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Sexual selection and its effect on the fixation of an asexual clone

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Sexual selection is a powerful and ubiquitous force in sexual populations. It has recently been argued that sexual selection can eliminate the twofold cost of sex even with low genomic mutation rates. By means of differential male mating success, deleterious mutations in males become more deleterious than in females, and it has been shown that sexual selection can drastically reduce the mutational load in a sexual population, with or without any form of epistasis. However, any mechanism that claims to maintain sexual reproduction must be able to prevent the fixation of an asexual mutant clone with a twofold fitness advantage. Here, I show that despite very strong sexual selection, the fixation of an asexual mutant cannot be prevented under reasonable genomic mutation rates. Sexual selection can have a strong effect on the average mutational load in a sexual population, but as it cannot prevent the fixation of an asexual mutant, it is unlikely to play a key role on the maintenance of sexual reproduction.

Keywords: sexual selection; maintenance of sex; deleterious mutations

1. INTRODUCTION

Sexual reproduction is thought to be twice as expensive as asexual reproduction (Maynard Smith 1978). This so-called twofold cost of sex occurs when the offspring of a sexual female individual contains on average 50% male individuals, and when males do not contribute anything to the reproductive output except their genes. Both assumptions are met in many species (Bell 1982), in which case the average per capita growth rate of sexuals is half that of asexuals. If this reasoning is correct, then there must be an advantage of sexual reproduction, which is at least twofold in order to compensate its cost. Dozens of theories have been proposed that could explain such an advantage (Kondrashov 1993), but there is not yet a common agreement on the theory which is the most relevant to explain the widespread maintenance of sex in natural populations (West *et al.* 1999).

One idea that has often been ignored is that differential male mating success (via sexual selection) can reduce the mutational load in sexuals. Two recent papers, however, have shown analytically that differential male success can pay the full twofold cost of sex when sexual selection is strong (Agrawal 2001; Siller 2001). The main idea is that if females choose fit males that have fewer deleterious mutations than the average male, then the total selection on males is stronger, and consequently, the equilibrium frequency of deleterious mutations is lower in sexual populations. Agrawal and Siller have argued that if sexual selection is strong enough, it can maintain sex even with reasonable genomic mutation rates (Keightley & Eyre-Walker 2000; Gong *et al.* 2005).

One potential problem with this kind of reasoning is that it compares the fitness of two groups, i.e. the sexual population and the parthenogenetic population. This is problematic because the average fitness of a group with a certain wild-type allele and a group with a mutant allele does not tell us anything about whether the mutant allele can replace the wild-type allele. In the case of sex, we must therefore understand under what circumstances a mutant allele causing parthenogenesis can invade and replace the wild-type allele responsible for sexual reproduction. Asexual mutants are often generated directly from sexual females, and are expected to replace a sexual population of any size within a few generations, given the twofold cost of sex (Jokela *et al.* 1997). Therefore, any mechanism that claims to maintain sexual reproduction must be able to prevent the fast fixation of an asexual mutant clone (Charlesworth 1990).

To test whether sexual selection can prevent the fixation of an asexual mutant, I used an individual-based computer model where a single asexual clone arises in a finite sexual population in which strong sexual selection is acting, and observed under which circumstances an asexual mutant succeeds or fails to overtake the population.

2. MATERIAL AND METHODS

I used a modified version of a previously described computer simulation model (Salathé *et al.* 2006), with individuals that are haploid and have 512 loci (termed 'selection loci'), where deleterious mutations can accumulate over time. Additionally, individuals have one locus that defines the mode of reproduction (either sexual or asexual) and another locus that defines the sex of a sexual individual (male or female). Asexual individuals are always female. Initially, N sexuals with randomly chosen sex and without deleterious mutations are created. The completely sexual population can then converge towards a mutation–selection equilibrium for 200 generations, after which an asexual mutant is introduced into the population by choosing a random sexual female individual and switching its reproduction mode to asexual. In each generation, the following processes occur: reproduction (with previous sexual selection in the sexual population), mutation and natural selection. The following describes each of the processes briefly.

Sexual selection: a female that is about to reproduce chooses n males from the population at random, and selects the one with the fewest deleterious mutations.

Reproduction: in sexuals, each female selects a male (described earlier) and produces 10 offspring individuals (by recombining its selection loci with the male's selection loci 10 times randomly). All loci are completely unlinked. The sex of each offspring individual is determined by chance. Asexual individuals reproduce by generating 10 clones. Since males do not actively reproduce themselves, but are only chosen as mates by females, the per capita growth rate of sexuals is on an average half of that of asexuals.

Mutation: the selection loci of each offspring accumulate on average U new deleterious mutations per generation.

Natural selection: after reproduction, the parent generation dies, and the offspring generation is subjected to natural selection in order to maintain a constant population size of N. The individuals are selected with a probability that is proportional to their fitness. Fitness is calculated as $(1-s)^i$, where i denotes the number of mutations in the mutation loci, simulating a multiplicative fitness function (i.e. no epistasis), and s denotes the selection coefficient (i.e. the relative fitness loss per deleterious mutation).

A simulation is run until a reproduction mode, sexual or asexual, has been fixed in the population. If the population is entirely sexual before 10 time-steps after the introduction of the

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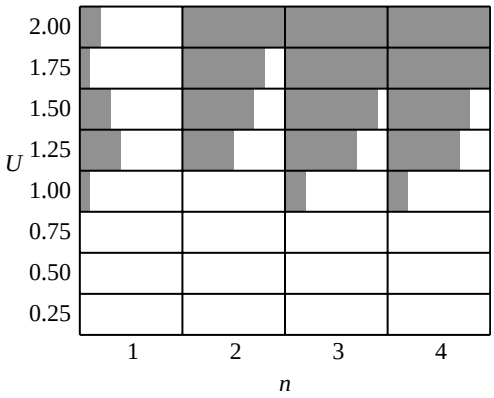

Figure 1. The maintenance of sexual reproduction in a population with or without sexual selection. The variable n denotes the number of males that a female scans before choosing the best ($n=1$ means no sexual selection), and U is the genomic mutation rate. Each combination of n and U was simulated 10 times, and the width of the grey bar is proportional to the maintenance of sex (i.e. full grey bar, sexuals win in 10 out of 10 simulation runs; no bar, sexuals never win).

asexual mutant (due to the random loss of the asexuals), the simulation is restarted and not included in the final census. All simulations were run with $N=2500$.

3. RESULTS AND DISCUSSION

The main result of this paper is that an asexual mutant can easily replace a sexual population despite strong sexual selection, unless genomic mutation rates are high (figure 1). With genomic mutation rates higher than 1, the spread of an asexual clone can be prevented by strong sexual selection in the majority of cases.

The model presented here simulates a very basic mechanism of sexual selection (a female scans n males and chooses the one with the fewest mutations), which is certainly too simple (Charlesworth & Charlesworth 1981; Seger 1985), but the relevant point in this context is to simulate strong sexual selection. In order to compare it to the previously reported analytical results, I measured the average fitness of the sexual population in the course of 500 generations, without the introduction of an asexual mutant. According to the previous analytical results (eqn 2 in Agrawal 2001), with $U=1$ and $s=0.02$, the twofold cost is fully paid if the fitness loss per deleterious mutation in males is 2.084 times higher than in females. I simulated such a scenario (i.e. $s=0.02$ in females and $s=0.0417$ in males) with the model presented here, and as can be seen in figure 2, already $n=2$ leads to massively stronger selection. Yet, sex cannot be maintained against the invasion of an asexual mutant (figure 1), which on average takes only 25–30 generations.

The population size used in the simulations is not very large ($N=2500$). Increasing the population size would increase the time of fixation of an asexual mutant. However, since the time of fixation scales approximately with log N (Salathé *et al.* 2006), an increase in population size is not expected to have a decisive effect on the time of fixation. Another effect of increasing population size is that the variation of mutational load increases, and the asexual mutant is more likely to derive from a sexual individual with a high mutational load. Such an asexual mutant would not enjoy the entire twofold fitness advantage in competition with sexual individuals, and the probability of a successful asexual invasion might decrease. Results from the simulations with smaller and larger population sizes did not quantitatively differ from the results obtained with $N=2500$, but they might change for population sizes that are orders of magnitude larger.

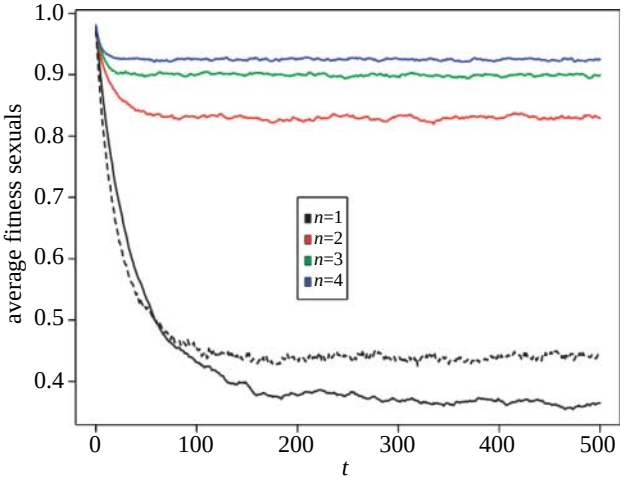

Figure 2. The effect of sexual selection on the average fitness of a panmictic sexual population ($U=1.0$). The variable n denotes the number of males that a female scans before choosing the best. For the solid lines, $s=0.02$ in both sexes. For the dashed line, $s=0.02$ in females and $s=0.0417$ in males.

Sexual selection is an important and ubiquitous force in sexual organisms, and the existing data indicate that males who are more successful in finding mates have genomes of higher quality (Andersson 1994; Kokko *et al.* 2003; Tomkins *et al.* 2004 and references therein). Hence, the idea that sexual selection influences the cost of sex is appealing. However, the real challenge in explaining the maintenance of sex despite its twofold cost is to find an advantage which pays quickly enough before parthenogenesis has replaced sexual reproduction in the population. As genomic mutation rates are mostly lower than 1, at least according to the data available at the moment (Keightley & Eyre-Walker 2000; Gong *et al.* 2005), sexual selection is unlikely to play a key role in maintaining sexual reproduction. More research is needed to address its role on the problem of sex, specifically when it acts together with a combination of mutation accumulation and other forces, e.g. parasites (Howard & Lively 1994) or limited dispersal (Peck *et al.* 1999; Salathé *et al.* 2006).

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