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On the adaptive value of Sex

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Using computer simulations I studied the conditions under which sex was evolutionary stable. The parameters that showed relevance to the stability of sex were: variable environments, mutation rates, ploidy, number of loci subject to evolution, mate selection strategy and reproductive systems. The simulations showed that mutants for sex and recombination are evolutionarily stable, displacing alleles for monosexuality in diploid populations mating assortatively when four conditions were fulfilled simultaneously: selection pressure was variable, mate selection was not random, ploidy was two or the reproductive strategy was haplo-diploid or hermaphroditic, and the complexity of the genome was large (more than 4 loci suffered adaptation). The results suggest that at least three phenomena, related to sex, have convergent adaptive values: Diploidy, sexual reproduction (recombination) and the segregation of sexes. 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. The divergence of the evolutionary adaptation of the sexes is a derived consequence of the emergence of sex. A corollary of these simulations is that gene mixing, achieved by sex, is advantageous if the degree of mixing is not very great, suggesting that an optimal degree of gene mixing should exist for each species.

1 Introduction

What selective forces maintain sexual reproduction and genetic recombination in nature? The answer to this question has been 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). This model has been shown to be incomplete in explaining the emergence and maintenance of sex (Ochoa and Jaffe, 1999 for example)

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. Experimental results have shown that this model can not explain the genetic dynamics of extant sexual organisms (Cutter and Payseur 2002 for example).

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, 2000). Here I want to explore this last model further.

The model *Biodynamica* used here (Jaffe 1996, 1998, 1999, 2000, 2001, 2004), has been shown to have heuristic properties in explaining or predicting experimental data. It explains many aspects of the emergence of genetic resistance to antibiotics and pesticides (Jaffe et al 1997), it predicted divergent behavior of production of males in facultative sexual nematodes (Rincones et al. 2001), the importance of economic aspects in the evolution of social behavior (Silva and Jaffe 2002), it predicted the existence of homophily among humans, such as the physical similarity between faces of married couples (Alvarez and Jaffe 2004) and the

similarities dog pets and their human owners (Payne and Jaffe 2005), and it predicted sperm selection by sperm-plasma in humans (Jaffe et al. 2006). Thus it seemed promising in uncovering some remaining mysteries of sex.

2 Methods

In this multi-agent, adaptive model, each individual was simulated as an autonomous agent who 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 competitions between agents with alleles coding for different strategies. 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:

2.1 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.

2.2 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.

2.3 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).

2.4 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.

2.5 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:

$$\text{survival of individual } i \text{ at time step } t = \begin{cases} 0 & \text{if } r_1 * N_t \geq \text{ops} * r_2 \\ 1 & \text{if } r_1 * N_t < \text{ops} * r_2 \end{cases}$$

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.

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	Effect on phenotype for alleles
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)

3 Results

Mutant alleles coding for sexuality displaced the corresponding alleles coding for monosexual strategies when simulating hermaphrodites or diploid bisexual organisms which mated assortatively and produced more females than males. Mutant alleles coding for sexual diploid hermaphrodites with assortative mating displaced alleles coding for monosexual diploid alleles from the populations. Bisexuality seemed unable to displace monosexuality in haploid populations (see Figure 1).

When making tournaments, in which two alleles coding for different sexual phenotypes had to compete with each other in invading a population of agents, we find that the most successive sexual strategies in displacing asexuality in the evolutionary game are a combination of bisexuality with assortative mating, with a large variance in males reproductive success, and with sperm selection acting on spermatozoa prior to the production of a new offspring (see Figure 2).

More results can be obtained independently by downloading the program Biodynamica at <http://atta.labb.usb.ve/Klaus/klaus.htm>

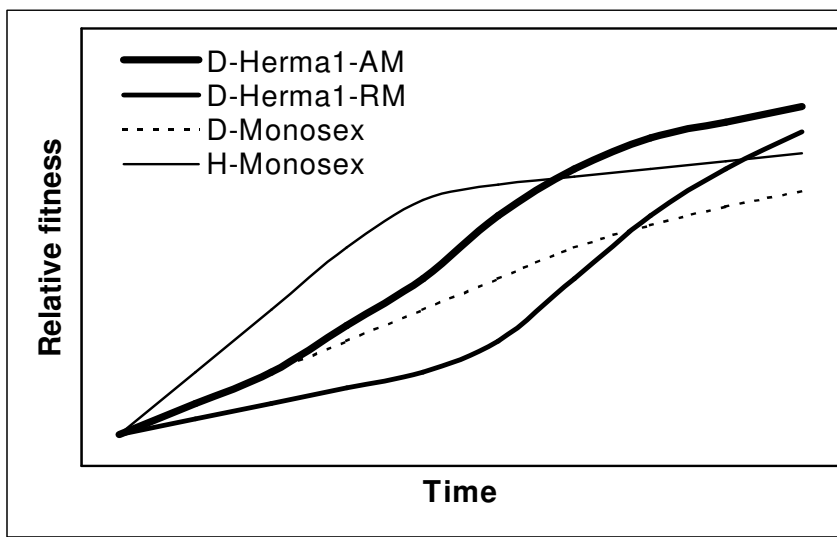


Figure 1: Approximate time course of mean fitness of organisms in an evolving population using different reproductive strategies.

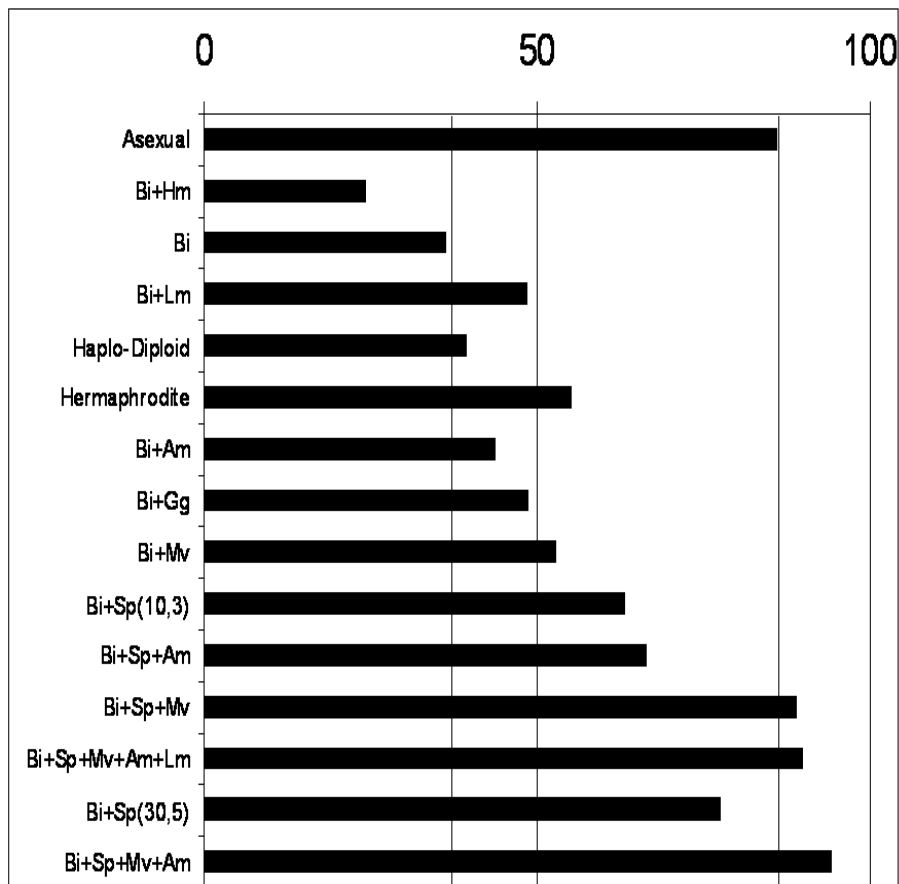


Figure 2: Percent of alleles of the given reproductive strategy present in a population of 800 agents in relation to alleles coding for monosexual diploids (thelytoky), when alleles coding for both strategies are competing between them in a single population. Bars smaller than 50% indicate strategies less efficient than monosexual diploids; bars larger than 50% indicate

strategies that are more successful than the monosexual diploids. Each bar is the average of 2000 simulations using the agent based computer simulation Biodynamica (Jaffe, 1995). Tournaments were run, starting with populations having a 50/50 proportion of alleles for monosexuality and for the ones indicated in the y axis. Abbreviations indicate; Bi: bisexual, Haplo-Diploid: females are diploid and males haploid, Am: assortative mating, Gg: females select males for good genes, Mv: reproductive variance in males was twice the variance for females, Lm: low mutation rate was 0.008 mutations per loci, Hm: high mutation rate was 0.2 mutations per loci. Else 0.04 random mutations per loci were simulated. Sp(x,y) indicate simulation of x spermatozoa per male expressing y genes.

4 Discussion

I postulated earlier (Jaffe 2000) 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). Most studies on the emergence and maintenance of sex have focused on models using random mating, failing to find this phenomenon.

Yet, other simulation models have not obtained these results. The careful comparative analysis of the various models used allows us to establish a list of critical features in models that may explain these differences:

- Modeling diploid organisms versus haploids
- Modeling true random mating versus truncated fitness determining reproduction
- Modeling agents with many genes versus agents with up to 3 genes
- Modeling various selection criteria versus assessing a fitness value

Thus, I suggest that the more realistic the agent based simulations become, the closer we get to understand the mysteries of biological evolution.

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