in this season or that a slight heterozygote

<table>
<thead>
<tr>
<th>Genotype</th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
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<tbody>
<tr>
<td>$AA$</td>
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</tr>
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<tr>
<td>$AC$</td>
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</tr>
<tr>
<td>$BB$</td>
<td>0</td>
<td>$\gamma$</td>
<td>0</td>
</tr>
<tr>
<td>$BC$</td>
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<td>1</td>
</tr>
<tr>
<td>$CC$</td>
<td>0</td>
<td>0</td>
<td>$\gamma$</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Season</th>
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<th>B</th>
<th>C</th>
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</thead>
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<td>$\times$</td>
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<td>$\times$</td>
<td>$AC$</td>
</tr>
<tr>
<td>A</td>
<td>$AA$</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
selection does (giving a slight benefit to any heterozygote $A_iA_j, i \neq j$). Thus, for $k \geq 0$,

$$\omega(S_0) = \begin{cases} 
1 + k & A_iA_j, \; i \neq j \\
1 & A_iA_i
\end{cases}$$

In addition, $S_i, 1 \leq i \leq n$, is a hostile season (often called "catastrophe"), in which individuals not carrying the allele $A_i$ (i.e., that are not of the genotype $A_iA_j, 1 \leq j \leq n$) survive with probability $\delta$, where $0 \leq \delta \leq 1$. Thus,

$$\omega(S_i) = \begin{cases} 
\delta & A_jA_k, \; j, k \neq i \\
w_2 & A_iA_j, \; j \neq i \\
w_1 & A_iA_i
\end{cases}$$

where $w_1, w_2 \gg \delta$.

Applying the concept of an irreducible Markov chain to the set of states $S$, the biological interpretation of this chain will be the occurrence of season $S_i, 0 \leq i \leq n$, every generation, with a positive probability that each season will occur at some time after any other season. In such a process, the absolute advantage of sex (whatever its cost) is obvious, given $\delta = 0$ and assuming $n \geq 3$. This is because no single individual can possibly be immune to more than two sorts of catastrophes while carrying at the most two different alleles. Hence, in a mixed population of sexual and asexual individuals, no asexual individual can survive three different catastrophes. By contrast, because of the exchange and rearrangement of genetic material, sexual individuals may have viable descendants after any number of generations.

Two quantitative aspects crucial for the applicability of this argument to real populations remain to be studied. First, how sensitive is the argument to a relaxation of the extreme assumption of $\delta = 0$ (certain death of the unfit)? Second, what is the "cost of evolution" required to keep the advantage of sex? In other words, what proportion of the sexual population must die in each generation if such a process is realized? (Following Haldane's terminology, the cost of evolution can be measured by the genetic load $L = (w_{\text{max}} - \bar{w})/w_{\text{max}}$, where $\bar{w}$ is the mean fitness of the population at any instance and $w_{\text{max}}$ is the fitness of the fittest individual.) To answer these questions, let us consider the following cases.

1. In a deterministic model, the catastrophes $S_i, 1 \leq i \leq n$, appear one after the other in a cycle of length $n$, with $k_i$ rest seasons between each season and the next. In this context, a rest season is a generation in which no selection operates on the specific locus. The example presented for three alleles is such a case, where $n = 3$ and $k_i = 0$ for all $i$. Assume that the population is large enough that genetic drift in those rest seasons is negligible; that is, the frequency of the alleles does not alter between one catastrophe and the next.

2. In a probabilistic model, the frequency of the catastrophes is low (under the same restriction mentioned above), and there exists a slight heterozygote selection in the rest seasons. As a result, the allele frequency approaches the uniform distribution $(\nu_n, \ldots, \nu_n)$ during the rest period between two consecutive catastrophes.

Furthermore, for the sake of simplicity and clarity, all calculations are per-
formed for a three-allele case \((n = 3)\). This does not limit the generality of the results, but the actual intensity of selection for a case with \(n > 3\) should be higher than that found in the present case. In addition, a perfect symmetry of all the alleles is assumed.

The Zero-Fitness Case

We start with \(\delta = 0\). Consider first the deterministic case. The fitness coefficients in table 4 can adequately describe the model. Polymorphism is always maintained in this model, as can easily be verified. (A more rigorous proof is provided below.)

The frequencies at season \(\text{S}_1\) of the alleles \(A_1, A_2, A_3\) are denoted \(q_1, q_2, q_3\), respectively; the transformation of the frequencies at the first season \(\text{S}_1\) are denoted \(f_i(q_j)\). At equilibrium, applying symmetry considerations,

\[
\begin{align*}
 f_1(q_1) &= q_3, & f_2(q_2) &= q_3, & f_3(q_3) &= q_1. \\
 q_1 &= f_2(q_2) = q_2/[2 - (1 - h)q_1] - q_2 = q_1[2 - (1 - h)q_1] \\
 q_2 &= f_3(q_3) = q_3/[2 - (1 - h)q_1] - q_3 = q_1[2 - (1 - h)q_1]^2 \\
 q_1 + q_2 + q_3 &= 1.
\end{align*}
\]

(8)

(This means, for example, that the frequency of allele \(A_2\) at season \(\text{S}_2\) equals that of \(A_1\) at season \(\text{S}_1\) at equilibrium.)

The mean fitness of the population will be

\[
\bar{w} = q_1(1 + h) + 2q_1q_2 + 2q_1q_3 = q_1[2 - (1 - h)q_1].
\]

(9)

In order to estimate this quantity as a function of the single parameter \(h\), the following set of equations, which are the explicit form of equation (8), should be solved:

\[
\begin{align*}
 q_1 &= f_2(q_2) = q_2/[2 - (1 - h)q_1] - q_2 = q_1[2 - (1 - h)q_1] \\
 q_2 &= f_3(q_3) = q_3/[2 - (1 - h)q_1] - q_3 = q_1[2 - (1 - h)q_1]^2 \\
 q_1 + q_2 + q_3 &= 1.
\end{align*}
\]

(10)

After some manipulations, this gives

\[
q_1(h) = (1/[1 - h]) \{\sqrt{3} + [\sqrt{2}K - h - 3\sqrt{2}/h]^{1/3} + \sqrt{2}K - h - 3\sqrt{2}/h]^{1/3}\}^{1/3}
\]

(11)

for

\[
K = [(1 - h)/[1 - h]] \left[(h + 3\sqrt{2}/2)^2 - 16/27\right]^{1/2}.
\]
where \( q_1(h) \) is the unique root when \( h \geq -\frac{22}{27} \). It can be shown that \( q_1(h) \) is a decreasing function of \( h \) in the proper range and, moreover, that \( q_1(h) > 0 \) always. After the proper substitution, \( \tilde{w}(h) \) can now be calculated (it can also be shown that it is a decreasing function of \( h \), as well).

To sum up, consider the values in Table 5 for the genetic load \( L \), as described above. One pleasing result is that, for a wide range of the parameter \( h \) corresponding to a heterozygote advantage \( (h < 0) \), the genetic load \( L \) remains fairly close to 0.7. This value is relatively low, allowing the survival of about 30% of the population after each catastrophe.

Consider next the probabilistic model. As stressed earlier, at the outburst of each catastrophe, the vector of allele frequency in the population is rather close to \((\frac{1}{3}, \frac{1}{3}, \frac{1}{3})\). Repeating the above calculations obtains the results in Table 6 \( (q_1 = \frac{1}{3}) \). Hence, once more, a rather low cost of evolution is implied. When heterozygote selection exists \( (h < 0) \), \( L \) is approximately \( \frac{1}{2} \), allowing the survival of half of the population after each catastrophe.

### The General Case

Assume \( \delta > 0 \). Because the advantage of sex ceases to be so immediate or independent of its cost, we must check the stability of the model against a cost of sex, \( \alpha \). Only the deterministic model will be examined. Since the general case is a perturbed version of the zero-fitness case, the above results should hold for small \( \delta \).

Notice, first, that if \( \delta \) is introduced into equation (10), subject to the constraint imposed to keep the advantage of sex, continuity with respect to \( \alpha \) and \( \delta \) is maintained. Thus, the existence of a polymorphism point for any \( \alpha \) is implied if \( \delta \) is small enough (since that is the case for \( \delta = 0 \)).

Next, the stability of this polymorphism is examined. The frequency of \( A_1 \) after one cycle of catastrophes is denoted \( f_q(q_1, q_2) \), and that of \( A_2 \) is denoted \( f_q(q_1, q_2) \), for the initial frequencies \( q_1 \) and \( q_2 \), respectively. The partial derivatives near a boundary point \( q_1, q_2 = 0, \tau (0 \leq \tau \leq 1) \) would be

\[
\frac{\partial f_{q_1}(q_1, q_2)}{\partial q_1} \bigg|_{(0, \tau)} = (1 - \delta)q_1^{\text{H}} + \delta \left(1 - \delta\right)q_1^{\text{H}} + \delta \frac{1}{\delta},
\]

\[
\frac{\partial f_{q_2}(q_1, q_2)}{\partial q_2} \bigg|_{(0, \tau)} = 0,
\]

### Table 5

<table>
<thead>
<tr>
<th>( h )</th>
<th>( q_1 )</th>
<th>( \tilde{w} )</th>
<th>( \omega_{\max} )</th>
<th>( L )</th>
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<tr>
<td>(-\frac{22}{27})</td>
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<td>2</td>
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### Table 6

<table>
<thead>
<tr>
<th>( h )</th>
<th>( \tilde{w} )</th>
<th>( \omega_{\max} )</th>
<th>( L )</th>
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<tr>
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<td>0.67</td>
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</table>
where \( q^{H}_{2} \) and \( q^{H}_{3} \) are the induced frequencies of the alleles \( A_2 \) and \( A_3 \) in the seasons \( S_2 \) and \( S_3 \), respectively (starting from \( q_1 = 0 \) and \( q_2 = \tau \)), and \( \omega(x) = [h - (1 - \delta)x^2 + 2(1 - \delta)x + \delta \]. Hence, a sufficient condition for a protected polymorphism (i.e., when all boundary equilibria are unstable) is \( \partial f_{A}(q_1, q_2)/\partial q_1 \mid (0, \tau) > 1 \). However,

\[
[(1 - \delta)x + \delta]/\omega(x) =
\]

\[
\frac{(1 - \delta)x + \delta}{[h - (1 - \delta)x^2 + 2(1 - \delta)x + \delta]} \xrightarrow{\delta \to 0} \begin{cases} 
1 & x = 0 \\
\frac{1}{2 + (h - 1)x} & 0 < x \leq 1 
\end{cases}
\]

We have, therefore, \( \lim(\delta \to 0)[\partial f_{A}(q_1, q_2)/\partial q_1] \mid (0, \tau) = \infty \) for all \( 0 \leq \tau \leq 1, h > -1 \), which implies the existence of a protected polymorphism if \( \delta \) is small. In the special case in which \( \tau = 0 \) or \( \tau = 1 \) (\( q^{H}_{2} = 0 \) and \( q^{H}_{3} = 1 \) or vice versa), the sufficient condition becomes the simple condition \( \delta(1 + \tau) < 1 \), that is, heterozygote superiority in the geometric mean over the three seasons. Such an advantage is required to prevent fixation of one of the three alleles.

Notice that when \( \delta = 0 \), a stable polymorphism is always maintained, since \( \partial f_{A}(q_1, q_2)/\partial q_1 = \infty \) for all values of \( \tau \). A more detailed inspection also reveals that the intensity of selection implied by the model decreases as a function of \( \delta \). Moreover, the analysis remains valid for any initial frequency of the asexual mutants.

**SEX IN HAPLOID POPULATIONS**

Until now, the discussion has disregarded the recombination effect, emphasizing multiple allelism. This, however, may not always be justified, since the single-locus models cannot apply to organisms such as mosses or haploid yeasts, which spend most of their lives as haploids. The aforementioned models may be applied to primary haploid populations by making the following natural modifications.

Assume two loci with \( n \) alleles segregating in each. Let us use a similar notation for these alleles: \( A_1, \ldots, A_n \) for the first locus, \( B_1, \ldots, B_n \) for the second. Next, consider an environment characterized by the random appearance of one of \( n + 1 \) distinct seasons. Among those, as in the previous model, \( S_0 \) is a rest season and \( S_i \), a catastrophe, wherein only the carriers of an allele resistant to the current selecting force, either \( A_i \) or \( B_i \), survive \( (1 \leq i \leq n) \). Hence, the following fitness coefficients (as a function of the season) are assumed:

\[
\omega(S_0) = \begin{cases} 
1 + k & A_iB_j, \quad i \neq j \\
1 & A_iB_i 
\end{cases}
\]

\[
\omega(S_i) = \begin{cases} 
0 & A_kB_j, \quad k \neq i, \quad j \neq i \\
1 & A_kB_j, \quad k = i \mbox{ or } j = i \\
1 + h & A_iB_i 
\end{cases}
\]

for \( 0 \leq k \) and \( 1 \leq i \leq n \).
This is essentially the same model as that presented for the single locus and \( n \) alleles. That is to say, no asexual individual can survive more than two catastrophes, whereas the sexual individual does survive, because sex along with recombination recreates soon-to-be-needed gene combinations that are currently nonexistent. Surely this argument does not hold if only two segregating alleles are considered at each locus.

Alternatively, consider three loci, each having two segregating alleles, denoted \((A, a)\), \((B, b)\), and \((C, c)\), respectively. Next, consider an environment in which one of three seasons occurs in each generation: \(S_0\), a rest season; \(S_1\), where only gametes of the type \(AB, BC, \) or \(AC\) survive; and \(S_2\), where only gametes of the type \(ab, bc, \) or \(ac\) survive. Once again, no asexual creature can survive both catastrophes, whereas a sexual one can because of recombination.

**DISCUSSION**

The general \( n \)-allele model may be shown to be plausible by means of a natural interpretation, that is, resistance to parasites as described above, when a carrier of each allele is resistant to a different type of parasite. The examples provided by May and Anderson in their description of some known parasite-host relations in mice seem to fit the conditions of the present model exactly: "Innate resistance in mice sometimes appears to be controlled by a single autosomal gene, segregating for incomplete dominance. . . . The genes for acquired resistance against specific infectious agents usually exhibit partial or incomplete dominance, with the heterozygote adopting a position intermediate between the very resistant hosts and the susceptible ones." (1983, p. 283.)

However, the two more special cases described and analyzed in the multiseason model may be interpreted almost as easily. In the first case, the catastrophes appear in a cyclic order of appearance with intermediate quiet seasons (of no selection). This may be justified by frequency-dependent selection and the ensuing antagonistic coadaptation between parasite and host, as explained above. In short, the most probable catastrophe in any generation is the one that has occurred least recently, since the frequency of its resistant allele is the lowest. In the second case, the catastrophes appear at random, very rarely, with intermediate seasons of slight heterozygote selection. In this case, the parasites are moderately active all the time, causing a constant selection in favor of the more resistant heterozygotes. In terms of our model, this constant "background" selection represents the rest seasons. A catastrophe, then, is the random occasional outburst of one of the parasites.

These models demonstrate three points. First, a stable polymorphism can be maintained even though the fitness of the heterozygotes is sometimes close to zero. Second, a very low fitness would be natural in the total absence of any allele resistant to the current selecting force. Third, each heterozygous genotype is crucial for the survival of the population because it preserves genes that will be needed soon. Thus, only with sex (or, in primarily haploid populations, with recombination) can a genotype disappear and then be recreated; this process may sometimes be vital for the population's and the individual's resistance.
In all these models the evolution of sex results solely from the effect of the reshuffling of gametes in diploid organisms or, alternatively, from recombination in primarily haploid organisms, under seemingly natural conditions and with a reasonable genetic load. It may be that the combination of these effects in sexual creatures is even more effective.

One more point should be raised. Under the conditions of the model, an asexual n-ploid organism is the most fit in an environment characterized by \( n \) such catastrophes, but it will become extinct if their number exceeds \( n + 1 \). In this context, sex carries two benefits. It obviates n-ploidy (or the superfluous duplication of genes), an expensive mechanism by itself. And it serves as a solution to an uncertain environment that can be characterized by an arbitrary number of "seasons," with no upper limit. This result is supported in a number of species by the empirical connection between parthenogenesis and polyploidy.

**SUMMARY**

In a single-locus symmetrical model, sexual reproduction has been shown to have an immediate absolute advantage over parthenogenesis in a fluctuating environment. This environment has been described as a certain lottery over \( n + 1 \) possible seasons (where \( n \geq 3 \)): \( n \) hostile seasons, for each of which the individual has a corresponding resistant allele (in the same locus) without which the individual would almost surely die in that same season; and one additional season, usually the most frequent one (and, hence, actually the normal case), in which either no selection or a very moderate one operates on the locus. This single-locus model is modified to apply to primarily haploid populations.

Two extreme cases of the model are analyzed: in one, the order of appearance of the hostile seasons is fixed (to create a cycle); in the other, they appear very rarely and randomly with intermediate seasons of moderate heterozygote selection. Thus, the following properties are demonstrated.

1. The benefit of the sexual population is maintained (as long as \( n \geq 3 \)), whatever the cost of sex may be. Hence, the model can serve as an explanation for the stability of sex when its cost is greater than two (e.g., if the cost of searching for a mate or courting is considered). It may also give some hint about the origin of sex, since the cost of evolving such an adaptation must be in orders of magnitude greater than only the cost of its maintenance.

2. For a stable polymorphism in only one locus, it is shown that sex offers a substantial advantage from the reshuffling of gametes alone. The advantage is measured in terms of the change in the frequencies of sexual and asexual reproduction resulting from natural selection on the individual level. Thus, the evolution of recombination, traditionally identified with the evolution of sex, may now be treated separately (which somewhat reduces the complexity of this last problem, though it does not eliminate it).

3. It is demonstrated that a relatively low cost of evolution is required to sustain the advantage of sex. In its most acute form, the model implies a cost of about 70% at each catastrophe (i.e., the extinction of 70% of the population), which occurs occasionally. In its least extreme form, the model implies a cost of
only 30%-45% at catastrophe time, whereas the assumptions of the model seem only more natural. If only partial extinction is allowed in each catastrophe, the advantage of sex is still maintained if the catastrophe is sufficiently intense. In this case, the cost is still lower than the figures mentioned above. This relatively low cost of evolution overcomes a major difficulty with most of the previous works on the subject by allowing the application of the model to low-fertility populations.

4. A natural application of the model may be to the interaction of host and parasite, following Jaenike (1978), Hamilton (1980), and others. Though a specific ecological model has not been investigated, a plausible example of the conditions of the model may be a sequence of random sudden outbursts of different parasites.

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APPENDIX

AN ASYMMETRICAL TWO-LOCUS MODEL

Consider the model of Hamilton et al. (1981), which describes two alleles segregating at one locus and exposed to a selection that varies over time, as described by the fitness coefficients in table A1. Multiply the viability of an asexual individual by $M_1$ and $M_r^{-1}$, respectively. The two environments also appear with equal probability.

This model was simulated to show a possible stability of sexual reproduction when the value of $r$ is high enough. It was considered less “natural” because of its asymmetrical structure. All the same, let us consider a more general asymmetrical model from which we can obtain analytically some of the results of the aforementioned computer simulations, as a special case. Our motivation for doing so is its relative simplicity mathematically, which enables us to obtain a complete mathematical solution and a detailed analysis, beyond that performed for the general symmetrical model with three segregating alleles. It should be kept in mind, however, that the analytic solution is limited to the deterministic model, in which the two seasons appear alternately. (By contrast, Hamilton et al. simulated a more general case, in which the seasons appeared at random with equal probability.) Yet, the present analysis is expected to reflect quite reliably the general stochastic process.

Using a similar terminology, define a set $S = \{S_0, S_1, S_2\}$, where $S_0$ is a rest season as defined before, at $S_1$ the heterozygotes suffer an almost total extermination, and at $S_2$ both homozygotes confront the same fate. Thus, assume the fitness coefficients of table A2.

Because $A_1$ and $A_2$ are symmetrical, $p = q = 1/2$ may be the only possible equilibrium point, where $p$ denotes the frequency of allele $A_1$ in the population and $q = 1 - p$ denotes that of $A_2$. The mean fitness of the population would be

$$\bar{w}_{S_0} = p^2 + q^2 + 2pq, \quad \bar{w}_{S_1} = 3(p^2 + q^2) + 2pq.$$ 

Assume, once more, a deterministic model, in which the seasons $S_1$ and $S_2$ occur alternately, with intermediate periods of the rest season $S_0$. In order to validate the stability of the proposed polymorphism point, consider the following necessary condition for equilibrium, after one cycle is complete:

$$f(p) = \frac{5p(p + 8q)^2 + q(p + 8q)(q + 8p)}{5[p(p + 8q)^2 + q(q + 8p)^2] + 2pq(p + 8q)(q + 8p)} = p.$$
TABLE A1
FITNESS COEFFICIENTS FOR THE MODEL OF HAMILTON ET AL. (1981)

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Environment A</th>
<th>Environment B</th>
</tr>
</thead>
<tbody>
<tr>
<td>gg</td>
<td>( r )</td>
<td>( r^{-1} )</td>
</tr>
<tr>
<td>gG</td>
<td>( r^{-1} )</td>
<td>( r )</td>
</tr>
<tr>
<td>GG</td>
<td>( r )</td>
<td>( r^{-1} )</td>
</tr>
</tbody>
</table>

TABLE A2
FITNESS COEFFICIENTS FOR THE TWO-ALLELE ASYMMETRICAL CASE

<table>
<thead>
<tr>
<th>Season</th>
<th>( S_0 )</th>
<th>( S_1 )</th>
<th>( S_2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( A_1A_1 )</td>
<td>1</td>
<td>1</td>
<td>( \delta )</td>
</tr>
<tr>
<td>( A_1A_2 )</td>
<td>1</td>
<td>( \delta )</td>
<td>1</td>
</tr>
<tr>
<td>( A_2A_2 )</td>
<td>1</td>
<td>1</td>
<td>( \delta )</td>
</tr>
</tbody>
</table>

which yields
\[
\delta pq(p + \delta q)^2 + q^2(p + \delta q)(q + \delta p) = \delta q^2(q + \delta p)^2 + pq(p + \delta q)(q + \delta p).
\]

Dividing by \( q^4 \) and substituting \( x \) for \( p/q \) yields
\[
F(x) = x^2[-\delta^3 + \delta^2 + \delta - 1] + x[\delta^3 - \delta^2 - \delta + 1] = 0.
\]
The root of this equation is \( x = 1 \); that is, \( p = q = \sqrt{2} \), as expected. This equilibrium point is stable if
\[
p > \sqrt{2} \rightarrow f(p) < p \quad \text{or (equivalently) } \quad x > 1 \rightarrow F(x) < 0.
\]
In other words, it is necessary that \( g(\delta) = -\delta^3 + \delta^2 + \delta - 1 < 0 \). But, since \( g(\delta) = (-\delta - 1)^2(1 + \delta) \), this inequality is true for all \( \delta > 0, \delta \neq 1 \). We can infer, now, the existence of a stable polymorphism at \( p = q = \sqrt{2} \) for all \( 0 < \delta < 1 \).

Examine the benefit of being sexual when the cost of sex is \( \alpha \approx 1 \). As we have seen, the following inequality should be satisfied:
\[
\delta^* \alpha > \alpha^2 \delta^*, \quad \text{for } \delta^* = \left( \frac{\delta}{2} - 2\delta(2\alpha^2 - 1) - 1 > 0 \right)
\]
Equivalently, \( \delta^* = 2\delta(2\alpha^2 - 1) + 1 > 0 \). A solution to this inequality is \( 0 < \delta < \delta^*(\alpha) \) for
\[
\delta^*(\alpha) = (2\alpha^2 - 1) - \sqrt{(2\alpha^2 - 1)^2 - 1}.
\]
It can easily be verified that \( \delta^*(\alpha) \) is a decreasing function of \( \alpha \), meaning that the sensitivity of the model to changes in the fitness coefficients depends on the cost of sex, as in the general symmetrical case. As an example, consider two cases. If \( \alpha = 1 \), then \( \delta^* = 1 \), and sex is always beneficial (for any \( \delta < 1 \)). If \( \alpha = 2 \), then \( \delta^* = 0.07 \), and an advantage to sex exists for \( 0 < \delta < 0.07 \).

This model is similar to that proposed by Hamilton et al. (1981) after substituting \( \delta = r^{-2} \) (see above), giving their results as a special case. In other words, for every cost of sex, \( \alpha \), there is a possible \( r \) that is big enough (equivalent to \( \delta \) small enough) for sexual individuals to benefit under the conditions of the model. Moreover, an area of stable polymorphism can be shown to exist for every \( \delta \) (or \( r \)).

Similar results can be obtained if three different coefficients, \( \beta, \gamma, \delta \), are used instead of assuming the same viability, \( \delta \), for all susceptible individuals (which removes the singularity of the above case).

LITERATURE CITED


