

ON GENETIC SEGREGATION AND THE  
EVOLUTION OF SEX

*Pamela Wiener, Marcus W. Feldman,  
and Sarah P. Otto*

*Paper number 0042*

Department of Biological Sciences  
Stanford University  
Stanford, California 94305

1991



## Introduction

The past twenty-five years of research into the advantages of sexual reproduction over asexual has emphasized the evolutionary dynamics of recombination (see Michod and Levin, 1988, for the current status of this subject). Kirkpatrick and Jenkins (1989) recently drew attention to a different aspect of the evolution of sex, namely the role of genetic segregation in diploids. They claimed that segregation in a diploid sexual population would allow advantageous mutations to accumulate in homozygous form, while in asexual organisms it would require two separate mutations to produce such a homozygote. An advantageous mutation would, therefore, be held in heterozygous form in parthenogenetic diploids, thus delaying its passage to fixation compared to sexual diploids. Kirkpatrick and Jenkins computed a "selective load" incurred by the asexuals relative to the sexuals resulting from this delay and determined that this may be more than enough to compensate for the classical twofold disadvantage of sexual reproduction.

In this note we first question some aspects of Kirkpatrick and Jenkins' analysis and then present results of a simulation study that compares finite asexual and sexual populations. In our simulation sexual reproduction usually cannot outcompete asexual reproduction.

## Kirkpatrick and Jenkins' Formulation; Comments

The problem is posed in terms of the genetic load that accrues to an asexual diploid population due to loci heterozygous for advantageous mutations that may occur at  $L$  loci, each of which is originally homozygous. At each locus, the fitnesses of the mutant heterozygote and homozygote are  $1 + hs$  and  $1 + s$ , respectively, relative to 1 for that of the wild-type ( $s > 0$ ,  $0 \leq h < 1$ ). It is assumed that only one of these  $L$  loci has a favorable mutation segregating at any time. This assumption is made to control for (and, in fact, eliminate) any possible "advantage to sexual reproduction derived from recombination". A simpler assumption to control for recombination would be absolute linkage among all  $L$  loci.

Next it is assumed that the population is of size  $N$  and the mutation rate per allele to advantageous alleles is  $\mu$ . Kirkpatrick and Jenkins then calculate two "fixation rates", the

first being the rate at which loci become “fixed” in the heterozygous state in asexuals and the second is the rate at which loci fixed as heterozygotes are converted to advantageous homozygotes. The former is computed as  $2N\mu LP_0$  where  $P_0$  is the “probability that a single advantageous mutation at a homozygous type locus spreads” (to fixation in the heterozygous state). The second rate is  $\mu Nn P_1$  where  $P_1$  is “the probability that a mutation converting a heterozygote to an advantageous homozygote spreads” (also to fixation), and  $n$  is the number of loci fixed in the heterozygous state. This leads to Kirkpatrick and Jenkins’ differential equation

$$\frac{dn}{dt} = 2N\mu LP_0 - \mu Nn P_1, \quad (1)$$

from which  $n(t)$  converges to  $2LP_0/P_1$  at the exponential rate  $\mu N P_1$ .

The values assumed for  $P_0$  and  $P_1$  come from the diffusion approximations to random genetic drift in haploids with selection. The diffusion analysis gives the probability of ultimate survival of a mutant with selective advantage  $\sigma$  over its wild-type allele as  $2\sigma$  provided  $N\sigma$  is not too small (see Ewens, 1979, p. 83). The favored heterozygote is assumed to arise via mutation and to begin at a frequency  $1/N$  in a population of size  $N$ . Kirkpatrick and Jenkins compute  $P_0$  as the probability that “fixation” occurs on the heterozygous type with fitness  $1 + hs$  relative to the wild-type, namely  $P_0 = 2hs$ . In the same way  $P_1$  is the probability of fixation on the advantage homozygote which has a fitness increment of  $s(1 - h)/(1 + hs)$  over the heterozygote, and is therefore  $2s(1 - h)/(1 + hs)$ . The segregational load borne by the asexuals is then said to be

$$W_s = \left( \frac{1 + s}{1 + hs} \right)^n \quad (2a)$$

where

$$n = 2LP_0/P_1 = 2Lh(1 + hs)/(1 - h). \quad (2b)$$

When  $W_s > 2$ , the claim is that sex has overcome its twofold disadvantage and numerical examples with  $L = 10, 50, 100, 500, 1000$  and  $s = .001, .005, .01, .05, .1$  are presented to show that  $W_s$  can be much larger than 2.

The assumptions that underlie these arguments are that at most a single advantageous mutation is segregating in the population at any time, that there is a constant pool of  $L$

loci in the wild-type homozygous state, and that all  $n$  loci which are heterozygous in the asexuals could be of the favored homozygous genotype in sexuals. The interpretation of the constant pool of loci is troublesome because it is obvious from (2b) that if  $h > 1/3$  then  $n > L$ , i.e. the number of loci fixed in heterozygous state can become very much larger than the number of loci in the system. Thus, there is some vagueness in the assumptions concerning how many genes are actually in the model; even if  $L$  is viewed as an average of a fluctuating number of loci, within the constraints of the model it is difficult to entertain  $n$  very much greater than  $L$ . A key assumption, however, is that only one mutant allele is spreading through the population at a time, which is difficult to reconcile with  $n > L$  favored alleles "fixed" in the heterozygous state.

Kirkpatrick and Jenkins note that their value of  $W_s$  does not depend on  $\mu$  or  $N$ , but that the rate at which loci fixed as heterozygotes accumulate in a newly arisen parthenogenetic population does. They illustrate this with a numerical example using  $N = 10^5$  and  $\mu = 10^{-9}$ . They then claim that their assumption that a single advantageous mutation is segregating at a time is justified if  $4NL\mu \ln(2Ns)$  is of smaller order of magnitude than 1. We have set  $h = 1/2$  and examined the examples they used to illustrate  $W_s > 2$ , together with  $N = 10^5$  and  $\mu = 10^{-9}$  in order to determine the magnitude of  $4NL\mu \ln(2Ns)$ . The following Table 1 gives the values in the cases where  $W_s$ , computed from (2) is greater than 2. It is obvious that  $4NL\mu \ln(2Ns)$  is not of smaller order than 1 for  $L \geq 50$ . Further, if a mutation rate of  $10^{-8}$  were used instead of  $10^{-9}$ , then no entry of Table 1 would be of smaller order of magnitude than 1 and some become quite large.

**Table 1 here.**

In light of this, the condition on  $4NL\mu \ln(2Ns)$  warrants further investigation. One heuristic way to derive a condition under which only one mutation spreads through a sexual population at a time is as follows. Denote the probability that a new mutation that is initially at the frequency  $1/2N$  and has advantage  $s/2$  achieves the frequency 0.05 by  $p_s^{2N}(.05)$ . The rate of production of favorable mutations in the population is  $2NL\mu$  per generation. Thus the rate of production of mutations that achieve a frequency of 0.05 is  $2NL\mu p_s^{2N}(.05)$  per generation and the average time between the occurrence of

these is  $[2NL\mu p_s^{2N}(.05)]^{-1}$  which we write as  $T(s, L, 2N; 0.05)$ . The frequency 0.05 is used as a reasonable representation of polymorphism. Let the expected time to fixation (conditional on fixation), starting from frequency  $p$ , of a favorable mutant be  $t_1(p)$ . Then if  $t_1(p) \ll T(s, L, 2N; 0.05)$  it is most likely that only one mutant would be segregating at any time. Computation of  $t_1(p)$  with  $p = 1/2N$  was carried out by numerical integration of formula (17) of Kimura and Ohta (1969) in the case  $h = 1/2$ . The relevant formulae in this case are given as (5.51) and (5.52) in Ewens (1979). The lower and upper bounds of the integrals were set at  $10^{-10}$  and  $1 - 10^{-10}$ . For the calculation of  $p_s^{2N}(.05)$  we used the well-known formula (4.35) of Ewens (1979 p. 123). The results are reported in Table 2, where the value of  $\mu$  was set at  $10^{-7}$  and the values of  $s$  and  $L$  reported are those used by Kirkpatrick and Jenkins. Clearly we cannot say  $t_1(1/2N) \ll T(s, L, 2N; 0.05)$  except for  $N = 100$  and  $1000$  with  $L = 10$  and  $N = 100$  with  $L = 50$ . Ewens (personal communication) has pointed out that with  $h = 1/2$  as  $Ns$  becomes large (with  $N$  large as well) the value of  $t_1(1/2N)$  is extremely well approximated by  $4[\gamma + \ln(2Ns)]/s$  where  $\gamma$  is Ewen's constant, approximately 0.577 and this certainly is in excellent agreement with the numerical integration. This clarifies the relationship between Tables 1 and 2. In general, using the criteria described above, the time to fixation is often greater than the average time between mutations so that during the average time it takes a mutation to fix other mutations will arise. Results like this are, of course, sensitive to the value of the mutation rate which means that  $s$  and  $L$  alone are insufficient descriptors of the process.

A final point concerning the use of  $P_0$  and  $P_1$  and the assumption of equilibrium should be made. According to Kirkpatrick and Jenkins at equilibrium the rate of production of favorable homozygotes in the asexuals is  $\mu N n P_1 = 2\mu N L P_0 = 2\mu N L s$  if  $h = 1/2$  at equilibrium. Under the same assumption,  $h = 1/2$ , the probability of fixation in the favored homozygote in a sexual population is also  $P_0$  so that in a sexual population the rate of production of favored homozygotes is the same as that of heterozygotes and homozygotes in the asexual case. This suggests that there may be no genetic load on the asexuals unless there are different processes of recruitment of new genes to maintain  $L$  constant in the sexual and asexual cases (or distinct processes of removal of the favored homozygotes, due for example to fluctuating environments). In fact, a case can be made for an advantage to

the asexuals which can accumulate advantageous heterozygotes, unlike the sexuals.

One of the criticisms often made of load arguments of the kind used by Kirkpatrick and Jenkins is that they do not actually address the process of competition that would occur between the types being compared. The same remark applies to the comment by Hedrick and Whittam (1989) who compare the frequency of the advantageous mutant in deterministic populations with different amounts of sexual reproduction. They do not ask whether sexual reproduction outcompetes asexual. We proceed to address this using a finite population simulation in which sexual and asexual individuals are in the population at the same time, competing with one another.

### **A Stochastic Simulation of Competition Between Sexuals and Asexuals.**

All of the following results are for a population of 100 individuals where *sexuals produce only sexuals* and *asexuals produce only asexuals*. This would be the case if the sexual-asexual dichotomy were controlled by one locus with the sexual phenotype recessive to the asexual. Each simulation was repeated 1000 times. The model is a Monte Carlo simulation where individuals mate, reproduce, undergo selection and mutation, and then form the next generation. The basic scheme is as follows: an individual is chosen at random and if it is sexual, another sexual is chosen at random as its mate and an offspring produced, receiving one chromosome from each parent. If it is asexual, it produces a clone which carries both of its chromosomes. We examined two models of sexual reproduction which we called hermaphrodites and separate sexes. In the hermaphrodites model, sexuals can produce both male and female gametes so that each individual can function as either mother or father. In the two-sex model, sexuals are marked as male or female, asexuals as female. In the initial population, the sexuals are divided equally into males and females. The program searches for a female and if she is sexual, it then searches for a male mate and reproduction occurs. Again, the offspring receives one chromosome from each parent. The sex of the offspring is determined at random with an equal probability of being male or female. If the mother is asexual, she produces a female clone of herself so that the offspring receives both of her chromosomes. In the two sexes model the "cost of sex" exists because at each mating, only half of the sexuals are "eligible" to be chosen as the primary parent

(i.e. the females), whereas in the asexual population, and in the hermaphrodites model all individuals are eligible. While males contribute to the next generation by mating, the effective size of the sexual population is the number of females. Since the sexual part of population is, on average, half male, its size is halved relative to the asexual population every generation.

After an offspring is produced, it undergoes selection. The selection scheme is multiplicative in accordance with the model of Kirkpatrick and Jenkins described above. That is homozygous mutants have a fitness value of  $1 + s$ , heterozygotes have a fitness value of  $(1 + hs)$ , and homozygous wild-types have a fitness value of 1. For example, an individual with two homozygous wild-type loci, one heterozygous locus and 9 homozygous mutant loci has relative fitness value of  $(1 + hs)(1 + s)^9 / (1 + s)^{12}$ . If an individual's relative fitness value is greater than a random number chosen uniformly in  $[0,1]$ , then it enters the next generation. If the individual is not saved, a new set of parents is chosen and a new offspring produced. This continues until an offspring enters the next generation. The final step in producing an offspring is mutation to the advantageous alleles at a rate which we set at 0.001 for each locus. Mutation is recurrent and unidirectional. Once the offspring has undergone mutation, a new parent is chosen and the whole cycle continues until an entire new generation is produced. Parameters in the model include  $s, h$  (which was set to 0.5 for comparison with Kirkpatrick and Jenkins), the initial frequency of sexuals, mutation rate ( $\mu$ ), and  $L$  (either 10 or 20), the number of loci under selection.

## Results

The results for the hermaphrodites model are reported in Table 3. For  $s = 0.01$ , the sexuals "win" (fix in the population) with about the frequency to be expected under a neutral model (approximately their initial frequency in the population). Kirkpatrick and Jenkins predict that sexuals will do better as  $s$  increases since the load advantage to sexuals will increase. In our simulation, this pattern holds when the initial number of sexuals is low (4, 20, 50) but not large (92, 96, 99). Presumably in the last three cases drift is the overriding force.

The simulations using two sexes are an attempt to address the case where sexuals

have a two-fold cost with respect to fertility. The results are reported in the first two columns of Table 4. In these simulations, the sexuals win only if they start at very high frequency (they do not win for initial numbers of 92 and under). When they start at 96, they win, on average, 0.2% of the time. (Here drift dominates so that there is no apparent relationship with  $s$ .) When they start at 99, they win, on average, 19.5% of the time. When the viability of the asexuals is halved in this model, the last column of Table 4 shows that the sexuals do better but not as well as in the hermaphrodites model, implying that this two-sex model imposes a cost on sex that is greater than two-fold.

In order to assess how long it takes for the build-up of mutations to have some effect on the dynamics, we simulated populations that were either completely sexual or completely asexual and compared the mean fitness of the population at set time intervals. These results are seen in Figure 1. The sexual and asexual populations do diverge but not immediately. After 50 generations, the sexual and asexual populations had equal fitnesses (for all three values of  $s$ ). For  $s = 0.10$ , the mean fitness of sexuals grew much faster than the asexuals soon after 50 generations. For  $s = 0.01$ , the mean fitnesses remained nearly constant and almost equal. For comparison, we kept track of the average number of generations until fixation in the two-sex simulations where sexuals were eliminated. These increased from around 2 to 10 generations as the initial number of sexuals increased. Thus, the exclusion of sexuals by asexuals appears to occur on a much shorter time scale than the build-up of advantageous mutations in a finite population.

### **Is the Cost of Sex Two-fold?**

Since the simulations of the two-sex model in which the viability of asexuals was halved suggested that the cost of sex is greater than two-fold in this model, we used simulations to investigate other factors that might have influenced the results. The first set of simulations tests the effect of a build-up of mutations. The second set tests the effect of a skew in the sex ratio in the two-sex model.

To test the effect of mutation, we made two changes to the model. First, we set  $s = 0.0$  in the two-sex model in order to see whether selection and mutation were playing a role. The results of this simulation are in Table 5 and are somewhat difficult to interpret since

the results from the comparable simulation with  $s = 0.01, 0.05$  or  $0.10$  did not show a clear relationship between success of sexuals and  $s$ . Nevertheless it appears from the average success over  $s$  values in the above set that setting  $s = 0.0$  had no effect on the frequency of wins by sexuals. The sexuals did about as well as in the  $s = 0.05$  case for initial values of both 96 and 99. (As in the  $s \neq 0.0$  simulation, sexuals did not win with initial values of 92 or less.) The second change we made to the model was to double the mutation rate in the sexuals for  $s = 0.01, 0.05, 0.10$  to see whether the size of the mutation rate could have an effect. As can be seen in Table 5, the sexuals did a little better but not enough to explain their earlier failure. With initial values of 99, the sexuals won approximately 20.4% of the time. This is slightly greater than 19.5% in the original model (where there are equal mutation rates for the sexuals and asexuals).

The other variation on the original model was designed to test the effect of skewed sex ratio. It is possible that sexuals may have less than half the overall fitness of the asexuals because there is a chance that the sexual subpopulation will fix on one sex and be lost to subsequent generations which signifies an automatic loss for them. In order to look at the effect of skew in the sex ratio the model was changed in two ways. First, we modified the program so that the sex ratio was set to 50:50 (or as close as possible, with any excess set as females) at the end of each generation. The results of this change are in the third column of Table 4 and are again not very informative. Comparing the last three numbers of the first and third columns, the sexuals do not seem to do any better than in the original model. The second change we made was to modify the hermaphrodites model so that mating between sexuals produced an offspring with a probability of  $1/2$ , relative to the asexuals. Comparison of the last six numbers in the third column of Table 3 with the last six numbers in the first column of Table 4 shows that this has little effect. With these starting conditions random drift is undoubtedly by the major influence.

## Discussion

The overall conclusions that we draw from this study are that, in general, Kirkpatrick and Jenkins' argument appears not to work at least in a finite population. This should not be regarded as surprising since our models do not involve load arguments and include

the following three factors: (1) we use a fixed number of loci, so that fixation of a mutant reduces the number of wild-type loci, (2) our assumption of recurrent mutation permits more than a single mutation in the system at any time, and (3) in our model sexuals appear to be eliminated from the population before they have a chance to successfully compete against asexuals with regard to the acquisition of advantageous mutations. Our model also involves a kind of group selection because there is no gene flow between the sexuals and asexuals. Kirkpatrick and Jenkins compare the two species only after attainment of the equilibrium defined by (1), whereas in our model there is *competition* to produce a fixed number in the population at every generation. The selection process at the viability loci is much slower than the competition process between the sexuals and asexuals. We conjecture that gene flow, or segregation among the sexuals and asexuals would have even worse consequences for the sexuals. In fact, in a few simulations with sexuals dominant to asexuals the former fixed less often than in Tables 3 and 4 above. The cost of sex appears to be higher than two-fold but we have been unable to better describe it any more specifically in the relatively small populations we studied. It may be that the development of skew in the sex ratio (due to demographic stochasticity) has an effect on the overall fitness of the sexuals but this will not be clear without a more complete statistical analysis.

As tables 1 and 2 reveal the assumption that only one advantageous mutation is segregating may not be tenable. Kirkpatrick and Jenkins made the assumption to preclude any “advantage to sexual reproduction derived from recombination”. The mutation rate of  $10^{-3}$  to advantageous alleles in our simulations almost certainly entails that more than one mutation is segregating at some time during the evolution of our population. Recombination is not an issue since only one locus is examined. Although the criterion (1) does not involve  $\mu$  the rate at which the “equilibrium” that produces (1) is attained is faster the higher is  $\mu$ . If (1) is the appropriate criterion, then its validity should be enhanced by a higher mutation rate to advantageous alleles. In other words it is unlikely that the inability of the sexuals to outcompete asexuals in our simulation is due to the mutation rate of  $10^{-3}$ .

If the mutations occurred at different loci with recombination, it is not at all clear that the presence of recombination would benefit the sexuals. With multiplicative fitnesses

recombination is expected to provide no advantage (Maynard Smith, 1968). With epistasis it may be advantageous or disadvantageous (Eshel and Feldman, 1970) depending on the shape of the fitness surface. The effect of drift on the advantage of recombination is ambiguous, although the work of Karlin (1973) suggests that the expected time to fixation in the multiple favorable mutant chromosome is increased by recombination. It therefore appears that a stochastic treatment of the process of accumulation of favored mutations does not support the idea that segregation either with or without recombination provides sufficient advantage to the sexuals that they overcome asexual competitors.

**Acknowledgment:** The authors are grateful to Professor Warren Ewens for numerous discussions and comments on the manuscript and for sharing his unpublished results, and to Professor Marcy Uyenoyama who made a number of important suggestions.

## References

- Eshel, I. and M.W. Feldman. (1970) On the evolutionary effect of recombination. *Theor. Pop. Biol.* 1: 88-100.
- Ewens, W.J. (1979) *Mathematical Population Genetics*. Springer Verlag, Berlin.
- Hedrick, P.W. and T.S. Whittam (1989) Sex in diploids. *Nature* 342: 231.
- Karlin, S. (1973) Sex and infinity: A mathematical analysis of the advantages and disadvantages of recombination. In Bartlett, M. and R. Hiorns (eds.) *The Mathematical Theory of the Dynamics of Biological Populations*. Academic Press, New York.
- Kimura, M. and T. Ohta (1969) The average number of generations until fixation of a mutant gene in a finite population. *Genetics* 61: 763-771.
- Kirkpatrick, M. and C.D. Jenkins (1989) Genetic segregation and the maintenance of sexual reproduction. *Nature* 339: 300-301.
- Michod, R.E. and B.E. Levin (1988) *The Evolution of Sex*. Sinauer, Sunderland, Mass.
- Maynard Smith, J. (1968) Evolution in sexual and asexual populations. *Amer. Natur.* 102: 469-473.



**Table 1:**  $4N\mu L \ln(2Ns)$ ;  $N = 10^5$ ,  $\mu = 10^{-9}$

$s \backslash L =$	10	50	100	500	1000
0.001					2.12
0.005				1.38	2.76
0.01			0.30	1.52	3.04
0.05		0.18	0.37	1.84	3.68
0.1	0.04	0.20	0.40	1.98	3.96

Here  $h = 1/2$

**Table 2:  $t(1/2N)$  and  $T(s, L, 2N; 0.05)$  with  $\mu = 10^{-7}$**

	$s$	$t(1/2N)$	$T(.05)$ $L = 10$	$T(.05)$ $L = 50$	$T(.05)$ $L = 100$	$T(.05)$ $L = 500$	$T(.05)$ $L = 1000$
N = 100	.001	398	49,776	9,955	4,978	996	498
	.005	394	48,893	9,779	4,889	978	489
	.01	379	47,820	9,564	4,782	956	478
	.05	220	40,339	8,068	4,034	807	403
	.1	140	33,213	6,643	3,321	664	332
N = 1000	.001	3,795	45,605	9,521	4,761	952	476
	.005	2,213	38,445	7,889	3,945	789	394
	.01	1,407	31,764	6,353	3,176	635	318
	.05	413	10,183	2,037	1,018	204	102
	.1	234	5,254	1,051	525	105	53
N = 10 <sup>4</sup>	.001	14,079	31,622	6,324	3,162	632	316
	.005	4,137	9,957	1,991	996	199	100
	.01	2,347	5,025	1,005	502	100	50
	.05	598	1,025	205	103	21	10
	.1	326	525	105	53	11	5
N = 10 <sup>5</sup>	.001	23,480	5,002	1,000	500	100	50
	.005	5,986	1,003	201	100	20	10
	.01	3,270	503	101	50	10	5
	.05	782	103	21	10	2	1
	.1	418	53	11	5	1	0.5

Note  $T(.05)$  stands for  $T(s, L, 2N; 0.05)$ .

**Table 3: Number of Wins By Sex Out Of 1000. Hermaphrodites Model**

Initial % Sexual	s	10 loci	20 loci	Sexuals Fertility
				Halved (10 loci)
4	0.01	37	39	0
	0.05	51	54	0
	0.10	79	58	0
20	0.01	224	*	0
	0.05	256	*	0
	0.10	269	*	0
50	0.01	498	*	0
	0.05	549	*	0
	0.10	586	*	0
92	0.01	928	943	0
	0.05	942	942	0
	0.10	934	959	0
96	0.01	965	962	1
	0.05	970	968	0
	0.10	966	977	1
99	0.01	992	988	194
	0.05	991	993	205
	0.10	989	993	218

\*Not done.

**Table 4: Number of Wins By Sex Out Of 1000. Separate Sexes Model**

Initial % Sexual	s	10 loci	20 loci	even	asexual
				sex ratio	viability
				10 loci	halved
					10 loci
4	0.01	0	0	0	13
	0.05	0	0	0	5
	0.10	0	0	0	20
20	0.01	0	*	0	88
	0.05	0	*	0	117
	0.10	0	*	0	150
50	0.01	0	*	0	317
	0.05	0	*	0	384
	0.10	0	*	0	431
88	0.01	0	*	0	*
	0.05	0	*	0	*
	0.10	0	*	0	*
92	0.01	0	0	0	*
	0.05	0	0	0	*
	0.10	0	0	0	*
96	0.01	2	1	1	*
	0.05	3	4	1	*
	0.10	1	4	0	*
99	0.01	201	194	216	*
	0.05	195	212	192	*
	0.10	188	194	179	*

\*Not done.

**Table 5**  
**Number of Wins By Sex Out of 1000. Tests of Mutation Effects**

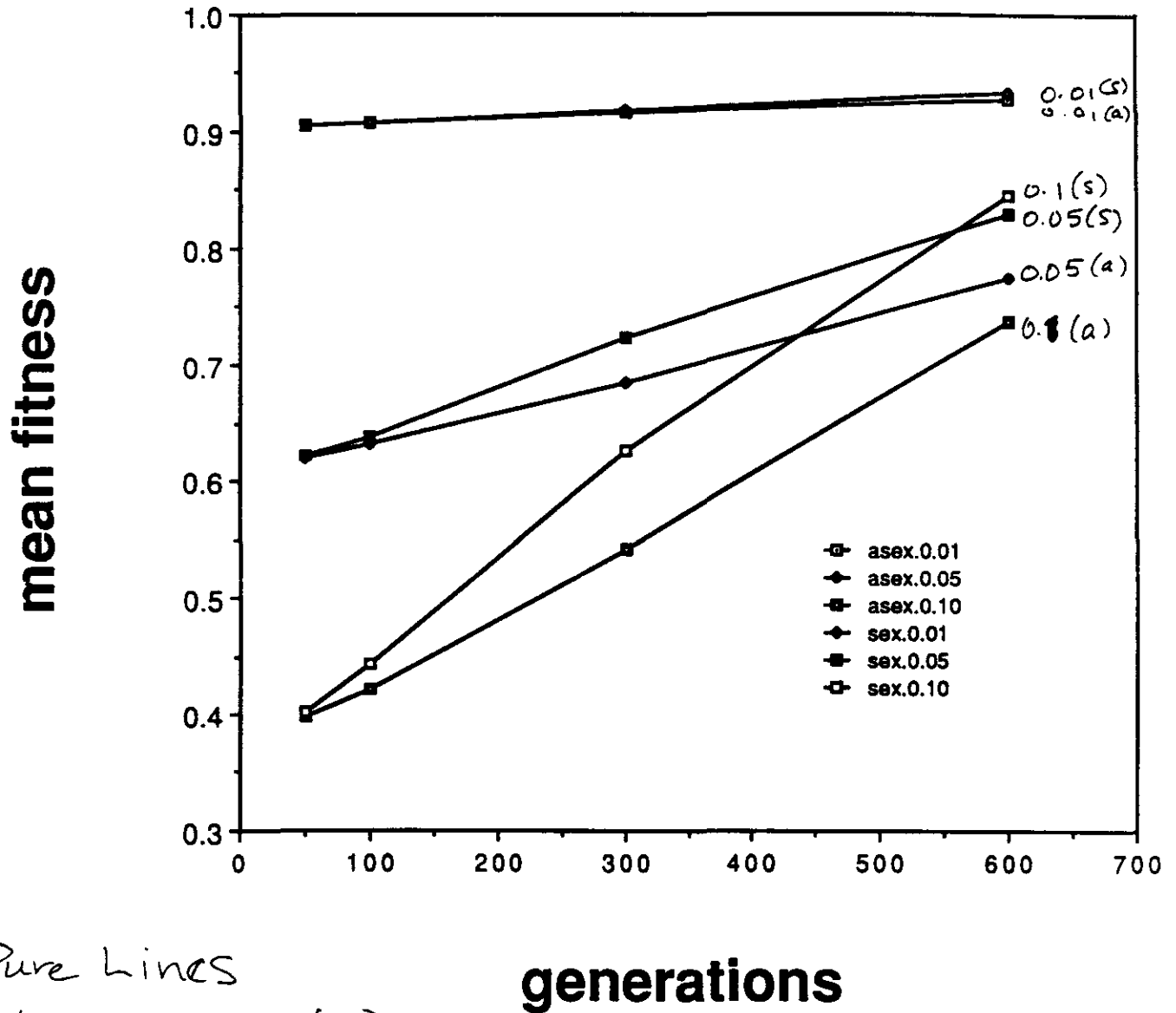
Initial % Sexual	s	10 loci
4	0.0	0
20	0.0	0
50	0.0	0
88	0.0	0
92	0.0	0
96	0.0	3
*96	0.01	1
*96	0.05	2
*96	0.10	4
99	0.0	191
*99	0.01	206
*99	0.05	203
*99	0.10	204

\*Mutation rate for sexuals 0.002, mutation rate for asexuals 0.001.

### Figure Legend

Figure 1. Comparison of mean fitness over time between purely sexual and purely asexual populations with advantageous mutation. The selective advantage of mutant alleles,  $s$ , takes values 0.01, 0.05, and 0.10 and  $h = 1/2$  in both cases. The population size is 100 and recurrent mutation occurs at rate 0.001 per locus per generation. In the sexual case mating is by random union of gametes.

Pop = 100



Pure Lines  
(No competition)

(s) refers to sexuals  
(a) refers to asexuals

Figure 1.

