

The Evolution of Sex

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1 Introduction

Sexual reproduction raises the major unsolved problem of evolutionary biology today: Why do almost all organisms reproduce sexually rather than asexually? After 50 years of literature on this question, one may suspect that every conceivable answer has already been explored. Nonetheless, this article proposes a simple theory to explain the evolution of sex. What appears to be new here is the explanation of *how* sex is generally advantageous in a fluctuating environment—it is a sexual population’s *non*responsiveness to changing conditions that is beneficial. This article also reexamines the cost of sex to develop a measure of cost that can be directly compared with the benefit of sex. The cost of sex is shown to depend on the mating system, specifically on the fraction of monogamous matings, and may be much less than most estimates discussed previously in the literature.

I accept that the main benefit of sexual reproduction is somehow associated with a fluctuating environment. Maynard Smith (1971) posed the question: What use is sex? And Williams (1975, p. 3) answered with Bonner’s (1958) conclusion that “sex is a parental adaptation to the likelihood of the offspring having to face changed or uncertain conditions.” The basis for this conclusion is that species having capabilities for both sexual and asexual reproduction typically reproduce sexually at times, such as the end of summer, when conditions are soon to change, and asexually otherwise. Thus, I agree with Williams (1975, p. 7) “that the association between sexual re-

production and changed conditions . . . is adequately supported, even though . . . what is implied by changed conditions is not yet clearly specified.” This article explicates what *is* implied for the evolution of sex by changing conditions. Earlier studies have sought to show that a sexual population has a greater ability to adapt to a changing environment than a comparable asexual population. In contrast, this study shows that the nonresponsiveness of a sexual population to changing conditions endows it with a higher long term growth rate than a comparable asexual population. This long term advantage is quite general and does not depend on restrictive assumptions about the pattern of environmental fluctuation or a particular genetic system.

The main cost to sexual reproduction was discovered by Maynard Smith (1971), and termed the “cost of meiosis”. The idea is that, because males do not produce offspring, a completely female species that produces solely female offspring asexually grows twice as fast as a sexually reproducing species that produces only 50% females. Hence, an asexually reproducing species quickly displaces a comparable sexually reproducing species. Alternatively, the cost of meiosis may be expressed in terms of kin selection: a female that can raise a fixed number of offspring leaves twice as many of her genes in the next generation by reproducing asexually rather than sexually. These alternative statements of the cost of meiosis are not necessarily equivalent, and the way the cost should be measured so that it can be compared with the benefit of sex is not obvious (Charlesworth 1980, Lloyd 1980, and Uyenoyama 1984). This article develops a measure for the cost of meiosis that can be compared with the particular benefit of sexual reproduction emphasized here, and shows that the magnitude of this cost depends on the degree of monogamy in the species. Specifically, the cost may be substantially less than a 50% loss if the matings are mostly monogamous.

Because the cost of meiosis may be as high as a 50% loss, the dilemma is to find an advantage to sexual reproduction that offers some gain by a factor of two or more. The net advantage to sexual reproduction would then exceed the cost, and the evolution of sex will have been explained. But as Williams (1975, p. 11) writes, “Anyone familiar with accepted evolutionary thought will realize what an unlikely sort of quest this is. . . . Nothing remotely approaching an advantage that could balance the cost of meiosis has been suggested. The impossibility of sex . . . would seem to be . . . firmly established . . . Yet this conclusion must surely be wrong.”

The resolution of the dilemma proposed in this article has certainly occurred to many. For example, Maynard Smith (1978, p. 2) writes, “An

individual female which abandons sexual reproduction obtains thereby an immediate short-term advantage. This means that once a parthenogenetic strain is established, it will selectively displace the sexual species. In the long run, however, the new parthenogenetic species is doomed to extinction . . .” Moreover, lineages of asexual species are observed to be phylogenetic “dead ends” that have sprung only recently from sexual ancestors (Stebbins 1950, Van Valen 1975, Bell 1982). And even Williams (1988, p. 289), whose research in the past has strongly criticized group selection, concedes that “Selection at the level of species or higher groups . . . may be required to explain the global prevalence of sexual groups and the low [taxonomic] rank of the exclusively asexual.” Why then has this attractive, and possibly correct, explanation not been universally adopted as the answer to why sex has evolved?

Both Williams (1975) and Maynard Smith (1978) rejected the explanation of species selection overcoming natural selection because they attached great theoretical significance to species that are polymorphic with sexual and asexual morphs. Williams (1975, p. 11) writes that such a polymorphism represents a “*currently* adaptive optimum maintained by selection. In these populations there can be no net disadvantage to sexual reproduction [my emphasis]”. Similarly, Maynard Smith (1978, p. 5) writes “Since sex continues, it must have some short-term advantages.” Thus, both these authors presume that a polymorphism between sexual and asexual morphs must be maintained by natural selection operating in the present-day environment of the polymorphic population. They do not consider it possible that a stable genetic polymorphism could be maintained by a balance between natural selection and group or species selection. Yet, Eshel (1972) has demonstrated mathematically that a stable genetic polymorphism can be maintained by a balance of natural selection favoring one morph and group selection favoring the other. A numerical example of such a polymorphism is displayed in Fig. 14.11 of Roughgarden (1979). Thus, the existence of species polymorphic for sexual and asexual morphs does not *ipso facto* imply that some force of ordinary natural selection favoring sexual reproduction must be identified to counteract that favoring asexual reproduction. This point is also consistent with recent calculations by Nunney (1989) showing how sex can be maintained through species selection. Nunney’s calculations assume that sex is somehow disadvantageous to individual selection, and advantageous to species selection, without explicating the basis for the costs and benefits of sex, as is the focus of this paper. Still further support for an approach

involving species selection comes from Gouyon et al. (1988) who urge an analysis of a hierarchy of processes as contributing to the evolution of sex.

Indeed, the difficulty of identifying a strong enough source of natural selection to maintain sex in face of the cost of meiosis led Williams (1975, p. 14) to despair of “ever finding a sufficiently powerful advantage in sexual reproduction with broadly applicable models that use only such general properties as mutation rates, population sizes, selection coefficients etc.” His book, therefore, introduces scenarios in which sexual reproduction is advantageous depending on a specified spatial or temporal pattern in the habitat or in the resources available to a population. Many papers in the most recent volume summarizing theory for the evolution of sex (Michod and Levin 1988) continue in this vein in hope that the union of all explanations covers enough species to account for the generality of sexual reproduction. As Michod and Levin (1988, p. vii) write “. . . the possibility of multiple mechanisms contributing to the evolution of sex is becoming accepted.” Yet, this article shows there *is* a simple and general benefit to sexual reproduction in terms of species selection in a fluctuating environment that may be sufficient to counteract the cost of meiosis.

The possibility of a fluctuating environment providing a benefit to sexual reproduction has been strongly discounted in previous theoretical studies. Maynard Smith (1978, p. 89) writes “. . . the belief that sex and recombination are favored in a variable or unpredictable environment is too simple. The environment must be unpredictable in a special and somewhat implausible sense.” Maynard Smith is referring to whether a fluctuating environment favors sexual reproduction *at all*—whether it is favored enough to overcome the cost of meiosis was not even raised. In this quotation, Maynard Smith is discussing models that synonymize the evolution of sex with the evolution of an increase in the recombination rate between two loci. Since the earliest theoretical writings on the evolution of sex (Muller 1932, Crow and Kimura 1965, Karlin 1973, Felsenstein 1974), the function of sex has been understood as permitting recombination among loci. This understanding has more recently led to models consisting of two “primary” loci whose gene products directly affect fitness, together with a third locus whose gene product modifies the linkage distance between the two primary loci (Nei 1967, Feldman 1972, Liberman and Feldman 1986). In such a model, the condition whereby evolution at the modifier locus will loosen linkage between the two primary loci can be determined, and as Maynard Smith reports, turns out to be implausibly restrictive (Charlesworth 1976). It is not necessary,

however, to focus on sex solely as permitting recombination to occur among loci. Another, and perhaps more fundamental, function of sex is to permit reassortment within a locus. That is, for every locus, mating leads to offspring genotypes that are a reassortment of the parental alleles at that locus, regardless of whether there is also recombination among loci. By focussing on this more simple aspect of what sex does, this article shows that, apart from the cost of meiosis, sexual reproduction *is* typically better in a fluctuating environment than asexual reproduction. Moreover, the magnitude of this advantage may be sufficient to offset the cost of meiosis.

A single-locus approach to the evolution of sex has also been severely criticized in previous theoretical studies. Hamilton *et al.* (1981, p. 371) write “. . . 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.” Weinshall (1986) recently provided a model to support a one-locus approach to the evolution of sex, but concluded that a two-state environment cannot explain the evolution of sex—the environment was required to have at least three states. However, this article shows that there *is* a one-locus two-allele model that may be plausibly sufficient using a simple two-state environment for each genotype. Moreover, an extension of this paper’s one-locus model to two loci shows an enhanced benefit to sexual reproduction in a fluctuating environment, but with a magnitude that appears independent of the recombination rate between the two loci. Hence, the evolution of linkage relationships among loci may not be relevant to the evolution of sex. This paper’s emphasis on the importance of within-locus assortment rather than between-locus recombination is shared by a recent contribution from Kirkpatrick and Jenkins (1989). They show that assortment in a diploid sexual population allows selection to carry a single advantageous mutation to a homozygous state, whereas two separate mutations are required in an asexual population. They suggest that the higher selective load thereby incurred by the asexual population can in some circumstances offset its two-fold reproductive advantage.

Before proceeding, I emphasize that my analysis of the cost of meiosis merely refines of the insight of Maynard Smith (1971), and my treatment of a fluctuating environment as favoring sexual reproduction draws on the thought in Hamilton (1980), and May and Anderson (1983). Let us begin

then with the bad news: is the cost of meiosis genuine, how large is this cost, how can this cost be measured in terms that allow comparison with the potential benefits of sex in a fluctuating environment, and why does this cost exist at all?

2 Cost of Sexual Reproduction

The cost of sexual reproduction turns out to depend on whether the mating within the species is promiscuous or monogamous. So, to explore this dependency on mating system, we first investigate a completely promiscuous mating system, then a completely monogamous system, and finally a system in which both promiscuity and monogamy are mixed. In all cases we focus on a simultaneous hermaphrodite, that is, an organism with both male and female gonads. This is probably the most common type of individual that exists, seeing that most individual flowering plants produce both seeds and pollen. Also, a great many invertebrates, such as barnacles and snails, have both male and female gonads. Dioecy, where each individual has only a single sex, can presumably be considered a derivation of simultaneous hermaphroditism whereby a parent, rather than packaging both male and female gonads within its body, instead produces males that function as physically detached extensions of itself at the cost of deferring male function until the male can mature.

The major assumptions are that an egg is much larger than a sperm, and that an egg is capable of developing into a new individual, while a sperm is not. A sperm functions only in fertilizing eggs. Thus, the number of offspring an individual leaves is determined by its egg production, while the number of genes it leaves is determined by both its egg production and sperm production. The total quantity of material a parent has for manufacturing eggs and sperm is finite, Q . This material can be allocated into sperm, V , and eggs, W . If the cost of making a sperm and egg are c_s and c_e , respectively, the reproductive activity by an individual obeys

$$c_s V + c_e W \equiv Q. \quad (1)$$

The maximum number of eggs that an individual can make is

$$W_m \equiv \frac{Q}{c_e}, \quad (2)$$

which results if Q is allocated entirely into eggs, with no sperm production. Our task then is to compute the allocation into sperm and eggs that evolves by natural selection. This egg production, called the optimum egg production W_o , is necessarily less than or equal to W_m .

The genetic system considered here consists of one locus with two alleles, A_1 and A_2 in a diploid individual. The frequency of the genotype $A_i A_j$ at time t is $X_{ij,t}$ where $X_{11,t} + X_{12,t} + X_{22,t} \equiv 1$. The total population size at time t is N_t , and its dynamics are determined solely by the egg production. Hence,

$$N_{t+1} = \bar{W}_t N_t \quad (3)$$

where the average egg production per individual at time t is

$$\bar{W}_t \equiv W_{11}X_{11,t} + W_{12}X_{12,t} + W_{22}X_{22,t}. \quad (4)$$

The W_{ij} , is taken as a constant here, but later in the article is assumed to fluctuate from generation to generation. Here \bar{W}_t , also called the ‘‘mean fitness’’, changes through time solely as a result of the changes in genotype frequencies, $X_{ij,t}$, that occur as the population evolves.

2.1 Promiscuous Mating

To specify the dynamics of the genotype frequencies we need a mating system. So, suppose that each individual is fertilized by a random sample of all sperm produced in the population. The average sperm production per individual at time t is

$$\bar{V}_t \equiv V_{11}X_{11,t} + V_{12}X_{12,t} + V_{22}X_{22,t}. \quad (5)$$

The frequency of the A_1 allele in the ‘‘sperm pool’’ is then

$$p_{s,t} \equiv \frac{V_{11}}{\bar{V}_t}X_{11,t} + \frac{1}{2} \frac{V_{12}}{\bar{V}_t}X_{12,t} \quad (6)$$

because all the sperm from $A_1 A_1$ parents and one half the sperm from $A_1 A_2$ parents contain an A_1 allele. Similarly, the frequency of A_2 in the sperm pool is

$$q_{s,t} \equiv \frac{V_{22}}{\bar{V}_t}X_{22,t} + \frac{1}{2} \frac{V_{12}}{\bar{V}_t}X_{12,t}. \quad (7)$$

Turning to the eggs, we also have

$$p_{e,t} \equiv \frac{W_{11}}{\bar{W}_t}X_{11,t} + \frac{1}{2} \frac{W_{12}}{\bar{W}_t}X_{12,t} \quad (8)$$

and

$$q_{e,t} \equiv \frac{W_{22}}{W_t} X_{22,t} + \frac{1}{2} \frac{W_{12}}{W_t} X_{12,t}. \quad (9)$$

With promiscuous mating each egg is fertilized at random from the sperm pool, so the genotype frequencies after mating are

$$X_{11,t+1} = p_{s,t} p_{e,t} \quad (10)$$

$$X_{12,t+1} = q_{s,t} p_{e,t} + p_{s,t} q_{e,t} \quad (11)$$

$$X_{22,t+1} = q_{s,t} q_{e,t}. \quad (12)$$

Consider now the condition under which a rare allele, say A_2 , can increase into a population consisting entirely of A_1 . A local stability analysis at the boundary equilibrium, $(\hat{X}_{11}, \hat{X}_{12}, \hat{X}_{22}) = (1, 0, 0)$ indicates that A_2 increases when initially rare if

$$V_{11}(W_{12} - W_{11}) + W_{12}(V_{12} - V_{11}) > 0. \quad (13)$$

To interpret this condition, suppose the phenotype produced by the rare allele differs only slightly from the established allele,

$$V_{11} \equiv V \quad (14)$$

$$V_{12} \equiv V + dV \quad (15)$$

$$W_{11} \equiv W \quad (16)$$

$$W_{12} \equiv W + dW. \quad (17)$$

Then the condition for increase of the rare allele becomes

$$VdW + WdV > 0, \quad (18)$$

which can be rewritten using a total differential as,

$$d[VW] > 0. \quad (19)$$

Therefore, in this mating system, natural selection maximizes the product of the sperm and egg production (MacArthur 1965, Charnov 1982, Lessard 1989).

Substituting for V from Eq. (1) yields

$$W_o = \frac{1}{2} \frac{Q}{c_e}. \quad (20)$$

Thus, the allocation to egg production that evolves by natural selection is exactly one half the maximal egg production, W_m . That is, an asexual species with the maximal egg production grows at twice the rate of this sexual species.

This finding traces to sperm-sperm competition; not interference competition, but competition resulting simply from the numerical ratios of the genes in the sperm pool (mass-action competition). If an individual's eggs are fertilized by a random sample of all sperm, than a gene can spread *via* the male route only by producing a large enough quantity of sperm to affect the gene frequency in the sperm pool. That is, the chance that a gene becomes incorporated into a zygote *via* a sperm depends on how many sperm carry a copy of that gene because fertilization is by a sperm selected at random from the sperm pool. Hence, for an individual to transmit genes to the next generation, an effort at sperm production is needed to influence the gene frequency in the sperm pool, and the optimum degree of this effort should equal that allocated to the production of eggs. A side effect, though, is that egg production is reduced by one half. This side effect is the “cost of meiosis”.

Thus, mass-action sperm-sperm competition emerges as a fundamental reason why the cost of meiosis exists. It is not clear whether the kin selection formulation of the cost of meiosis is equivalent to the sperm-sperm competition derivation, or whether the kin selection formulation is valid at all; in any case, the kin selection formulation seems superfluous.

2.2 Monogamous Mating

Given that sperm-sperm competition underlies the cost of meiosis, the mating system should influence this cost by determining which sperm are involved in fertilizing eggs. Consider now monogamous mating where an individual is fertilized by, and fertilizes, only one other individual. Couples form at random, but once formed, remain intact. Here the sperm from one parent can fertilize eggs regardless of how common the genotypes of those sperm are in the sperm pool, and mass-action sperm-sperm competition is absent. Hence, an individual need not invest the high quantity of sperm production needed to influence gene frequencies in the sperm pool. It need only produce the relatively trivial number of sperm needed to fertilize its partner's eggs.

As before, the frequencies of A_1 and A_2 in the eggs are

$$p_{e,t} \equiv \frac{W_{11}}{W_t} X_{11,t} + \frac{1}{2} \frac{W_{12}}{W_t} X_{12,t} \quad (21)$$

and

$$q_{e,t} \equiv \frac{W_{22}}{\overline{W}_t} X_{22,t} + \frac{1}{2} \frac{W_{12}}{\overline{W}_t} X_{12,t}, \quad (22)$$

respectively. These eggs are fertilized by sperm from individuals chosen at random—individuals are chosen at random from among the parents, not sperm from the sperm pool. Hence, if the frequencies of A_1 and A_2 among the parents are denoted

$$p_t \equiv X_{11,t} + \frac{1}{2} X_{12,t} \quad (23)$$

and

$$q_t \equiv X_{22,t} + \frac{1}{2} X_{12,t}, \quad (24)$$

respectively, we have for genotype frequencies at time $t + 1$

$$X_{11,t+1} = p_t p_{e,t} \quad (25)$$

$$X_{12,t+1} = p_t q_{e,t} + q_t p_{e,t} \quad (26)$$

$$X_{22,t+1} = q_t q_{e,t}. \quad (27)$$

An individual is presumed to have enough sperm to fertilize the eggs of one partner without materially affecting its own egg production. This requires sperm to be much smaller than eggs, as originally assumed.

Mathematically, this monogamy model is a special case of the promiscuity model with $V_{ij} \equiv 1$. Hence, a rare allele, A_2 , will increase if

$$W_{12} - W_{11} > 0. \quad (28)$$

If the phenotype expressed by this rare allele is a slight variation of the established phenotype, then the condition for increase becomes

$$dW > 0. \quad (29)$$

Hence, in this sexual population natural selection maximizes egg production, provided each individual also makes enough sperm to fertilize its partner's eggs. There is no sperm–sperm competition in this mating system, and no cost of meiosis evolves.

2.3 Mixed Promiscuous and Monogamous Mating

Let us formally define the *cost of meiosis*, C , as the inverse of the factor relating the optimal egg production for a given mating system to the maximal egg production,

$$W_o \equiv \frac{1}{C} W_m. \quad (30)$$

C equals 2 with promiscuity and 1 with monogamy. We might guess that C varies between these limits for mating systems that fall between the extremes of promiscuity and monogamy. To demonstrate this claim, let m be the fraction of an individual's eggs fertilized by a single partner, and $(1 - m)$ the fraction fertilized with a random sample of the sperm pool. The m is called the *monogamy index*. The genotype frequency dynamics are then

$$X_{11,t+1} = (1 - m)(p_{s,t}p_{e,t}) + m(p_t p_{e,t}) \quad (31)$$

$$X_{12,t+1} = (1 - m)(q_{s,t}p_{e,t} + p_{s,t}q_{e,t}) + m(q_t p_{e,t} + p_t q_{e,t}) \quad (32)$$

$$X_{22,t+1} = (1 - m)(q_{s,t}q_{e,t}) + m(q_t q_{e,t}). \quad (33)$$

A rare A_2 increases if

$$V_{11}(W_{12} - W_{11}) + (1 - m)W_{12}(V_{12} - V_{11}) > 0. \quad (34)$$

By regarding A_2 as expressing a phenotype slightly different from the established phenotype, we have

$$VdW + (1 - m)WdV > 0. \quad (35)$$

Upon multiplying both sides by $V^{(1-m)-1}$, we obtain

$$d[V^{(1-m)}W] > 0, \quad (36)$$

showing that natural selection maximizes a product of sperm and egg production, with the significance of the sperm diminishing as the fraction of monogamous fertilizations increases. Therefore, the optimum egg production becomes

$$W_o = \frac{1}{2 - m} \frac{Q}{c_e}. \quad (37)$$

The cost of meiosis, as a function of the monogamy index, then is

$$C = 2 - m. \quad (38)$$

Cost of Meiosis, C

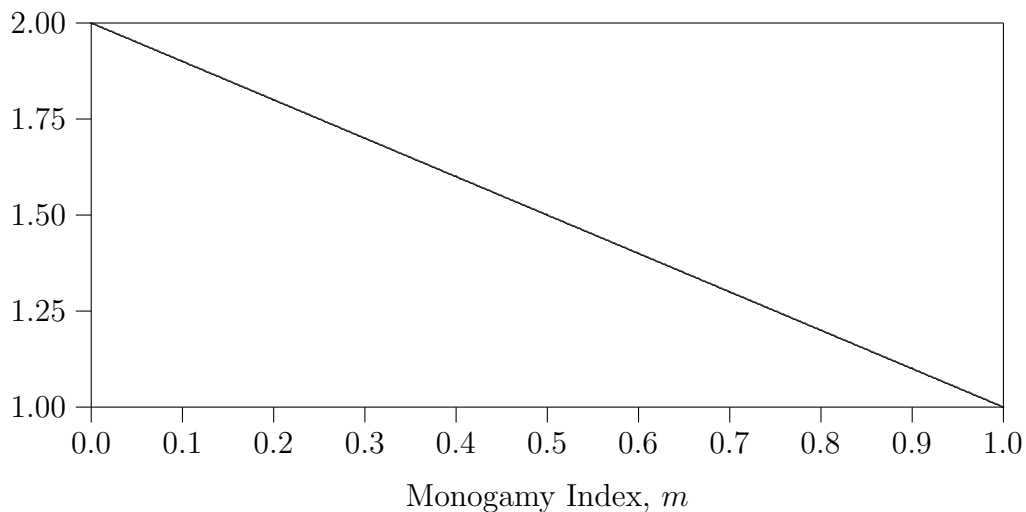


Figure 1: The cost of meiosis as a function of the monogamy index.

A graph of this simple relation appears in Fig. 1.

The cost of meiosis may be empirically estimated from data on resource allocation to male and female functions. Let the female and male reproductive “effort” be defined, respectively, as

$$E_e \equiv \frac{c_e W}{Q} \quad (39)$$

$$E_s \equiv \frac{c_s V}{Q} \quad (40)$$

where, by Eq. 1, $E_e + E_s \equiv 1$. With these definitions, the cost of meiosis is the reciprocal of the female reproductive effort

$$C \equiv \frac{1}{E_e}. \quad (41)$$

Thus, promiscuity leads to the evolution of a female reproductive effort of 0.5 and to a cost of meiosis of 2, and monogamy to an E_e and C that both equal 1. Mixed mating systems lie between these bounds.

3 Benefit of Sexual Reproduction

A numerical analysis is offered to show the benefit of sexual reproduction in a fluctuating environment. Also, a mathematical basis to the numerical analysis seems clear, although unproven formally.

Both sexual and asexual populations are assumed to consist of the same number of genotypes. In the sexual population these are the three genotypes formed from one locus with two alleles, or the ten genotypes formed from two loci with two alleles per locus. These genotypes are genetically distinct clones in the asexual population.

Each genotype sees a sequence of fitnesses through time realized from a two-state stationary Markov chain. As well known, if P_{12} is the probability that “bad” follows “good”, and P_{21} the probability that “good” follows “bad”, then the stationary probabilities of “good” and “bad” are

$$\pi_1 = \frac{P_{21}}{P_{12} + P_{21}} \quad (42)$$

$$\pi_2 = \frac{P_{12}}{P_{12} + P_{21}}, \quad (43)$$

respectively, and the serial correlation between consecutive states is

$$\rho = 1 - (P_{12} + P_{21}). \quad (44)$$

In particular, if $P_{12} + P_{21} = 1$ then consecutive states of the environment are independent (white noise), and if $P_{12} = P_{21}$ then “good” and “bad” are equally likely. This setup allows both the frequency of “good” and “bad”, and the environmental predictability, to be varied independently.

The fitness in a bad environment is defined using a strength parameter, s , as

$$W_b = \frac{1}{s}. \quad (45)$$

The fitness in a good environment is

$$W_g = s^{\frac{\pi_2}{\pi_1}}. \quad (46)$$

The geometric mean of W_b and W_g equals 1. If both “good” and “bad” are equally likely ($\pi_1 = \pi_2$), the fitnesses simply fluctuate between $\frac{1}{s}$ and s . If “bad” is rare, then the decline in a bad generation is severe relative

to the gentle increase during each of the intervening good generations. The bad generation thus appears as a rare catastrophe. Conversely, if “good” is rare, a good generation appears as a population outbreak punctuating long periods of slow decline.

The other ingredient is recurrent mutation. A sexual population does not differ from an asexual population if it becomes genetically monomorphic. Natural selection here does not, by itself, conserve genetic variation because each genotype sees the same fitnesses in the long run, and one of the alleles eventually wanders to extinction. Therefore, to conserve genetic variation, recurrent mutation between the alleles at each locus is included.

3.1 One Locus

The sexual and asexual populations differ only in that the sexual population undergoes meiosis and random union of gametes at the end of each generation. The sexual population begins each generation with its genotype frequencies in Hardy-Weinberg ratios, while the asexual population begins with whatever genotype frequencies remain from the preceding generation.

The sequence of processes is: selection; recurrent mutation; and, for the sexual population only, random union of gametes. The selection episode is

$$X'_{\alpha,ij,t} = \frac{W_{ij,t}}{\bar{W}_{\alpha,t}} X_{\alpha,ij,t}, \quad (47)$$

where α is either s or a to indicate the sexual or asexual population, and ij indicates the genotype, A_iA_j . The fitness for genotype A_iA_j at time t , $W_{ij,t}$ numerically equals either W_b or W_g depending on whether the environment is “bad” or “good” for that genotype at that time. The mean fitness is

$$\bar{W}_{\alpha,t} \equiv W_{11,t}X_{\alpha,11,t} + W_{12,t}X_{\alpha,12,t} + W_{22,t}X_{\alpha,22,t}. \quad (48)$$

Next, the recurrent mutation episode is

$$X''_{\alpha,11,t} = v^2X'_{\alpha,11,t} + uvX'_{\alpha,12,t} + u^2X'_{\alpha,22,t} \quad (49)$$

$$X''_{\alpha,12,t} = 2uvX'_{\alpha,11,t} + (u^2 + v^2)X'_{\alpha,12,t} + 2uvX'_{\alpha,22,t} \quad (50)$$

$$X''_{\alpha,22,t} = u^2X'_{\alpha,11,t} + uvX'_{\alpha,12,t} + v^2X'_{\alpha,22,t}, \quad (51)$$

where u is the recurrent mutation rate for $A_1 \rightleftharpoons A_2$, and $v \equiv 1 - u$ is the probability a mutation does not occur. For the asexual population, these

genotype frequencies simply become those for time $t + 1$,

$$X_{a,ij,t+1} = X''_{a,ij,t}. \quad (52)$$

For the sexual population, however, meiosis leads to the gamete gene frequencies

$$p''_t \equiv X''_{s,11,t} + \frac{1}{2}X''_{s,12,t} \quad (53)$$

$$q''_t \equiv X''_{s,22,t} + \frac{1}{2}X''_{s,12,t}, \quad (54)$$

and after random union of gametes the sexual genotype frequencies are

$$X_{s,11,t+1} = p''_t p''_t \quad (55)$$

$$X_{s,12,t+1} = 2p''_t q''_t \quad (56)$$

$$X_{s,22,t+1} = q''_t q''_t. \quad (57)$$

For both sexual and asexual populations, the population size is affected by the mean fitness as

$$N'_{\alpha,t} = \bar{W}_{\alpha,t} N_{\alpha,t}. \quad (58)$$

While both sexual and asexual populations see the same genotype fitnesses at each time, $W_{ij,t}$, their mean fitnesses, $\bar{W}_{\alpha,t}$, usually differ because their genotype frequencies, $X_{\alpha,ij,t}$ differ. For convenience, we renormalize the population sizes at the end of each generation to simulate a finite environment,

$$N_{\alpha,t+1} = \frac{N'_{\alpha,t}}{N'_{s,t} + N'_{a,t}}. \quad (59)$$

The conclusions are identical if an infinite environment is assumed instead. The renormalization is probably more realistic, however, and also ensures that the variables remain bounded.

Sexual reproduction may endow a population with a higher geometric mean fitness in a fluctuating environment. Therefore, we formally define the *benefit of meiosis*, B as a ratio of the long term geometric means achieved by a sexual population relative to an asexual population, assuming both experience the same conditions,

$$B \equiv \frac{\lim_{T \rightarrow \infty} \left(\prod_{t=1}^T \bar{W}_{s,t} \right)^{\frac{1}{T}}}{\lim_{T \rightarrow \infty} \left(\prod_{t=1}^T \bar{W}_{a,t} \right)^{\frac{1}{T}}}, \quad (60)$$

where the limit is taken over the same environmental sample path for both sexual and asexual populations.

A computer program carries out each ensemble of runs as follows. The parameters are read into the program. The random number generator, “drand48()” of UNIX, is initialized. A run is then initialized for both sexual and asexual populations with genotype frequencies equal to $(0, 0, 1)$, and population sizes equal to $\frac{1}{2}$. For each generation of the run, fitness states for the three genotypes are determined with the random number generator using the Markov transition probabilities, P_{12} and P_{21} together with a record of the previous environmental state for each genotype. For example, suppose good and bad states are equally likely, and that consecutive generations are independent. With a coin, let “heads” stand for “good” and “tails” for “bad.” Also, let the selection strength, s , equal 2. Then tossing “heads”, “heads”, and “tails” yields fitnesses in that generation, $W_{ij,t}$, of $(2, 2, \frac{1}{2})$. This set of fitnesses is applied to the genotypes in both sexual and asexual populations. Because the genotypes of the sexual and asexual population see the same fitness states, both populations are growing side by side. When each run is over, statistics for that run are calculated; and a new run initiated. When all the runs are completed, the ensemble statistics are determined. Then another parameter combination is read in to initiate another entire ensemble of runs, corresponding to these new parameters. The program terminates when no parameters remain. All computations used double precision.

For each run in an ensemble, the computer program returns the ratio of the geometric mean of $\overline{W}_{s,t}$ to the geometric mean of $\overline{W}_{a,t}$ from the same run. An ensemble of 50 runs has 50 such ratios, and the average of these is reported as the “benefit of meiosis” corresponding to the set of parameters used for the ensemble.

Fig. 2 pertains to an environment with good and bad states equally likely, and with no serial correlation in the environment. Evidently, the benefit of meiosis is *always* greater than 1, indicating that a sexual population is ultimately *always* better than an asexual population in this completely unpatterned environment. The sexual population excludes the asexual population in a finite world provided the runs are long enough. (1000 generations is usually sufficient.)

Figs. 2–4 explore some quantitative details about how strong the benefit to sexual reproduction is. Fig. 2 shows when the benefit of meiosis exceeds 2; in the absence of serial correlation, and with good and bad states equally likely, this critical point is found when the fitness fluctuates between 512 and

Benefit of Meiosis, B

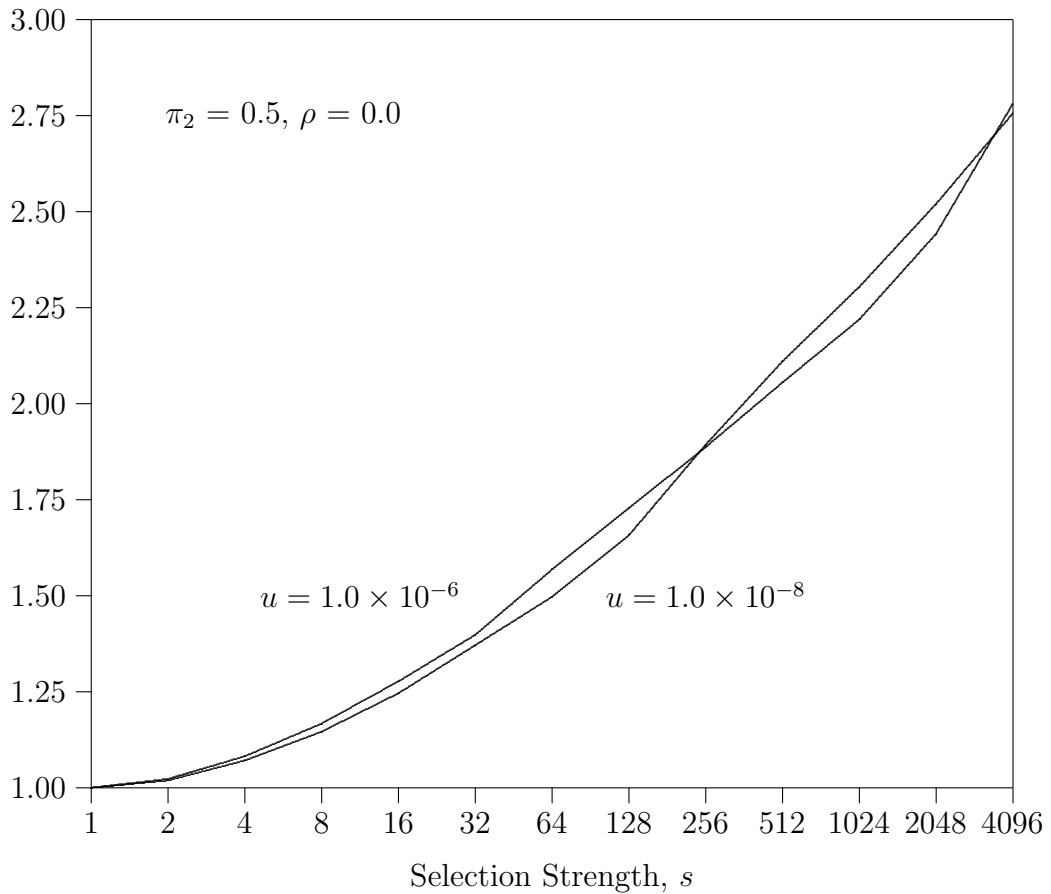


Figure 2: Benefit of meiosis as a function of the strength of selection in a fluctuating environment. u is the mutation rate of $A_1 \rightleftharpoons A_2$, π_2 is the probability of a bad environmental state, and ρ is the serial correlation between consecutive environmental states. Each point results from an ensemble of 50 trials lasting 5000 generations apiece.

Benefit of Meiosis, B

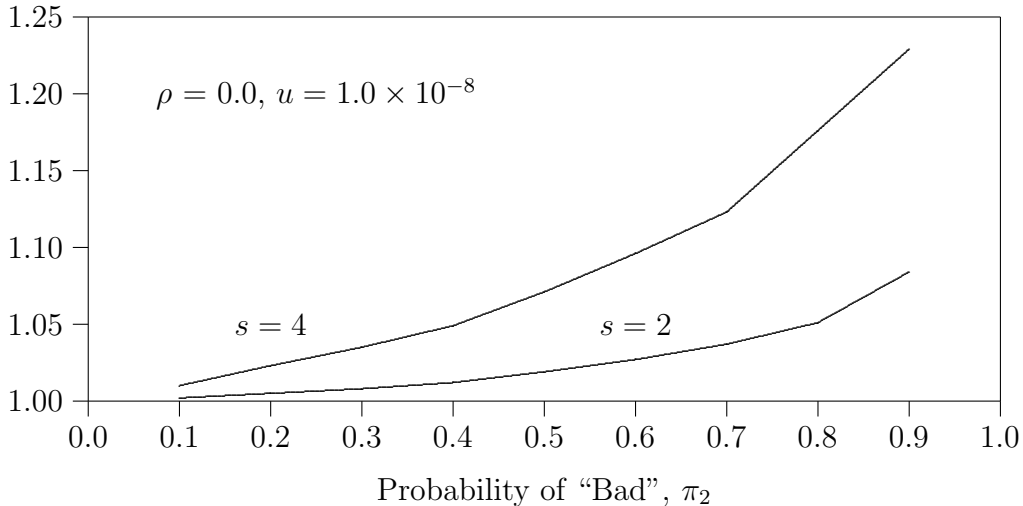


Figure 3: Benefit of meiosis as a function of the frequency of the bad environmental state. s is the strength of selection, ρ is the serial correlation between consecutive environmental states, and u is the mutation rate of $A_1 \rightleftharpoons A_2$. Each point results from an ensemble of 50 trials lasting 5000 generations apiece.

$\frac{1}{512}$. More extreme fluctuations yield higher benefits, less extreme fluctuations yield less. The figure also shows that increasing mutation improves the benefit slightly. Fig. 3 shows that the benefit increases as the probability of "bad" relative to "good" increases; in this figure there is no serial correlation. Fig. 4 shows that the benefit of meiosis increases as the serial correlation decreases; the figure assumes good and bad states are equally likely. If the serial correlation is high enough the benefit of meiosis drops to less than 1.

The mathematical basis to the benefit of meiosis is that the sexual population experiences less variance through time in its mean fitness than does an asexual population in the same circumstances. Hence, the geometric mean of $\bar{W}_{s,t}$ exceeds that of $\bar{W}_{a,t}$. The reason is that the sexual genotype frequencies are restored to the Hardy-Weinberg curve each generation, whereas the asexual genotype frequencies wander without constraint, leading to a lower variance of $\bar{W}_{s,t}$ relative to $\bar{W}_{a,t}$.

The *nonresponsiveness* of the sexual population to fluctuations in the environment is what underlies its success relative to an asexual population.

Benefit of Meiosis, B

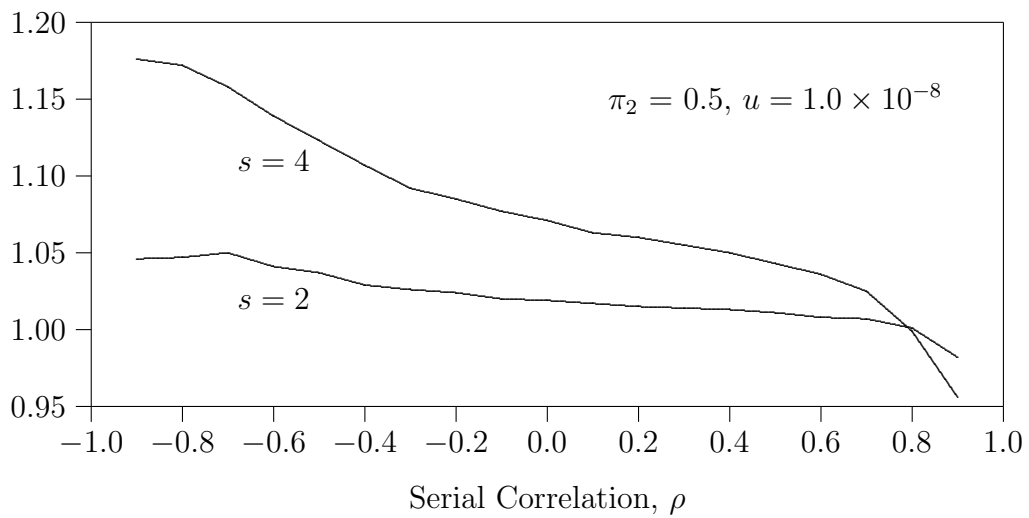


Figure 4: Benefit of meiosis as a function of the serial correlation between consecutive environmental states. s is the strength of selection, π_2 is the probability of a bad environmental state, and u is the mutation rate of $A_1 \rightleftharpoons A_2$. Each point results from an ensemble of 50 trials lasting 5000 generations apiece.

Previous studies have focussed on whether sexual reproduction endows a population with the ability to respond evolutionarily to changed conditions faster than an asexual population. Clearly, the advantage to sexual reproduction is just the opposite—a sexual population’s gene pool is “buffered” from random fluctuations in fitness, to use a term suggested by R. May (personal communication).

3.2 Two Loci

To explore whether the results just obtained from a one-locus model are also true of a more complicated genetic system, an extension to two loci with two alleles is offered. The gamete types are AB , Ab , aB , and ab . These lead to ten genotype frequencies, $X_{\alpha,i,t}$ where the index, i , varies from 0 to 9 to indicate genotypes in the order: $AB-AB$, $AB-Ab$, $AB-aB$, $AB-ab$, $Ab-Ab$, $Ab-aB$, $Ab-ab$, $aB-aB$, $aB-ab$, and $ab-ab$. Double heterozygotes have index 3 and 5. These genotypes have the same alleles, but in a different chromosomal arrangement. A parameter, r , called the recombination fraction, describes the crossing over that occurs between these loci—it varies from 0, indicating no crossing over, to 0.5.

For both sexual and asexual populations, the selection episode yields

$$X'_{\alpha,i,t} = \frac{W_{i,t}}{\bar{W}_{\alpha,t}} X_{\alpha,i,t}. \quad (61)$$

The fitness of each genotype fluctuates between “good” and “bad” according to a Markov chain, as before. However, at any one time the fitnesses of both double heterozygotes are identical—they fluctuate through time in unison. The mean fitness is

$$\bar{W}_{\alpha,t} \equiv \sum_{i=0}^9 W_{i,t} X_{\alpha,i,t}. \quad (62)$$

The mutation episode for both sexual and asexual populations yields

$$\mathbf{X}''_{\alpha,t} = \mathbf{M} \mathbf{X}'_{\alpha,t} \quad (63)$$

where $\mathbf{X}''_{\alpha,t}$ and $\mathbf{X}'_{\alpha,t}$ are column vectors of genotype frequencies, and \mathbf{M} is

the mutation matrix,

$$\begin{pmatrix}
v^4 & uv^3 & uv^3 & u^2v^2 & u^2v^2 \\
2uv^3 & u^2v^2 + v^4 & 2u^2v^2 & u^3v + uv^3 & 2uv^3 \\
2uv^3 & 2u^2v^2 & u^2v^2 + v^4 & u^3v + uv^3 & 2u^3v \\
2u^2v^2 & u^3v + uv^3 & u^3v + uv^3 & \frac{1}{2}(u^4 + 2u^2v^2 + v^4) & 2u^2v^2 \\
u^2v^2 & uv^3 & u^3v & u^2v^2 & v^4 \\
2u^2v^2 & u^3v + uv^3 & u^3v + uv^3 & \frac{1}{2}(u^4 + 2u^2v^2 + v^4) & 2u^2v^2 \\
2u^3v & 2u^2v^2 & u^4 + u^2v^2 & u^3v + uv^3 & 2uv^3 \\
u^2v^2 & u^3v & uv^3 & u^2v^2 & u^4 \\
2u^3v & u^4 + u^2v^2 & 2u^2v^2 & u^3v + uv^3 & 2u^3v \\
u^4 & u^3v & u^3v & u^2v^2 & u^2v^2 \\
\hline
& u^2v^2 & u^3v & u^2v^2 & u^3v & u^4 \\
& u^3v + uv^3 & 2u^2v^2 & 2u^3v & u^4 + u^2v^2 & 2u^3v \\
& u^3v + uv^3 & u^4 + u^2v^2 & 2uv^3 & 2u^2v^2 & 2u^3v \\
\frac{1}{2}(u^4 + 2u^2v^2 + v^4) & u^3v + uv^3 & 2u^2v^2 & u^3v + uv^3 & 2u^2v^2 & 2u^2v^2 \\
& u^2v^2 & uv^3 & u^4 & u^3v & u^2v^2 \\
\frac{1}{2}(u^4 + 2u^2v^2 + v^4) & u^3v + uv^3 & 2u^2v^2 & u^3v + uv^3 & 2u^2v^2 & 2u^2v^2 \\
& u^3v + uv^3 & u^2v^2 + v^4 & 2u^3v & 2u^2v^2 & 2uv^3 \\
& u^2v^2 & u^3v & v^4 & uv^3 & u^2v^2 \\
& u^3v + uv^3 & 2u^2v^2 & 2uv^3 & u^2v^2 + v^4 & 2uv^3 \\
& u^2v^2 & uv^3 & u^2v^2 & uv^3 & v^4.
\end{pmatrix}$$

In \mathbf{M} , u is the recurrent mutation rate for $A \rightleftharpoons a$ and for $B \rightleftharpoons b$, and $v \equiv 1 - u$.

The asexual population's genotypes at $t + 1$ then become

$$X_{a,i,t+1} = X''_{a,i,t}. \quad (64)$$

The sexual population must undergo meiosis, including recombination, leading to the gamete frequencies

$$\begin{aligned}
p''_{0,t} &= X''_{s,0,t} + (1/2)X''_{s,1,t} + (1/2)X''_{s,2,t} + (1/2)(1-r)X''_{s,3,t} + (1/2)rX''_{s,4,t} \\
p''_{1,t} &= (1/2)X''_{s,1,t} + (1/2)rX''_{s,3,t} + X''_{s,4,t} + (1/2)(1-r)X''_{s,5,t} + (1/2)X''_{s,6,t} \\
p''_{2,t} &= (1/2)X''_{s,2,t} + (1/2)rX''_{s,3,t} + (1/2)(1-r)X''_{s,5,t} + X''_{s,7,t} + (1/2)X''_{s,8,t} \\
p''_{3,t} &= (1/2)X''_{s,3,t} + (1/2)rX''_{s,5,t} + (1/2)X''_{s,6,t} + (1/2)X''_{s,8,t} + X''_{s,9,t}. \quad (65)
\end{aligned}$$

These gametes unite at random, leading to the genotype frequencies for the sexual population

$$X_{s,0,t+1} = p''_{0,t}p''_{0,t}$$

$$\begin{aligned}
X_{s,1,t+1} &= 2p''_{0,t}p''_{1,t} \\
X_{s,2,t+1} &= 2p''_{0,t}p''_{2,t} \\
X_{s,3,t+1} &= 2p''_{0,t}p''_{3,t} \\
X_{s,4,t+1} &= p''_{1,t}p''_{1,t} \\
X_{s,5,t+1} &= 2p''_{1,t}p''_{2,t} \\
X_{s,6,t+1} &= 2p''_{1,t}p''_{3,t} \\
X_{s,7,t+1} &= p''_{2,t}p''_{2,t} \\
X_{s,8,t+1} &= 2p''_{2,t}p''_{3,t} \\
X_{s,9,t+1} &= p''_{3,t}p''_{3,t}
\end{aligned} \tag{66}$$

As in the one-locus model, the population dynamics are determined by the mean fitness

$$N'_{\alpha,t} = \bar{W}_{\alpha,t} N_{\alpha,t} \tag{67}$$

$$N_{\alpha,t+1} = \frac{N'_{\alpha,t}}{N'_{s,t} + N'_{a,t}}. \tag{68}$$

Results appear in Fig. 5, wherein the benefit of meiosis as a function of the recombination fraction, r , is plotted. The flat curves suggest that the benefit is independent of the recombination between the loci. (The linkage disequilibrium coefficient between the loci evidently approaches zero quickly.) However, the benefit is higher than that from the corresponding one-locus model. Apparently then, the greater number of genotypes available in a two-locus system itself enhances the benefit of meiosis, regardless of the recombination between those loci.

4 Discussion

Why *do* almost all organisms reproduce sexually? This article confirms that a cost is fundamental to sexual reproduction, whereby the material that is placed into sperm production correspondingly lowers egg production, leading to a net decline in the species' growth rate. The evolution of this allocation to sperm production is driven by sperm-sperm competition, and depends on ratio of monogamous to promiscuous matings. This article also shows that a benefit in long-term species growth rate is typically realized by a sexual population in a fluctuating environment. This benefit results from a less variable

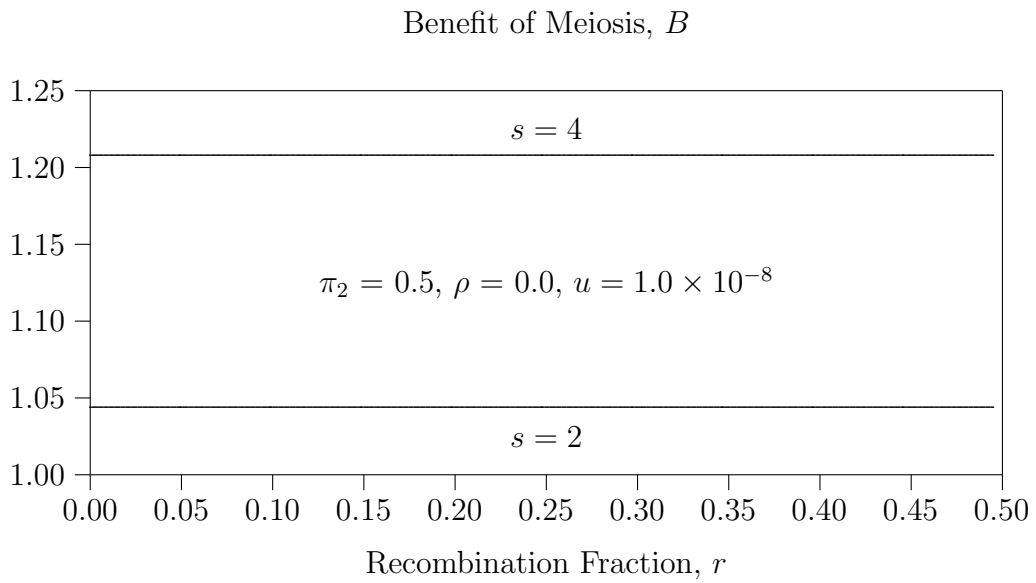


Figure 5: Benefit of meiosis as a function of the recombination fraction between the two loci. π_2 is the probability of a bad environmental state, s is the strength of selection, ρ is the serial correlation between consecutive environmental states, and u is the mutation rate of $A \rightleftharpoons a$ and for $B \rightleftharpoons b$. Each point results from an ensemble of 50 trials lasting 5000 generations apiece.

species growth rate for a sexual population relative to an asexual population in the same conditions. The generality of sexual reproduction could therefore be explained if this cost is usually less than this benefit. Moreover, the magnitude of the benefit to sexual reproduction increases with the degree of environmental fluctuation, thus possibly explaining a long recognized correlation between sexual reproduction and the prospect of changing environmental conditions.

Alas, an *a priori* argument is insufficient to show that the cost usually is less than the benefit, although at first glance it might appear so. After all, random environmental fluctuation is ubiquitous, suggesting a ubiquitous advantage to sexual reproduction. Also, a negative serial correlation in fitness which enhances the benefit of sex may be produced by ecological factors such as predation, parasitism, and disease. Monogamy ameliorates the cost of meiosis. The finite dispersal capabilities of organisms entail that they mate monogamously more than possible in a completely mixed population. Thus, the cost of meiosis should rarely, if ever, be a full halving of the species growth rate. Indeed, data summarized by Lloyd (1988) show that allocation to female function in flowering plants usually exceeds 50%, implying a less than maximal cost of meiosis.

But there may still be a large gap between cost and benefit. As Fig. 1 shows, the cost is still high until over 50% monogamy is achieved. As Figs. 2–5 show, the benefit remains low unless the environmental fitness fluctuation is substantial. Furthermore, the benefit to sex discussed here is consistent with other putative benefits, such as the advantage in selective load suggested by Kirkpatrick and Jenkins (1989) mentioned earlier, and an enhanced double-stranded DNA repair capability discussed by Bernstein et al (1987). These additional mechanisms may promote the evolutionary maintenance of sex by serving to reduce the overall gap between cost and benefit, but do not address the connection between the evolution of sex and fluctuating environmental conditions. This connection has been the focus of this paper, and any theory that does not address this connection cannot explain the phenomenology of when and where sexual and asexual reproduction occur. So, a general answer to why most organisms reproduce sexually may be available, but its truth turns on the magnitude of specific costs and benefits that have yet to be measured.

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