On Sex, Mate Selection and the Red Queen

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Abstract

The widespread occurrence of sexual reproduction despite the twofold disadvantage of producing males, is still an unsolved mystery in evolutionary biology. One explanatory theory, called the 'Red Queen' hypothesis, states that sex is an adaptation to escape from parasites. A more recent hypothesis, the mate selection hypothesis, assumes that non-random mating, possible only with sex, accelerates the evolution of beneficial traits. This paper tests these two hypotheses, using an agent-based or 'microanalytic' evolutionary algorithm where host-parasite interaction is simulated adhering to biological reality. While previous simpler models testing the 'Red Queen' hypothesis considered mainly haploid hosts, stable population density, random mating and simplified expressions of fitness, our more realistic model allows diploidy, mate selection and variable population densities. Results suggest that the two hypotheses tested seem to explain partially but not exhaustively the adaptive value of sex. However, mate selection seems to be crucial in explaining the adaptive value of sex.

Introduction

Sex remains an enigma within a mystery as there is still no widely accepted consensus for the existence and maintenance of sex (Maynard Smith, 1971, Judson and Normak 1996). The central question stated by John Maynard Smith is as follows: "what selective forces maintain sexual reproduction and genetic recombination in nature?" (Maynard Smith, 1978). If males provide little or no aid to offspring, a high (up to twofold) extra average fitness has to emerge as a property of sexual parentage if sex is to be stable. Maynard Smith explains the twofold disadvantage of producing males in the following terms:

> "Suppose that, in a sexual species, with equal numbers of males and females, a mutation occurs causing females to produce only parthenogenetic females like themselves. The number of eggs laid by a female, *k*, will not normally depend on whether she is parthenogenetic or not, but only on how much food she can accumulate over and above that needed to maintain herself. Similarly, the probability, *S*, that an egg will survive to breed will not normally depend on whether it is parthenogenetic. With these assumptions the following changes occur in one generation:

	Adults	Eggs		Adults in next generation		
Parthenogenetic	EE*n*	→	*kn*	→	*Skn*	
Sexual	EE	*N*	→	½*kN*	→	½*SkN*
	ΓΓ	*N*	→	½*kN*	→	½*SkN*

> Hence, in one generation, the proportion of parthenogenetic females increases from $n/(2N+n)$ to $n/(N+n)$; when *n* is small, this is a doubling in each generation."

Thus, an obligate parthenogenetic (asexual) female would produce twice as many daughters - and four times as many granddaughters - as the average sexual female.

Elucidating the nature of the suspected advantage of sex is one of the major challenges of evolutionary biology. Several alternative models of explanation have been presented (Jusdon and Normak, 1996). However, two broad classes seem to predominate (Hurst & Peck, 1996). First we have the ecological genetic models which postulate that sex is adaptive in variable environments because it enables the rapid spread and creation of advantageous traits. Second there are the mutation-accumulation models, which suggest that sex is adaptive because it performs the efficient removal of deleterious genes.

Additionally, a more recent model, the mate selection model, has been suggested (Jaffe, 1996) which claims that sex allows for mate selection, which in turn may restrict future variability in relevant traits, orienting the evolutionary process towards the fixation of these beneficial traits.

Enclosed within the first category, are three major hypotheses. Two of which emphasize the importance of variation in the physical environment. The third hypothesis, called the Red Queen hypothesis (Hamilton, 1980 for example), emphasizes the importance of frequency-dependent selection resulting from interspecific interactions, such as those between hosts and their parasites. The Red Queen hypothesis states that sex is an adaptation to escape from parasites. Under this hypothesis, obligate asexuality is believed not to be viable because high rate coevolving parasites efficiently adapt their strategies for infiltrating host defenses. As asexuals often stay genetically the same over several

generations, unless a mutation occurs, an obligate asexual lineage would accumulate coadapted harmful parasites. Previous computer models have been proposed to test the Red Queen hypothesis. (e.g. Bell & Maynard Smith, 1987; Hamilton et al., 1990) These models used simplified analytical methods or game theoretical versions of host-parasite interaction, with fixed population sizes and fixed patterns of parasite infection. Most of these models considered only haploid organisms, random mating and a simplified expression of fitness.

One of the most important criticisms concerning the use of models in biology, is the fact that biological and ecological systems are too complex to characterise analytically (Levin et. al. 1997). Analytical models do not allow for the simultaneous analysis of various dynamic processes such as natural selection and sexual selection. This criticism is hard to refute given the large and currently increasing evidence of the emergence of unexpected properties from complex system simulations (e.g. Cliff and Miller 1994; Jefferson et al. 1991; Kauffman and Johnson 1991; Ray 1991). In this respect, evolutionary computer simulations are ideal tools for studying co-evolution. They allow modelling of more complex and realistic genotypes, phenotypes and interactions than population-genetic or evolutionary game theory models. Moreover, they allow researchers to make detailed measurements during and after co-evolution, revealing much more information than conventional methods. Within the Artificial Life community, the use of computer simulation methods has been shown to be important in understanding the dynamics of co-evolution (Cliff and Miller 1995; Hillis 1991; Kauffman and Johnson 1991). Computer simulation has also been shown to be useful in understanding the reciprocal interactions within species between mate preferences and sexually selected traits (Jaffe 1996; Miller and Todd 1993). Additionally, from the point of view of epidemiology, a simulation model allows the monitoring of single hosts or parasites, even when they are inside hosts.

The aim of this work is to create an 'agent-based' simulation model of host- parasite interaction as close as possible to biological reality. Then to study the performance of different host reproductive strategies to evolve defenses against multiple parasites under the 'Red Queen' effect. Finally, compare the results with those of simpler models, in order to assess if the results previously reported are dependent on particular simulation assumptions and/or simplifications.

Methods

The model aimed at reproducing the features of Hamilton´s (1990) model as close as possible, but including variable population sizes, diploidy, mate selection, and more realistic patterns of infection by parasites and density dependent natural selection. The computer model simulated the interaction of mating, reproduction, selection, and parasite infection on randomly generated virtual populations of parasites and hosts. The two separated populations competed and co-evolved against each other. Parasites were modeled based on the life cycle of pathogenic nematodes, having life stages inside and outside the host. Outside hosts they lived as free-living larvae, inside hosts they had a three-stage life: egg, larva and adult. They reproduced only as adults, and pre-adult larva infected hosts if they randomly encountered a host and if the relation between the virulence of the parasite and the resistance of the host was adequate. If the infection was successful, the parasite penetrated into the host, fed on it and reproduced asexually. The progeny continued to feed and reproduce in the host as long as there remains food to be eaten. Each parasite consumed a fixed quantity of the hosts biomass at each simulation step. When this biomass was exhausted, the host was eliminated and the parasite larvae dispersed, increasing the external larval parasite population. In addition, some parasite larvae dispersed from the hosts at a fixed rate during the host’s lifetime.

Hosts could be sexual or asexual (all female and parthenogenetic). Sexual hosts were hermaphroditic, and when they reach the reproductive age they choose a mate from among all members of the population. Thus, in the simulations, asexuals and sexuals had equal fertility, simplifying the analysis, as was done by Hamilton et al. (1990) . Asexual diploid organisms were simulated so that there was crossover between the two allele copies in each loci (i.e. they were monosexual diploids). Asexual haploids had no crossover.

Simulations started normally with 500 parasites and 100 hosts, then populations were free to grow, restricted by the parasite-host interaction and by a density-dependent selection mechanism. The organism's genotype consisted of several loci, each with one (if haploid) or two (if diploid) alleles. In hosts, one locus was used to model the reproductive strategy (sexual, asexual), another to model the mate selection criteria (random, similar, dissimilar) and the rest to model resistance. In parasites, one locus accounted for parasite type and another for virulence. Randomly selected genes coding for these traits mutated, changing their allelic values at random within a predetermined range for each gene. Mutations occurred according to a fixed mutation rate. All other traits, i.e., life span, reproductive age, clutch size and mutation rate, were fixed and equal for all organisms, although different for hosts and parasites. The parameter values used for the simulations reported are:

Parameter	**Parasites**	**Host**
Life span	5	6
Clutch size	4	2
Reproductive age	3	3
Biomass	4	100
Mutation rate	0.04; 0.0016	0.04; 0.0016

Life span of parasites refers to the maximum number of time steps they may live as larvae outside a host. Inside hosts they always pass through a three-stage life (egg, larva, and adult) and reproduce as adults. Free living larvae become adults one time step after infecting a host. The clutch size and the amount of food available inside host control the number of offspring produced by a parasite.

Mating and Reproduction of Hosts. Hosts were either sexual hermaphrodites or asexual parthenogenetic females. Sexual hosts selected their mates following one of the following criteria; random mating, assortative mating (favoring similar organisms) and dissortative mating (favoring dissimilar organisms). Additionally, two different genotypes; haploid and diploid were modeled. Hosts reproduced for the first time when they reached their reproductive age, and then continue to reproduce at each simulation step . When a reproductive event occurred, the number of descendants produced was determined by their clutch size. For asexual reproduction, the genes of the offspring were identical to those of their single parent, unless changed by mutations. In the case of sexual reproduction, hosts choose a mate according to their mate selection criterion. For random mating, a mate was chosen at random from the host population. In the case of assortative mating, a fixed sized set of randomly selected potential mates was screened, and the genotypically most similar individual was chosen for mating. Dissortative mating was similar to assortative mating, but the preference was for mates with the most dissimilar alleles. Mated individuals transmitted their genes to the offspring according to the rules of bisexual diploid reproduction (i.e. meiosis), so that they received a mix of genes from both parents. The operator used for recombination is similar to the *uniform crossover* (Syswerda, 1989) employed in conventional Genetic Algorithms (Goldberg, 1989).

Infection. The infection model used was inspired by Hamilton et. al (1990), however, no artificial fitness scores were used. The Red Queen effect was simulated in the sense that hosts could not evolve an optimal genotype that conferred them resistance to parasites. Host resistance against parasites was simulated as multiple loci, each possibly resistant to one parasite type. When a random encounter between a host and a parasite was simulated, the allelic value of the resistance locus of a host, and the virulence allele of parasite type had to be the same in order for infection to occur. As in Hamilton's model, two alleles (0 and 1) were considered for each host resistance locus and parasite virulence locus. For diploid organisms the convention was that the first allele of the double chain was always expressed, although in the crossover process, there was a shuffle of the alleles of both chains. In this model of virulence-resistance, we had a 'Red Queen Effect' arising from the co-evolutionary arms race. In this co-evolution of hosts and parasites, the host's resistance evolved against the parasite virulence, which also evolved, so that each lineage's fitness landscape changed perpetually. Under this co-evolution scenario, adaptive advantage is continually undermined.

Selection. The model did not assume any simplified expression of fitness. Parasite survival depended only on their fixed life span outside hosts and the rate of reproduction (clutch size) inside hosts. Given that parasites reproduced only inside hosts, their ability to reproduce and survive was related to their ability to invade the hosts, which in turn depended on the parasites type and virulence alleles in relation to the respective host's resistance allele. Hosts survival depended on their life span and clutch size, with parasites imposing a strong selective pressure upon them. When born, hosts had a pre-determined quantity of biomass. Each individual parasite, once inside a host, consumes a fixed quantity of this biomass at each simulation step. When the first parasite invaded a host, there was a period of latency of one time step, after which the host was "killed", that is, it was unable to reproduce. Parasites inside a "dead" host were able to live on and reproduce, consuming the dead body until exhausting the available biomass. At a fixed rate of 30 %, newborn parasites dispersed from the hosts at each simulation step. When the biomass of the host was exhausted, the host was eliminated from the simulated population and the remaining internal parasites larvae were liberated as free living larvae. In addition organisms (parasites and hosts) were randomly excluded from their populations each simulation step, with a probability which increases with population density.

Results

Several thousand simulations were performed, and thus, only the most relevant results are discussed. Simulations where asexual and sexual hosts coexisted and where the gene coding for reproductive strategy was subject to mutation and recombination, showed that the population rapidly eliminates the allele for sex. The allele for asexual reproduction almost always got fixed in the population. That is, sex proved to be evolutionary unstable in our simulations. Only for very few and extreme parameter settings could alleles coding for sex invade a population of asexual organisms.

Another set of experiments simulated co-evolution on host populations in which all individuals had the same reproductive strategy, and no mutation in the reproductive strategy gene was allowed. The four reproductive strategies modeled (asexual, sexual with random mating, sexual with assortative mating and sexual with dissortative mating) were tested considering distinct number of loci for host resistance. Also simulations with haploid and diploid genotypes were run. Each simulation lasted 150 time steps, which corresponds to approximately 50 host generations. In order to cope with the considerable stochastic noise of our model, 200 replicas for each experiment were computed. The fitness measure used was the percentage of host population which survived after a given number of simulation steps (% survival = survival rate). Simulation results obtained for these experiments are as follows.

Fig. 1, shows the % of survival for the same 200 simulations after 50, 100 and 150 time steps. Results show that the simulations were robust in time, as the same qualitative differences between the strategies were observed, although the relative differences among the various strategies increases with time. Thus, for future comparisons, we present the results of simulations after 150 time steps.

In all the simulations (Fig. 1, to 3) we observe that sexual reproduction with dissortative mating produced the highest survival rates. This difference was large and consistent. In almost all cases, an increase in the number of parasite types produces a reduction in the survival rate for all reproductive strategies. Fig. 2 shows that the mutation rate does not critically affect the relative performance of the distinct strategies. However, the survival rates in all the strategies diminished considerably with the reduction of the mutation rate. Fig. 3 shows that when diploid hosts are simulated, curiously, sexual reproduction with random mating outperforms asexual reproduction, but even here, the criterion of choosing dissimilar mates proved to be the most successful mate selection criterion.

Discussion

The simulation model explored adaptation in populations of variable densities, using more elaborate selection mechanisms than those used by Hamilton et al (1990). Under these more realistic conditions, we could not replicate the findings from the model by Hamilton et al. (1990), which found that sexual reproduction with random mating in haploid host, accelerates the evolution of defensive alleles against multiple parasite species. Another discrepancy with the model by Hamilton et al (1990) is that the success of sex against asexuals increases with the number of loci involved in defense against parasites ; whereas our simulations did not show this tendency, although we worked in the same range of number of parasite types. Also, low mutation rates increase the success of sex in Hamiltons model but not in ours, although we tested approximately the same range of mutation rates. We suggest that these contradictory results are due to some critical assumptions in Hamilton's model. In particular, the most critical assumption may be the 'soft selection' by means of artificial truncation of the host population in which infected hosts could survive, whereas in our model infections were always lethal.

When studying diploid hosts, sexual population with random mating and with dissortative mating largely outperformed asexual ones, suggesting that the adaptive value of sex is higher in diploid organisms. Also, only among diploids did random mating sexual organisms outperform asexual ones. That is, the effect of diploidy in simulation models could be significant and thus, results obtained modeling haploid organisms should not be extended to diploid organisms. This may support the importance of ploidy cycles or meiosis in the evolutionary dynamics of sex (Kondrashov, 1994; Jaffe 1996)

Our results are consistent with simulations of organisms with absolute optimal genetic configurations (Jaffé, 1996, Jaffé et al. 1997), confirming the importance of mate selection as an evolutionary catalyst, helping in maintaining sex.

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References

Bell, G., and Maynard Smith J. (1987) "Short-term selection for recombination among mutually antagonistic species". *Nature*, 328, pp. 66-68.

Cliff, D. and Miller, G. F. (1994). "Protean Behavior in Dynamics Games : Arguments for the co-evolution of pursuit-evasion tactics". In D. Cliff, P. Husbands, J. A. Meyer and S. Wilson, editors, *Proc. Third International Conf. Simulation Adaptive Behavior (SAB 94)*, M.I.T. Press Bradford Books.

Cliff, D. and Miller, G.F. (1995) "Tracking the Red Queen: Measurements of adaptive progress in co-evolutionary simulations". In F. Moran, A. Moreno, J. J. Merelo and P. Cachon (editors) *Advances in Artificial Life: Proceedings of the Third European Conference on Artificial Life (ECAL95). Lecture Notes in Artificial Intelligence 929*, Springer Verlag, pp.200-218.

Ewald, P.W. (1995). "The evolution of virulence: a unifying link between parasitology and ecology". *Journal of Parasitology*, 81(5), pp. 659-669.

Goldberg, David E (1989). *Genetic Algorithms in Search, Optimization and Machine Learning*. Addison-Wesley Publishing Company, Inc.

Hamilton, W. (1980). D. "Sex versus non-sex versus parasite". *Oikos* 35 :282-290.

Hamilton, W.D., Axelrod, R. and Tanese, R. (1990). "Sexual reproduction as an adaptation to resist parasites : A review." *Proc. Nat'l Acad. Sciences (USA)*, 87(9) : 3566-3573.

Hillis, W.D. (1991). "Co-evolving parasites improve simulated evolution as an optimization procedure."

Artificial Life II, SFI Studies in the Sciences of Complexity, Ed. C. Langton, C. Tylor, J. D. Farmer,

& S. Rasmussen, Addison-Wesley Publishing Company.

Howard, R. S. and Lively, C. M. (1994). "Parasitism, mutation accumulation and the maintenance of sex". *Nature*, 367, pp. 554-556.

Hurst, L.D. and Peck, J.R. (1996). "Recent advances in understanding of the evolution and maintenance of sex". *TREE*, vol 11, no. 2, pp. 46-52

Jaffe, K. (1996). "The dynamics of the evolution of sex, why the sexes are, in fact, always two ?" *Interciencia*, 21 : 259 :267.

Jaffe, K. Issa, S., Daniels, E, and Haile, D. (1997) Dynamics of the emergence of genetic resistance to pesticides among asexual and sexual organisms. J. Theor. Biol. (in press)

Jefferson, D., Collins, R., Cooper, C., Dyer, M., Flowers, M., Kork R., Tylor, C. and Wang, A. (1991). "Evolution as a Theme in Artificial Life: The Genesys / Tracker System." *Artificial Life II, SFI Studies in the Sciences of Complexity, Ed. C. Langton*, C. Tylor, J. D. Farmer, & S. Rasmussen, Addison-Wesley Publishing Company.

Judson O.P. and Normak B.B. 1996. Ancient asexual scandals. Trends in Evolutionary Ecology (TREE) 11 : 41-46.

Kauffman, S.A., and S. Johnson. (1991). "Co-Evolution to the Edge of Chaos : Coupled Fitness Landscapes, Poised States, and Co-evolutionary Avalanches". *Artificial Life II, SFI Studies in the Sciences of Complexity*, Ed. C. Langton, C. Tylor, J. D. Farmer, & S. Rasmussen, Addison-Wesley Publishing Company.

Kondrashov A. (1994) The asexual ploidy cycle and the origin of sex. Nature 370: 213-216.

Levin S.A., Grenfell B., Hastings A. and Perleson A.S. (1997). “Mathematical and Computational Challenges in Population Biology and Ecosystems Science”. *Science*, 275, pp.334-343.

Maynard Smith, J. (1971). “The origin and maintenance of sex”. In G. C. Williams (ed.), *Group Selection.* Aldine Atherton, Chicago, USA.

Maynard Smith, J. (1978). *The Evolution of Sex*. Cambridge University Press, Cambridge, UK.

Miller, G.F., and Todd, P.M. (1994). “Evolutionary wanderlust: Sexual selection with directional mates preferences”. In D. Cliff, P. Husbands, J. A. Meyer and S. Wilson, editros, *Proc. Third International Conf. Simulation Adaptive Behavior (SAB 94)*, M.I.T. Press Bradford Books, 1994

Muller, H.J. (1964). “The relation of recombination to mutational advance”. Mutat. Res. , 1, pp. 2-9.

Ray, T. (1992). “An Approach to the Synthesis of Life.” *Artificial Life II, SFI Studies in the Sciences of Complexity*, Ed. C. Langton, C. Tylor, J. D. Farmer, & S. Rasmussen, Addison-Wesley Publishing Company.

Syswerda, G. (1989). “Uniform crossover in genetic algorithms.” In J. David Shaffer (ed.) ,*Proceedings of the Third International Conference on Genetic Algorithms*. Morgan Kaufman Publishers.

Fig. 1: Average rate of survival for haploid host populations calculated from the results of the same 200 simulations after 50, 100 and 150 time steps. Results are for simulations with 2, 5, 10 and 15 loci for resistance (parasite types). Mutation rate for both parasites and host was 0.04 mutations per loci. Reproductive strategies tested were: Asex: parthenogenetic; SexRnd: sexual with random mating; SexSim: sexual with assortative mating, SexDif: sexual with dissortative mating.

Fig. 2: Average rate of haploid host population survival for 200 simulations after 150 time steps, for simulations using mutation rates 0.04 and 0.016 for both, hosts and parasites. Results are for simulations with 2, 5, 10 and 15 loci for resistance (parasite types). Else as in Fig 1.

Fig. 3: Average rate of population survival for 200 simulations after 150 time steps, for haploid and diploid hosts. Results are from simulations with 2, 5, 10 and 15 loci for resistance (parasite types). Mutation rate for all organisms was 0.04. Else as in Fig. 1.

Fig. 1

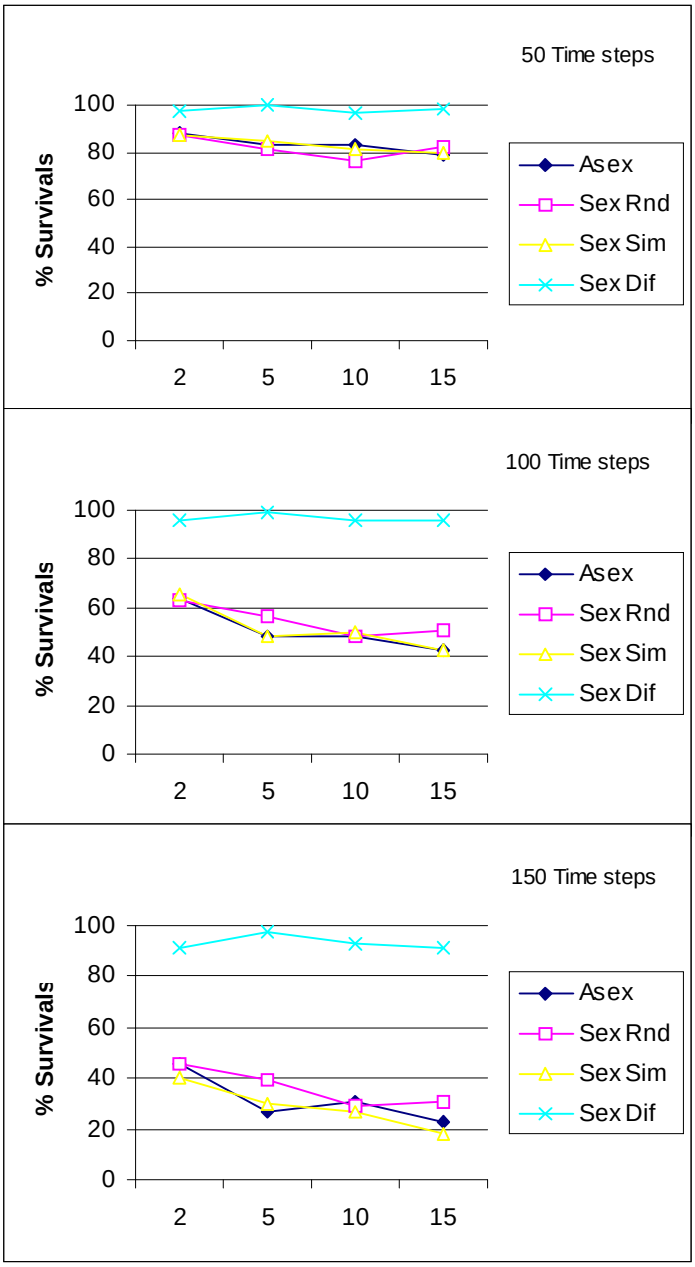
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Fig. 2

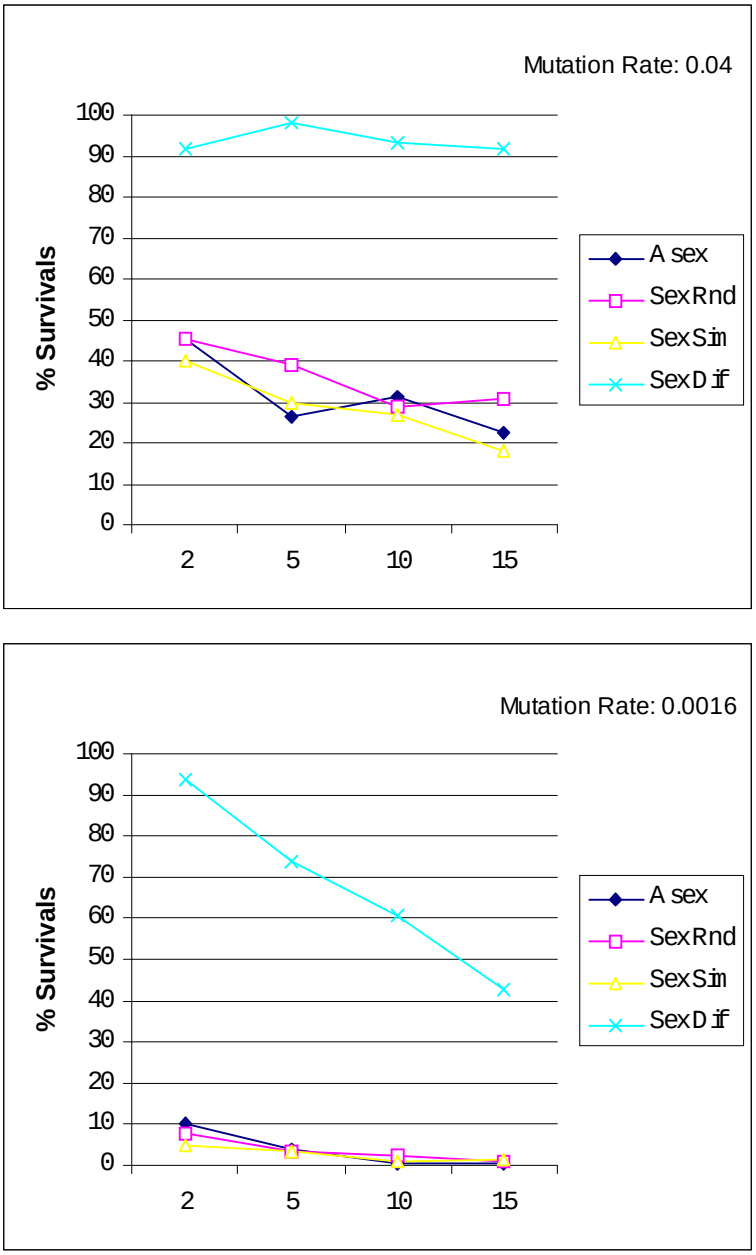
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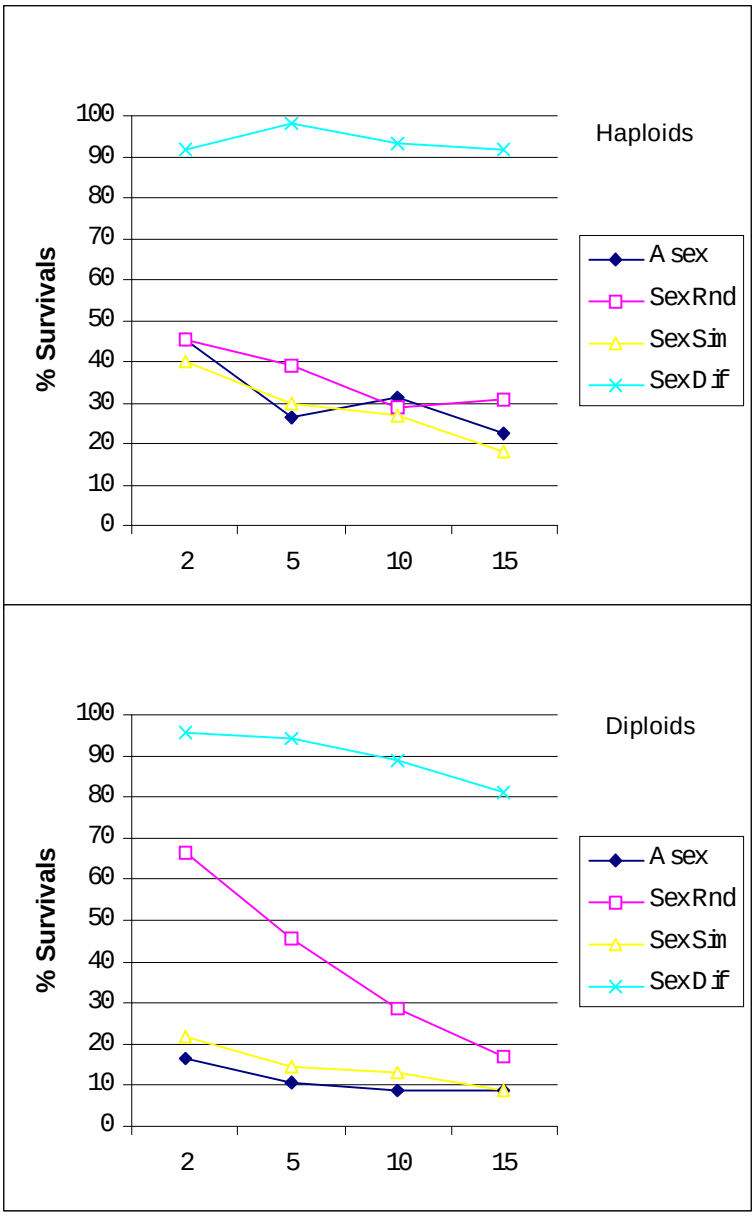
Fig. 3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