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Can Males Contribute to the Genetic Improvement of a Species?

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In the time evolution of finite populations, the accumulation of harmful mutations in further generations might have lead to a temporal decay in the mean fitness of the whole population. This, in turn, would reduce the population size and so lead to its extinction. The production of genetically diverse offspring, through recombination, is a powerful mechanism in order to avoid this catastrophic route. From a selfish point of view, meiotic parthenogenesis can ensure the maintenance of better genomes, while sexual reproduction presents the risk of genome dilution. In this paper, by using Monte Carlo simulations of age-structured populations, through the Penna model, I compare the evolution of populations with different repoductive regimes. It is shown that sexual reproduction with male competition can produce better results than meiotic parthenogenesis. This contradicts results recently published, but agrees with the strong evidence that nature chose sexual reproduction instead of partenogenesis for most of the higher species.

KEY WORDS: Recombination; Monte Carlo methods; population genetics; mutations.

1. INTRODUCTION

Recently, Rosemary Redfield published a paper discussing the genetic cost of sex for females.[1] In that paper, by performing computer simulations, she concluded that parthenogenesis may produce higher fitness than sexual reprodution. Note that reproduction is not necessarily tied to recombinations. That production of offspring can occur sexually or asexually, with or without recombination. The simplest form of asexual reproduction is fission (*mitosis*). In this case, the offspring are identical copies of the mother,

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disregarding eventual mutations that may appear in the reproductive process. In fact, some parts of the genetic code may be changed (mutated) during reproduction. This variability might be a strong mechanism to allow the survival of species in time-changing environments, when better fitted individuals (with higher reproductive capacity) can arise in the population. However, the mutations in the reproductive process are generally deleterious mutations. Harmful mutations apparently arise at a rather high rate in most organisms, perhaps as high as one per gamete,[2,3] and it is important to note that the frequency of backward mutations (reverse mutations deleting harmful ones) is about 1/100 the frequency of forward mutations.[4]

Three decades ago, Muller[5] conjectured that in a small asexual population, the random loss of better fitted individuals would lead to the time decay of the population mean fitness, a process called "Muller's ratchet." In this picture, the production of genetically diverse offspring would be the escape from this trajectory to extinction.

In meiotic division, the two members of each pair of chromosomes present in the cell nucleus come together in close union and the strings duplicate, twist, and cross over, breaking at identical positions. The broken ends exchange, *recombining* the genes. Now, each chromosome has a full set of genes, but not identical to the set with which it started. The two doubled chromosomes then pull away from each other to the poles of the cell, *segregating* at random. The mother cell divides and divides again, resulting in four daughter cells—*gametes*—each with half of the number of chromosomes (one of each pair). Asexual reproduction does not imply that recombination is not present. Even some viruses and bacteria can mimic the recombination process. Some multicellular organism reproduce by *parthenogenesis*, i.e., the production and development of uninseminated eggs: meiotic parthenogenesis results in genetically diverse offspring, whereas ameiotic parthenogenesis produces identical genetic copies of the mother (present in some plants, for instance)[6].

If one thinks that in the evolutionary process the main point is genome reproduction, asexual reproduction should be advantageous from the point of view of proliferating an individual genome. Sexual reproduction contains the risk of genome dilution. However, asexual reproduction may lead to a higher accumulation of harmful mutations. Thus, meiotic parthenogenesis could put together the different advantages of different types of reproduction. In her paper, Redfield assumed different mutations rates in the male population: equal to or higher than the female mutation rate. For higher male mutation rates—which she assumed as the natural feature[7]—parthenogenesis should prevail. Recently, it was shown that the results obtained by Redfield change if one assumes some type of dominance

effect in her program.[8] With this change, which may be understood as mimicking certain sexual features, the better fitness was obtained by sexual reproduction.

Recently several aspects of evolutionary theory have been studied by Monte Carlo simulations, particularly after the introduction of the bit-string Penna model[9, 10] (for a review of computer simulations of the ageing problem see ref. 11). This model is an efficient way to simulate the population dynamics in age-structured populations, since each individual can be represented by its genome and the model takes into account the inheritance of deleterious mutations and the natural selection pressure during the evolutionary process. The introduction[12] of a sexual version of the Penna model (used in the present paper) was done in order to study the problem of different survival probalities observed in sexual populations which present gender differentiation. By using this version, I showed that sexual reproduction allows a species to escape from extinction, where an asexual one does not survive, in contradiction to Redfield's results.

The aim of this paper is to compare the evolution and the equilibrium properties of meiotic parthenogenic and sexual populations. In the sexual reproduction case, male competition may or may not be taken into account.

2. MODEL

In this sexual version of the Penna model, a diploid individual is represented by two computer words (with 32 bits), representing the pair of homologous chromosomes present in the cell nucleus. An individual can live at most 32 time intervals. Each gene on its genome is related to one life-threatening disease that could appear starting from a specific age (all diseases have the same detrimental effect). Then, a harmful mutation is represented by a bit = 1, while the normal allelic form is represented by bit = 0. As a diploid has two paired chromosomes, a threatening disease will apear if at the same position (locus) one has both bits = 1. Otherwise, if at the same locus one finds different states (heterozygous), this disease will appear only if the harmful type is dominant. To incorporate dominance I define a dominance parameter h, varying in the interval (0, 1), which is the probability that "1" is dominant in a heterozygous locus. As the genes are placed in temporal order, the survival (or death) of an individual will be defined as follows: an individual dies as soon as the sum of its diseases (up to its actual age) is greater than or equal to some threshold T.

For sexual reproduction an offspring is generated as follows: a female older than R chooses at random a male older than R and they produce

offspring with probability B. The genetic code of the offspring (two computer words) is formed by the junction of the two parents gametes. Each gamete (a computer word) is formed by crossing over the two homologous chromosomes on the same individual, the location of the crossover position being drawn at random. Then the two gametes are combined to form the two words of the genome for the new individual (for more details see ref. 12). After that, the offspring gender is chosen at random: 50% male, 50% female. In case of meiotic parthenogenesis, a female older than R produces a gamete (in the same way as described for sexual reproduction) and this gamete is repeated, generating the zygote (again, a diploid organism).

When a new zygote is formed, new mutations may be acquired, all of them harmful (these mutations are produced at random in the offspring genome, but each mutation only in one locus and one chromosome). Two mutation procedures were implemented: (i) through an **OR** instruction with a mutation rate M; if a randomly chosen bit is already "1," it remains unchanged. (ii) The number of mutations events is given by a Poisson distribution with average $\bar{M}$. In this case, a $0 \rightarrow 1$ operation is always implemented. For small populations, the version with Poisson-distributed mutation events produces more realistic results than that with **OR** instruction[11].

An important aspect of sexual reproduction is the existence of male competition in species which present gender differentiation.[13] To include competition between males we define the parameter T_m, which means that only males that have a number of diseases less than or equal to T_m can be chosen (mutations at the actual age, disregarding the eventual mutations that will appear later). From this definition, if $T_m = T$, there is no competition and any male can reproduce; on the other hand, if, for instance, $T_m = 0$, just the males without diseases can mate.

All the simulations start with mutation-free genomes, i.e., all the bits set to "0". To prevent the population $N(t)$ from growing to infinity, we use a population-dependent Verhulst factor $P_l(N(t)) = 1 - N(t)/N_{\max}$, $N_{\max}$ being the environmental carrying capacity. It describes the probability P_l that an individual survives the next time step, i.e., individuals die randomly with rate $1 - P_l$.

3. RESULTS

Figure 1 shows the results obtained for large populations. These results were averaged over ten independent samples. The simulations started with $N_0 = 10^6$ individuals: either 50% males and 50% females for sexual reproduction or all of them females for meiotic parthenogenesis. The Verhulst factor $N_{\max} = 5 \times 10^6$ and the other parameters are defined in the

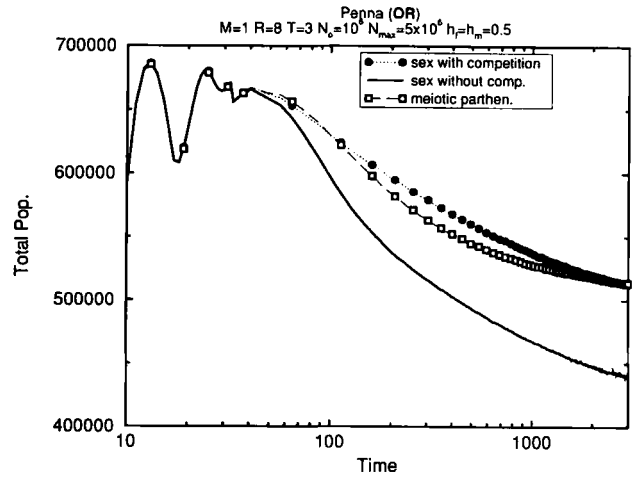

Fig. 1. Evolution of populations for three different reproductive regimes: sexual with and without competition, and meiotic parthenogenesis. $M=1$, $T=3$, $R=8$, $T_m=3$ for sexual reproduction without competition and $T_m=0$ with competition. These results were averaged over ten statistically independent samples using different seeds of the random number generator.

figure caption. In order to compare the evolution of the different populations, I define the reproductive rate $B_s=1$ for sexual reproduction and $B_p=0.5$ for meiotic parthenogenesis. As one can see, in the beginning all three populations evolve in the same way, up to time 40 years. Notice that sexual reproduction without male competition produces worse results than the other two regimes (the smaller population observed). The performances of sexual reproduction with competition and meiotic parthenogenesis seem to be almost the same.

However, when one compares the accumulation of harmful mutation in the final populations (Fig. 2), it is possible to see that the population with meiotic parthenogenesis has a smaller part of their genome filled with harmful mutations. This result represents the average between the two homologous chromosomes. This will cause a higher survival probability for the meiotic parthenogenic population (they live longer) than both with sexual reproduction. Nevertheless, it is important to compare the accumulation of harmful mutations in the younger part of the genomes. Here, the worst result is obtained for the meiotic parthenogenic population, while the best result is obtained for the sexual population with competition. The meiotic parthenogenesis population accumulates more mutations in the younger portion of the genome. The result for large population might lead to the conclusion that meiotic parthenogenesis is better than sexual reproduction.

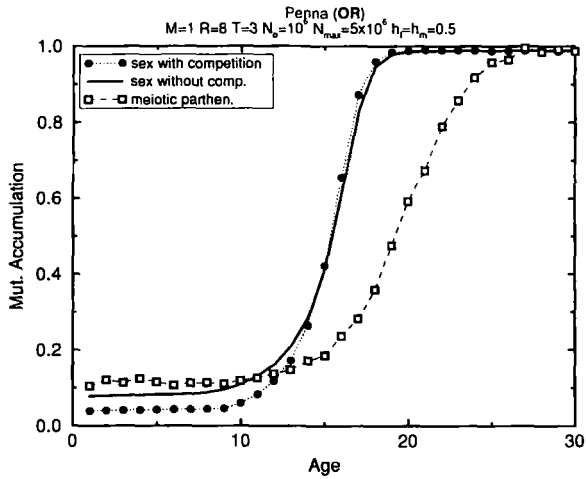

Fig. 2. Mutation accumulation observed in the final populations shown in Fig. 1. For older portions of the genome, sexual reproduction with or without competition gives the same result. For the younger portion, sexual reproduction with competition produces the lowest accumulation of harmful mutations. The meiotic parthenogenic population lives longer, but they accumulate more mutations in the younger portion of the genome.

An important aspect of evolutionary theory is that the problem of accumulation of harmful mutations is stronger and can exert dangerous effects in small populations, through the random loss of better fitted individuals, for instance.[5] Another aspect is the accumulation of slightly deleterious mutations.[14] They may accumulate in the population, leading later to a strong decay in the population mean fitness. By taking into account that usually the effective size of populations is in the order of 10^3–10^4 individuals, we see that an adequate comparison between reproductive regimes should be made for populations of $\sim 10^3$ individuals. Once again, as I have recently discussed,[15] the assumption of a large population which has attained equilibrium may obscure the main result, as one can see below.

Figure 3 shows the results obtained for small populations $N_0 = 2000$ and $N_{\max} = 10^4$ for the same set of parameters of Figs. 1 and 2. Here the results were averaged over 30 samples. The difference between the filled region of the genome was drastically reduced. However, the meiotic parthenogenic population shows again the higher concentration of harmful mutations in the younger portion of the genome. The final populations (not shown) attained the largest value for sexual reproduction with competition.

By introducing Poisson-distributed mutation events, it is possible to see more clearly the effects of accumulation of mutations in small populations.[11] As I have pointed out above, in this case a $0 \rightarrow 1$ operation is

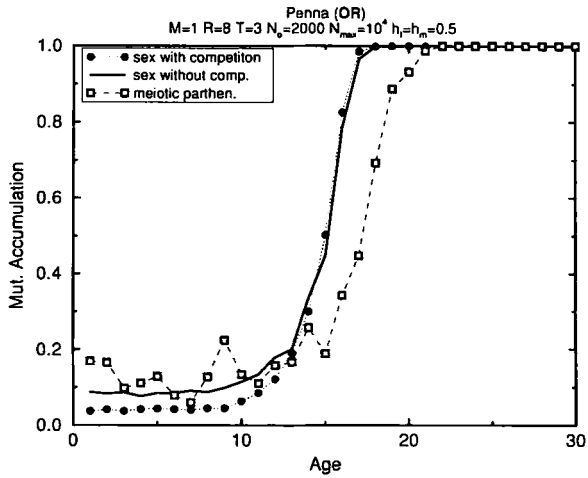

Fig. 3. Mutation accumulation observed in the final populations for small populations: $N_0 = 2000$ and $N_{max} = 10^4$. The other parameters are the same as in Figs. 1 and 2. The same picture of accumulation of harmful mutations in the younger portion of the genome is observed. However, now the difference in the filled portion of the genome is smaller than in the previous case. These results were avaraged over 30 samples.

always performed. Figure 4 shows the results obtained for small populations, $N_0 = 2000$ and $N_{max} = 10^4$, with a higher mutation rate $\bar{M} = 1.4$ and higher threshold of threatening diseases $T = 4$. Reproductive age as well as the dominance coefficients are the same as in the points discussed above. The results represent averages over 30 samples. For sexual reproduction without competition all 30 populations become extinct during at most 4000 generations. In the case of meiotic parthenogenesis, 14 of the 30 samples become extinct before 10,000 generations (up to 20,000 generations, 23 of the 30 samples extinct). The mean population size for the remaining samples is around 300 individuals. On the other hand, for sexual reproduction with competition all 30 populations survive up to 20,000 generations. The final populations fluctuate around 600 individuals.

In conclusion, by introducing recombination in the Penna model, it is possible to show that sexual reproduction produces better results than meiotic parthenogenesis, if one assumes the existence of male competition. For larger populations, meiotic parthenogenesis produces individuals which live longer. However, the proper problem is addressed to small populations. In this case, meiotic parthenogenesis does not avoid the extinction, where sexual reproduction does so. I did not test the hypothesis of a higher male mutation rate (as tested by Redfield). This will be done in the Charlesworth model[16, 17], because this model uses larger

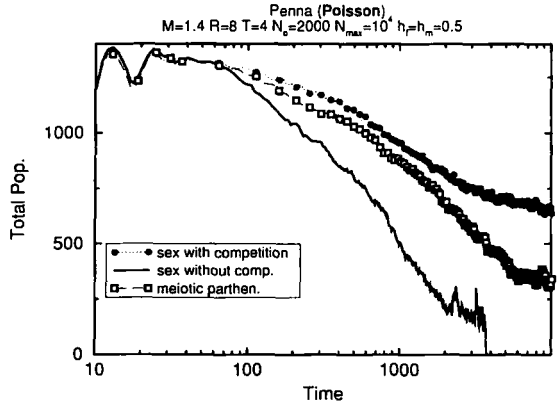

Fig. 4. Evolution of populations with three different reproductive regimes. Now the mutation events are Poisson distributed and a $0 \rightarrow 1$ operation is always performed. Sexual populations without competition become extinct in all the samples. In the case of meiotic parthenogenesis, almost half of them become extinct up to 10,000 generations ($\sim 80\%$ extinct up to 20,000, not shown). In the case of sexual reproduction with competition, no extinction happened up to 20,000 generations.

chromosomes and therefore allows higher mutation rates than in the Penna model.

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