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**EMERGENCE AND MAINTENANCE OF SEX AMONG DIPLOID ORGANISMS
AIDED BY ASSORTATIVE MATING**

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ABSTRACT

Using computer simulations I studied the simultaneous effect of variable environments, mutation rates, ploidy, number of loci subject to evolution, and random and assortative mating, on various reproductive systems. The simulations showed that mutants for sex and recombination are evolutionarily stable, displacing alleles for monosexuality in diploid populations mating assortatively under variable selection pressure. Assortative mating reduced excessive allelic variance induced by recombination and sex, especially among diploids. Results suggest a novel adaptive value for sex and recombination and show that the adaptive value of diploidy and that of the segregation of sexes is different to that of sex and recombination. The results suggest that the emergence of sex had to be preceded by the emergence of diploid monosexual organisms and provide an explanation for the emergence and maintenance of sex among diploids and for the scarcity of sex among haploid organisms.

INTRODUCTION

What selective forces maintain sexual reproduction and genetic recombination in nature? The answer to this question is an elusive mystery (Maynard-Smith 1978, Judson and Normak 1996, Hurst and Peck 1996). Asexual reproduction is theoretically much more likely to occur than sexual one due to at least three inherent advantages: parthenogenic females do not need to find mates; they produce twice as many daughters and four times as many granddaughters compared to the average sexual ones; and natural selection drives adaptation and thus selection of relevant genetic traits much faster in asexual organisms compared to sexual ones (Maynard-Smith 1978, Jaffe 1996). Despite these relative theoretical advantages of asexuality, most higher organisms are sexual. The various hypotheses put forward to explain this mystery can be grouped into three broad categories:

- 1- The ecological genetic models and the Red Queen Hypothesis which postulate that sex is adaptive in variable environments or variable parasite pressure because it enables genetic variation and the rapid spread and creation of advantageous traits (Bell and Maynard-Smith 1987, Hamilton et al 1990, Ebert and Hamilton 1996, Howard and Lively 1994).
- 2- The mutation-accumulation models (Muller 1964, Hill and Robertson 1966, Kondrashov 1984, 1988, 1994, Taylor and Williams 1982, Heisler 1984), which suggest that sex is adaptive because it performs the efficient removal of deleterious mutations or DNA repair.
- 3- The mate selection models, which assume that sex allows for the selection of 'good genes' by orientating the evolutionary process towards the fixation of beneficial traits (Kodric-Brown and Brown 1987, Jaffe 1996, 1999). Specifically, assortative mating has been shown to be very successful in increasing the fitness of sexual species (Davis 1995, Jaffe 1998).

Regarding the mate selection models, computer simulations showed that non-random mate selection accelerates evolution by focusing natural selection on the appropriate genes (Jaffe 1996), specially in the case of assortative mating (Davis 1995, Jaffe 1998). Mate

selection may explain evolutionary phenomena such as sympatric speciation (Todd and Miller 1991), changing adaptive behavior (Miller and Todd 1994), and behavioral diversity (Werner and Todd 1997). Mate selection strategies aiming at reducing mutation loads were slightly better than random mating (Kondrashov 1988), and assortative mating improved the fitness of simulated populations up to four times compared to random mating (Jaffe 1988).

The concept of assortative mating used here includes passive and active assorting. Active assorting means that individuals chose similar mates by actively searching for them, using mechanisms such as imprinting of sex models (Lorenz 1935, Kendrick et al 1998) for example; whereas passive assorting means that mating may occur among genetically similar individuals due to population viscosity, low motility, inbreeding and/or geographical or ecological restrictions (Peck et al 1998, Queller 1992, Peck 1996), which make mating with neighbors more likely and at the same time make neighbors genetically more similar. In both cases the result is that mating occurs among genetically similar individuals.

Previous work (Jaffe 1999) showed that assortative mating may reduce excessive allelic variability caused by sexual reproduction, especially when selection acts on various loci simultaneously. Assortative mating also allows fit females to maintain (not diluting) their advantageous genetic composition during reproduction, providing their offspring with a similar advantageous genetic outfit. It may accelerate the extinction of sub-optimal genetic combinations, as contrary to strategies selecting for good genes, assortative mating induces suboptimal females to mate with suboptimal males, thus accelerating the extinction of suboptimal alleles. The intensity of mate selection affected the success of assortative mating, as excessive screening for potential mates diminished the efficiency of mate selection, slower so in large populations.

The ploidy of organisms is also relevant to the evolutionary dynamics of sex. Asexual ploidy cycles have been proposed as leading to the origin of sex by reducing mutation load (Kondrashov 1994), and poliploidy has been shown to maintain higher genetic variance under strong selection (Jaffe 1996). However, most studies on the emergence of sex have focused on haploid organisms (Hamilton et al 1990, Kondrashov 1988, Miller and Todd 1994, Peck et al 1998 for example). The genetic complexity (number of loci subject to adaptation) of the simulated organisms also affects the evolutionary advantage of sex and recombination (Maynard-Smith 1978, Jaffe 1998), as does the complexity of the changing environment simulated (Maynard-Smith 1978, Hamilton et al 1990).

The present model allows the study of the dynamic evolutionary interaction between ploidy types, mutation rates, assortative mating, sex determination mechanisms, variable numbers of loci subject to adaptation and changing environmental selection pressures, showing features which are not evidenced when studying these systems on their own, but which emerge as a result of the interaction.

METHODS

The model used here (Jaffe 1996, 1998, 1999), has been shown to have better precision than analytical models in explaining experimental data regarding the emergence of genetic resistance to antibiotics and pesticides (Jaffe et al 1997). In this multi-agent, adaptive model, each individual was simulated as an autonomous agent which interacted with the environment and with other individuals according to five evolutionary steps (see below) and to the alleles it carried in its set of up to 8 loci as given in Table 1. Simulations were “tournaments” between two alleles coding for reproductive strategy (gene 1, Table 1), in interbreeding populations. When creating the initial population, each individual was outfitted with any of two alleles for the gene coding for reproductive strategy (gene 1 in Table 1) and with a set of alleles chosen at random (uniformly distributed) for each of the remaining genes. All organisms had the same allele for gene 0 in each simulation. The temporal variations of the relative frequency in the population of the two alleles coding for reproductive strategy was monitored. The tournament tested which allele for reproductive strategy was able to displace the other. Results represent the average frequency of a given allele after a given number of time steps, calculated from hundreds of simulations having the same parameters but initiated with different random seeds. The random seeds were also chosen at random.

The population of agents (organisms) after being created with a given random seed, suffered a 5 step evolutionary process which mathematically speaking (the program was built in visual-basic) is equivalent to the following:

Mate selection: Females of bisexual species choose a male of the same species, whereas hermaphrodites mated with a conspecific individual. When random mating was simulated, females and hermaphrodites mated with a randomly chosen mate, whereas in assortative mating females and hermaphrodites mated with the genetically most similar mate among 20 randomly chosen individuals (This number had been shown to be close to the optimal for assortative mating to work under the present set of parameters, see Jaffe 1999). Genetic similarity was estimated by comparing the phenotypes of both individuals. In some rare moments of some simulations no sexually mature mate was found by some females or hermaphrodites. Then the individual did not reproduce during that time step if bisexual, or reproduced monosexually if hermaphrodite. The simulations did not distinguish between mate selection and gamete selection, as the model simulated the transmission of only one gamete in each mating act.

Reproduction: The reproductive strategy could be for haploid (**H**) or diploid (**D**) organisms. If sexual (i.e. not monosexual) organisms could mate randomly (**RM**) or assortatively (**AM**). Thus, ten different reproductive strategies were simulated. Monosexuals simulated parthenogenesis or thelytoky. That is, monosexual organisms did not mate. In **H-Monosex** (monosexual haploids), the individual transmitted all its genes to the offspring (cloning) with no variance except that allowed by mutations, simulating asexuality. **D-Monosex** (monosexual

diploids) did not mate and produced offspring by uniform random crossovers of the alleles in each loci of the parent. Bisexuals (either H- or D- and -RM or -AM) produced equal numbers of males and females randomly (**Bisexual-r**) or produced a biased ratio of 60 % more females (**Bisexual-b**). Males could mate several times each reproductive step. Hermaphrodites (either H- or D- and -RM or -AM) produced only females and reproduced similar to bisexuals if finding another hermaphroditic female (**Herma1**) or any female (**Herma2**), or else reproduced as the corresponding H- or D- monosexuals. Herma1-RM, thus, mated assortatively with females having the same disposition for sex, even when mating randomly regarding all other loci.

Females produced offspring according to their phenotypically determined clutch size (see below), transmitting their genes following Mendelian rules of reproduction (free recombination). If sexual, each parent provided half of its alleles to the newborn, so that for each locus, one allele came from each parent if diploid, or each parent had a probability of 0.5 to transmit its allele to each locus if haploid.

Variation: In each offspring, randomly selected genes mutated, changing their allelic value randomly in those loci which allowed for allelic variance, with a probability determined by their allele in gene 2 (Table 1).

Phenotypic expression: As commonly done with genetic algorithms and as it is known to occur frequently in real organisms, total allelic dominance was simulated. That is, in diploid organisms, only one allele per loci was expressed phenotypically during the lifetime of each organism, which was selected randomly at birth. In simulations comparing the relative evolutionary success of two alleles and which in addition aimed to assess the effect of allelic dominance on the competition between the two alleles, the dominant allele, defined by the experimenter, was phenotypically expressed if present in the diploid genome. For example, in experiments assessing the relative evolutionary between diploid hermaphrodites and diploid monosexuals (Fig 1) when the allele for hermaphroditism was programmed as dominant, then, if the corresponding allele was present in the organism, the individual would behave as a diploid hermaphrodite.

Selection: The model did not assume any simplified expression of fitness but reproduction and individual survival were decomposed into different aspects for selection to act. Individuals were excluded from the population at the end of each time step when any of the following criteria applied:

1- Their age exceeded their genetically prefixed life span.

2- When randomly selected with a probability which increased with population density as given by the formula:

$$0 \text{ if } r_1 * N_t \geq ops * r_2$$

survival of individual i at time step $t = \{$

$$1 \text{ if } r_1 * N_t < ops * r_2$$

where ops is the optimal population size, N_t the population size at time-step t and r_1 and r_2 are random numbers between 0 and 1

3- Individuals not possessing the resistant phenotype of genes 6 to 8 in Table 1 were killed randomly each time step with probabilities which varied randomly each time step from 0 to 0.6, simulating an environment in which two different biocides or parasites trimmed the population by killing non resistant individuals.

Optimal size of populations was 400 and the initial size of the populations was 200 individuals.

RESULTS

The results of the various possible combinations of variables explored with over 120,000 simulations are summarized in Figures 1 and 2. In these figures, data points greater than one indicate that the respective allele was more frequent than the corresponding monosexual variety. The most striking result is that alleles coding for sexuality displaced corresponding alleles coding for monosexual strategies when simulating hermaphrodites and diploid bisexual organisms which mated assortatively and produced more females than males. For example, simulated diploid hermaphrodites of type 1 (D-Herma1) organisms mating assortatively displaced diploid monosexual (D-Monosex) organisms which did not mate but reproduced asexually in a ratio $>4/1$ when simulations included 8 loci with allelic variance (Fig 1). Haploid organisms or diploids using random mating did not succeed in displacing the corresponding monosexual variety.

The relative advantage of sexuality over monosexual reproduction was dependent on a variety of factors in a non-linear pattern. More than four loci with allelic variance in the simulated organisms was needed for sex alleles to displace monosexual ones (Fig 1), and low mutation rates favored sexuality (Fig. 2). As had been shown before (Maynard-Smith 1978, Peck et al 1997) small population sizes also favored sex (not shown).

Experiments in which all individuals were monosexual in the initial population and during the simulation sexual organisms were allowed to appear through random mutation, or vice versa (Table 2) showed that alleles coding for sexual diploid hermaphrodites with assortative mating are evolutionarily stable vis-a vis alleles coding for monosexual reproduction and are clearly able to invade and displace monosexual diploid alleles from the populations. Alleles coding for other diploid hermaphroditic sexual strategies, although to a lower degree, may also displace eventually monosexual alleles. Bisexuality seemed unable to

displace monosexuality in this scenario.

The effect of allelic dominance on the evolutionary dynamics was studied using the simulation of populations consisting of a mix of D-Herma1 and D-Monosex with 7 variable genes. The simulations using random allelic expression of the phenotype (See Figure 1, Assortative mating) gave a mean value of $3.87 \pm \text{sd } 1.06$ more alleles coding for D-Herma1 than those coding for D-Monosex under the conditions described in Figure 1. When simulating the same situation but including allelic dominance, so that the alleles coding for D-Herma1 were dominant and those coding for D-Monosex were recessive, the relative advantage for D-Herma1 alleles increased (mean ratio = 5.43 ± 1.36). When simulating dominance of D-Monosex so that D-Herma1 alleles were recessive, the evolutionary advantage of D-Herma1 alleles remained, although to a lower degree (mean ratio = 2.48 ± 0.29). Repeating experiments in Table 2 using dominant alleles for sex (D-Herma1-AM) increased the likelihood for successful displacement of D-Monosex by D-Herma1-AM from 52 % to 71 %, whereas when sex alleles were modeled as recessive, this likelihood decreased to 30 %. That is, although simulation of dominance affected the results, the evolutionary advantage of sex was maintained even if genes coding for sex were recessive.

DISCUSSION

It has to be noted that assortative mating here, although modeled having mate selection in mind, can be extrapolated to gamete selection. For example, most sexual marine invertebrates simply spawn their gametes. The corresponding metaphor in the model would be that female gametes assortatively select male gametes through biochemical mechanisms after contact, or through passive assorting, by the fact that the probability of successful encounters between gametes and of subsequent fertilization is inversely proportional to the distance between the emitters of the gametes. A similar case can be made for sperm selection in which females not only may select males but also chose from different spermatozoa from one or several males (see Haig and Bergstrom 1995 for example). In the light of the findings here described, it would seem worthwhile to simulate sperm production in large amounts explicitly, modeling each spermatozoa with a slightly different allelic load.

The results suggest that the genetic variance produced by sex differs from that produced by random mutations in that sex with assortative mating produces a better blend of variation, allowing faster adaptation in scenarios with very large genetic combinatorial possibilities, if compared to random mating. That is, sex slows the speed of evolution (Jaffe 1996) as advantageous mutations are not always transmitted to the offspring and are often mixed with disadvantageous alleles in other loci during recombination. Assortative mating reduces the extent to which this "dilution effect" of advantageous mutations occurs (Jaffe 1999), by reducing the variance of allelic composition between mates and thus producing offspring which have a greater likelihood of possessing the advantageous genes of their

parents. Thus, assortative mating accelerates the fixation of advantageous alleles in the population canceling the effect of sex in slowing evolution. On the other hand, the long term advantage of sex is that it can produce advantageous blends of alleles faster than asexual reproduction does, but only if the number of loci is large (Jaffe 1998). For genomes with low genetic complexity (number of loci), mutations together with asexual reproduction is faster than sex in achieving optimal allelic combinations in the genome. Thus, the advantage of sex will be evidenced only if organisms do not mate randomly and the simulated genome has sufficient complexity (Ochoa and Jaffe 1999). Simulating diploidy here is in fact a way of increasing genetic complexity, making the advantages of sex much more evident. Our results (Fig. 1) though show that the complexity produced by diploidy is not identical to the complexity produced by an equivalent increase in the number of loci, due to the particularities of meiosis. An approximate representation of the dynamics of these processes is given in Figure 3.

Most studies on the emergence and maintenance of sex have focused on models using random mating. The present results suggest that the emergence of sex seems to be favored when the following conditions are present simultaneously: Variable environments (i.e Red Queen Hypothesis), assortative mating, low mutation rates, small founder populations, diploidy, hermaphroditic sex, and many loci suffering adaptation. These assumptions are compatible with what seems to happen with founder populations in biological allopatric speciation (Mayer 1988). That is, in founder populations variable environments select alleles in several different loci at the same time and assortative mating, i.e., mating among individuals in the founder population, is very likely to occur. Thus, sex, if not present, may emerge with higher probabilities when new species evolve, compared to a scenario where sex emerges in an established species.

The new feature analyzed here, not included in the exhaustive review on the emergence and maintenance of sex by Maynard-Smith (1978), is assortative mating. The most likely scenario for the emergence of sex, in the light of this new findings, is that sex may have emerged from diploid monosexual ancestors, and that mating occurred preferentially among individuals with the same predisposition for sex, creating the most simple form of assortative mating. This means that the evolutionary step from haploidy to diploidy had to precede the emergence of sex, so that organisms evolved from asexual haploids, to haploids which occasionally interchanged genetic information, to monosexual diploids, to bisexual diploids. Sexual haploids, thus, from this point of view, have to derive from sexual diploids. It has been proposed that anisogamy also stabilizes sex (Kodric-Brown and Brown 1987). The present simulations support this suggestion but they also showed that anisogamy is unlikely to displace monosexual reproductive strategies in one single evolutionary step. The body of evidence now available suggests that the adaptive value of diploidy, of recombination in monosexual diploids, of the exchange between organisms of genetic material (i.e. sex), and of anisogamy differ, as each process has a different effect on the evolutionary dynamics.

Assortative mating favors the maintenance of sex and recombination even among bisexuals, overcoming the two-fold disadvantage of sex relative to parthenogenesis. Other mate selection strategies, not explored here (but see Jaffe 1999), may produce similar biases towards sex, as is the case of mate selection criteria looking for “good genes” (Jaffe 1996, 1998). A testable prediction, derived from the simulations, is that sex should be rare or absent among haploid organisms, and that sex is maintained by selective forces which are different from those favoring diploidy (Kondrashov 1994) or polyploidy (Maynard-Smith 1978, Jaffe 1996) .

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Table 1: Genes and their possible alleles defining the agents-organisms. Simulations of genes with allelic variance allowed mutant alleles to appear in the range given below. Initial populations had individuals possessing any of the alleles indicated in that range. In simulations in which some genes had no allelic variance, the default allele, indicated in parenthesis, was assigned to all the corresponding loci in all organisms.

Gene	Range for alleles	Effect on phenotype
0	1-2	Ploidy. Either haploid or diploid.
1	1-6	Reproductive strategy
2	0-10	Mutation rate: from 0.2 to 10^{-7} mutations per gene in logarithmic decrements (0.008)
3	0-10	Maximum life span coding for life spans from 0 to 10 time steps (5)
4	0-10	Clutch size from 0 to 10 offspring (5)
5	0-5	Minimum age for initiating reproduction of females in t-steps (0)
6	0-10	Resistance to biocide 1: Only allele 0 was resistant to that biocide (0)
7	0-10	Resistance to biocide 2: Idem as gene 6 but for biocide 2 (0)
8	0-10	Resistance to biocide 3: Idem as gene 6 but for biocide 3 (0)

Table 2: Percentage of simulations where the strategy which appeared through mutation (invading strategy) displaced eventually all individuals possessing alleles of the original strategy (possessed by all individuals initially) in less than 200 time steps (250 simulations for each pair). A mutant of the strategy not present in the initial population was allowed to appear at random after time step 5. Other parameters as in Fig 1 with 7 loci.

Invading strategy vs original strategy: Percent of successful displacement:

When original population was homogeneously monosexual

D-Herma1-AM vs D-Monosex	52 %
D-Herma1-RM vs D-Monosex	31 %
H-Herma1-RM vs H-Monosex	9 %,
H-Herma1-AM vs H-Monosex	6 %
D-Bisex-b-AM vs D-Monosex	1 %

When original population was homogeneously sexual

D-Monosex vs D-Herma1-AM	7 %
D-Monosex vs D-Herma1-RM	23 %
H-Monosex vs H-Herma1-RM	17 %,
H-Monosex vs H-Herma1-AM	22 %
D-Monosex vs D-Bisex-b-AM	35 %

Figure 1: Frequency of alleles coding for sex relative to those coding for the corresponding monosexual population for varying genetic complexity (number of loci having allelic variance) as given in the x-axis in the order appearing in Table 1. Each data point is the average value at time step 20 (which in average represented 10 generations of agent-organisms) of a minimum of 200 simulations and until standard deviation was < 30 % of the mean. Relative frequency < 1 indicate prevalence of monosexual allele. Data for 1 locus represents evolution with no selection other than caused by the dynamics of the reproductive strategy. Results for H-Herma1 mating randomly seem analogous to those reported in (Hamilton et al. 1990)

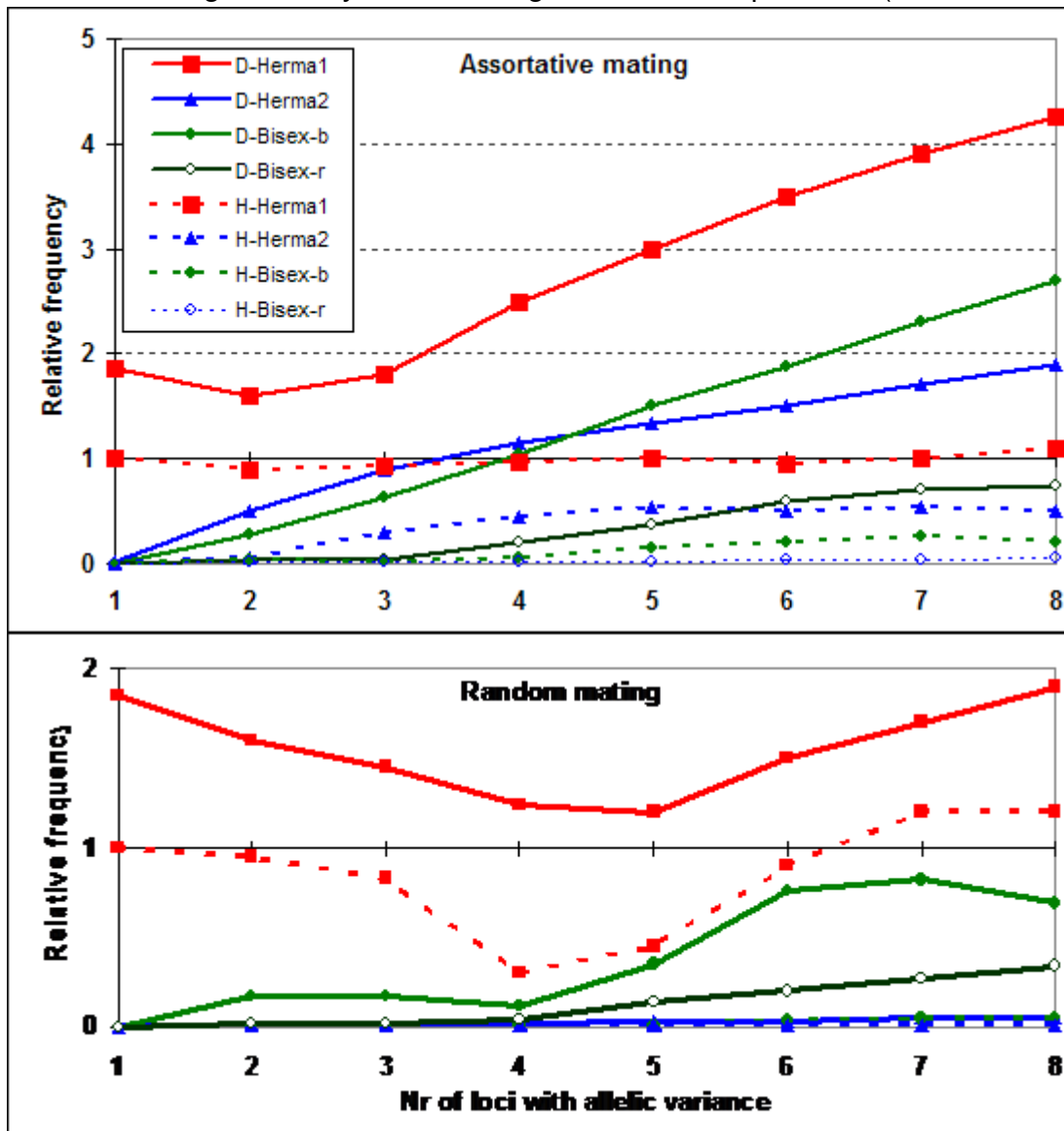


Figure 2: Relative frequency at the end of simulations of alleles coding for sex relative to those coding for monosexuality at different mutation rates. The first seven loci in Table 1, except gene 2, had allelic variance. Mutation rates are given in random mutations per locus per

generation. Else as in Fig 1.

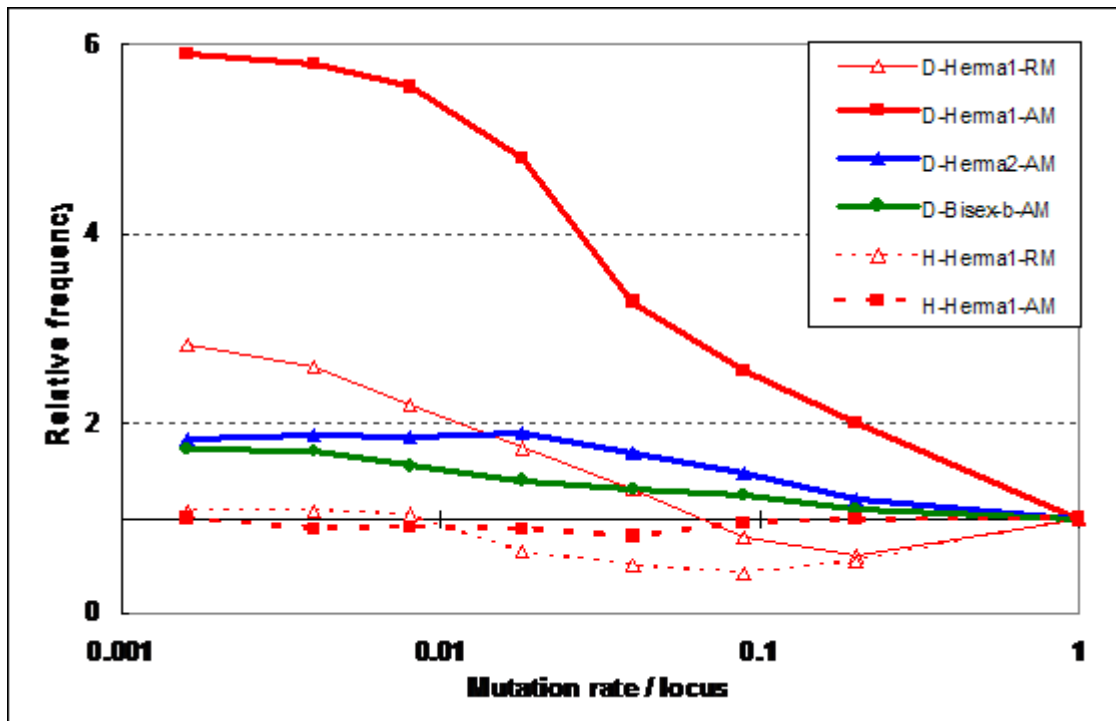


Figure 3: Approximate time course of mean fitness of organisms in an evolving population using different reproductive strategies.

