SIMULATION STUDY TO EXPLAIN
SEXUAL REPRODUCTION’S PREVALENCE

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Abstract: The prevalence of sexual reproduction has long been an outstanding problem of evolutionary biology because of its significant costs as opposed to asexual reproduction. Different explanations have been offered to explain the prevalence of sexual reproduction; most of these explanations focus on the benefits of sexual reproduction’s ability to shuffle and recombine genes. In this paper, we use computer simulation to show that sexual reproduction can also lead to another effect: genetic homogeneity, which leads to the maintenance of adaptational advantages. Sexual reproduction maintains adaptational advantages of organisms, and in stable conditions with strong selective pressures, the maintenance of desired adaptational advantages warrants the dominance of sexual reproduction. Various computer simulations are performed to show that (1) sexual reproduction promotes genetic homogeneity and (2) sexual reproduction is beneficial in conditions with stable and strong selective pressures. Sexual reproduction leads to species stabilization, which maintains desired adaptational advantages, thereby warranting the dominance of sexual reproduction in stable conditions with strong selective pressures. We also provide biological evidences to support our hypothesis by reevaluating available literature. We hope that this idea and our computer simulations will lead to a fresh perspective from which to approach this important problem in evolutionary biology.

Keyword - Computer simulation; evolution; sexual reproduction; genetic homogeneity; selective pressure.

1. INTRODUCTION

Why sexual reproduction? This question is of interest not only to biologists, but also to the general public as a whole (Bell 1982, Colegrave 2002, Hurstand Peck 1996, Misevic et al. 2006). The puzzle of sex and its evolution can be separated into two categories: (1) the origin and (2) the continued maintenance of sexual reproduction. The question of how sex originated will most likely be proven difficult to answer definitively. However, the fact of the matter is that once sex developed it was adopted as the predominant reproductive strategy of most living organisms. The prevalence of sexual reproduction suggests that there is a major benefit provided by this mode of reproduction. As such, the focus of this paper will be to use computer simulation to investigate one possible benefit that reinforces the maintenance of sexual reproduction.

In many ways, asexual reproduction is a better evolutionary strategy: only one parent is required, and all of the parent’s genes are passed on to its progeny (Agrawal 2006, Bachtrog 2006, Chasnov 2000, de Visser et al. 2007, Dolgin and Otto 2003, Ladle 1992, Otto 2003, Otto and Lenormand 2002). In a sexual population, the males are unable to produce offspring of their own and females only transfer half their genes to offspring, hence the twofold cost of sex. Sexual reproduction must also go through obstacles that do not hinder asexual reproduction. Sexually reproducing organisms must spend a great deal of time and energy to find and attract mates. The peacock is a good example. The male must grow a large and intricate tail to attract mates; not only is producing the tail energy consuming, the peacock must also carry around its tail at all times, leaving it vulnerable to predators. Furthermore, copulation in sexually reproducing organisms can leave both organisms vulnerable to predation. Despite these major drawbacks to sexual reproduction, it is still a very prevalent form of reproduction in most living organisms.

Many researchers have put forth numerous explanations for why sex is so prevalent. Current hypotheses to explain the maintenance of sex typically focus on the benefits of the inherent ability of sexual reproduction to recombine and
shuffle genetic information (Agrawal 2001, Agrawal and Chasnov 2001, Arkhipova and Meselson 2004, Barton and Charlesworth 1998, Charlesworth 1993, Elena and Lenski 1997, Feldman et al. 1980, Kondrashov 1988, Peck and Waxman 2000). These benefits are undoubtedly significant. However, we believe that aside from the ability to recombine DNA, sexual reproduction has another natural consequence: genetic homogeneity (Lin 2005) and maintenance of adaptational advantages. While the recombination of genes in sexual reproduction indeed provides significant genetic variation at one level, we believe sexual reproduction maintains genetic homogeneity at another more fundamental level. In this paper, we provide computer simulations and biological evidence to support the following parts of our hypothesis: (1) Sexual reproduction is a species stabilization mechanism that naturally maintains genetic homogeneity and species identity. (2) Asexual reproduction, which does not have this inherent species stabilization mechanism, actually leads to genetic diversity and no definitive species identity. (3) Sexual reproduction is beneficial because the maintenance of species identity maintains desired adaptational advantages, which is important when selective pressures are strong and stable.

Part (2) of our hypothesis is predicted by Muller’s ratchet (Muller 1964) and hence not new. To the best of our knowledge, Part (1) of our hypothesis has not been proposed before and cannot be derived from existing results on the sexual reproduction’s ability to recombine and shuffle genetic information. We also think that Part (3) of our hypothesis is the key to the benefit of sexual reproduction.

We perform computer simulations that simulate sexual and asexual reproduction. Simulation results support our hypothesis. We also believe that there is adequate literature to support our hypothesis.

2. BIOLOGICAL EVIDENCE

One of sexual reproduction’s most evident benefits is its ability to allow the recombination of different genes. However, this benefit can only occur when a successful mating takes place. Consequently, intrinsic to the sexual process are numerous reproductive barriers that limit the extent of dissimilarity that can be exchanged between the genetic information of two individuals. These reproductive barriers have been well documented. For example, one prezygotic reproductive isolating mechanism is ecological isolation where even though two different species dwell in the same environment, due to differences in resource utilization and other evolved adaptations, these species never come in contact with one another. As a result, they never mate with each other (Raven et al. 2005). Behavior isolation is another prezygotic isolating mechanism and occurs when different species have different courtship rituals, mating calls, and etc. that limit interspecies mating. These species may even be able to successfully mate in artificial situations such as a zoo, but the in the wild, due to different behaviors, these species will never normally mate (Raven et al. 2005). Other prezygotic isolating mechanisms include temporal and mechanical isolation. When one species is active during the day and another species is active during the night, they are temporally isolated and will rarely mate with each other even if they physically can. Species that differ greatly from one another, such as being different sizes or having incompatible reproductive organs are morphologically different and cannot mate with one another (Raven et al. 2005).

Perhaps the most obvious form of reproductive isolation is the prevention of fusion between different species’ gametes. Gametes with different chromosome numbers and different chromosome sizes cannot properly pair with each other, resulting in failed embryo development. Even if different species successfully fuse dissimilar gametes to form a viable offspring, the offspring typically have an abnormal, odd chromosome number. These offspring are usually sterile hybrids that can no longer contribute to the gene pool of either respective parental species (Raven et al. 2005).

All these reproductive isolating mechanisms are ultimately derived from an organism’s genetic coding. As a result, sexual reproduction indeed allows for genetic exchange, but on the other hand, this genetic exchange cannot be so drastic or profound, otherwise mating will never occur in the first place. Because gene exchange is limited to organisms having compatible genetic codes, we believe that sexual reproduction leads to the formation of clusters of individuals with a similar genetic makeup.

On the other hand, although asexual reproduction is believed to preserve genetic integrity from one generation to the next, the asexual genome is not static and it is prone to various changes such as mutation, horizontal gene transfer, and
chromosomal rearrangement (Alberts et al. 2004). These resulting genetic changes are copied directly to offspring. Any genome change that does not result in an asexual organism’s death will be carried into future generations. As a consequence, asexual genomes will diverge and differentiate from each other over time.

As an example, we first consider the asexual RNA viruses. RNA viruses have the highest mutation rate among any organisms (Elena et al. 2005). This high mutation rate allows RNA viruses to quickly diversify and explore new genotypic variations, often leading to the formation of ‘quasi species’ (Elena et al. 2005). This ability to quickly diversify has proven beneficial in rapidly changing environments such as the human body. Aside from viruses, there are numerous examples of genetic diversity found in other asexual organisms. It’s been observed that E. coli strains have a 55-60% conserved gene core (Konstantinidis and Tiedje 2005). Although this isn’t necessarily an accurate comparison, we briefly mention that interspecies identicalness between humans is now estimated around 99-99.5% (Levy et al. 2007).

In general, it is believed that asexual organisms have shorter evolutionary life spans and less ability to diversify than do sexual organisms. This however leads to the unique cases of several ancient asexuals. The class of bdelloids is believed to be at least 35-40 million years old. In that time, they have diversified to over 360 species and occupy nearly every freshwater habitat (Birky 2004). This diversification and long-term evolutionary survival defies current thinking about asexual organisms, and many failed attempts have been made to prove them sexual. In the attempt to confirm the long-term asexuality of these bdelloids, scientists have observed and measured the extent of allelic sequence divergence in these asexuels. For example, for each gene in the Bdelloid rotifers, the most similar copies differed 36-73% at synonymous sites, compared to a 0-2.4% difference in their sexual cousins, the monogonont rotifers, and other sexual animals (Birky 2004). Basic theory indicates that neutral sequence divergence between alleles in a protein-coding gene in an asexual may be greater than a sexual species by a factor of 2tu, where t is the number of generations since asexual reproduction was lost and u is the mutation rate per generation in the asexual linage (Birky 1996). The measuring of genetic divergence to prove the ancientness of asexual lineages seems to us to be in direct contrast to the idea of the asexual process as a means of maintaining genetic fidelity and direct proof of the genetic diversification of asexual organisms.

As evidenced from these examples, it appears that the definition of ‘species’ does not hold any real meaning for asexual organisms. The ‘boundaries’ of an asexual species definition appear to be primarily determined by the strength of natural selection, which eliminates nonviable genotypes, and the random effects of genetic drift.

In essence, this genetic diversification in asexual reproduction is what is predicted by Muller’s ratchet. Muller’s ratchet occurs when an asexual population’s mean fitness decreases due to the random loss of individuals with the least amount of deleterious mutations (Muller 1964, Kondrashov 1994). Therefore, this part of our hypothesis on asexual reproduction coincides with the existing and known results. However, the part of our hypothesis on sexual reproduction stating that sexual reproduction maintains genetic homogeneity and species identity cannot be derived from the existing results. In other words, this part of our hypothesis, although seemingly intuitive from biological evidences presented above, to the best of our knowledge, has not been formally proposed in the literature.

3. COMPUTER SIMULATIONS

To further support that sexual reproduction promotes genetic homogeneity and asexual reproduction promotes genetic diversity, we have performed computer simulations that simulate sexual and asexual reproduction. The computer simulations we designed were written with MATLAB, which is a programming language specifically designed for matrix manipulation.

The asexual simulation (Simulation 1) first generates a vector (a string of numerical values); the length of the vector and the range of numbers inserted into the vector are user defined. This vector is our representation of a genome; the individual numerical values are our representation of genes at certain loci in the digital genome. This first ‘genome’ is then replicated into more genomes (offspring) and each subsequent offspring genome generates their own offspring genomes, etc. Each genome is appended to a ‘population’ matrix, which contains all the genomes of all individuals in the current generation. In each replication process we allowed each generated genome to possibly undergo gene mutation (change in a numerical value) or chromosomal mutation (numerical values
were either added or subtracted from the genome), or both. The probability of such changes to the genome is user defined. The program is run for as many generations as the user defines, with each new generation replacing the genome values of the previous generation. Due to the exponential growth pattern in this simulation, population growth is limited to whatever value the user defines. Upon reaching this population limit, the simulation for the next generation starts. Random genomes from the previous generation are selected, and they generate offspring genomes until the next generation’s population size again reaches the population limit, this is repeated until the simulation has completed its set number of generation cycles.

Our sexual simulation (Simulation 2) is similar to the asexual simulation; it is capable of all the same mutations, etc. However, it also has a few modifications. Instead of one genome generated as our seed, we generate two seed genomes: one male, one female. These ancestral genomes can also be modified to be a certain percentage different from each other to simulate genetic diversity (we defined a 10% difference between the two genomes for most of our simulations). The male and female genomes are then aligned next to each other and a pairwise comparison between the two sequences is made. The program shifts the alignment between the two genomes to identify the orientation with the most pair-wise matches. The percentage of matches is observed and if the percentage is within the user-defined limits, offspring are generated by the combination of the two genomes (each numerical value is taken from either the male genome or the female genome with a 50% chance from either sex). The offspring is then randomly appended to the male or female population matrix. This is repeated for a random number of times to simulate the random number of children possible for each ‘couple’ (the range is defined by user). If the parent couple is not 100% identical, offspring of this couple are not necessarily identical due to the random inheritance of maternal and paternal genes.

Once all generation cycles are completed, both simulations undergo the same analysis. Our hypothesis that sexual reproduction retains genetic homogeneity means that if we were to cluster all the data into groups of similar values, sexual reproduction should generate a few, large clusters with relatively high similarity. Asexual reproduction should generate either one very large cluster with low similarity, or many small clusters.

To observe whether this is the case, the simulations take all vector values and submit them to a k-means clustering algorithm that analyzes and partitions the data into a certain number of clusters. We then use a third party multidimensional scaling program, VisRed (Dourado et al. 2007), to take our high dimensional population matrix and reduce it into three dimensions for easy visualization.

Figure 1: A typical asexual simulation. Ran for 1000 generations with genome length set between 30-50 gene loci. Each locus had 10 different gene allele possibilities. There was a 0.2% chance of a gene mutation and 0.2% chance of a genome length mutation. Each individual generated exactly 2 offspring. End population size was 2999 individuals.
Figure 2: A typical sexual simulation. Ran for 1000 generations with genome length set between 30-50 gene loci. Each locus had 10 different gene allele possibilities. There was a 0.2% chance of a gene mutation and 0.2% chance of a genome length mutation. Each couple generated 2-7 children. The original male and female seeds differed by 10% in gene content. Successful mating required a 90% genome match. Offspring that differed in length from the parents by 4% were labeled as mutants. End population size was 2942 individuals.

We ran Simulations 1 and 2 numerous times. All repeated simulations generated consistent results. A typical result of Simulation 1 is shown in Figure 1 and a typical result of Simulation 2 is shown in Figure 2. For these two simulations, we ran them for 1000 generations with a 0.2% chance of chromosomal and gene mutation. The silhouette plots in Figures 1 and 2 are a graphical representation of how well suited the vectors are to their corresponding cluster. A value of 1 is a perfect match and a value of -1 is a complete mismatch. The pictures on the right of the silhouette plots are simply spatial visualizations of the population matrices scaled down to three dimensions with the resulting distribution of clusters represented.

As we can see from these specific results (Figures 1 and 2), asexual reproduction led to the formation of 23 small clusters (typical range is 15 to 30 clusters) and sexual reproduction led to the formation of 4 large clusters (typical range is 1 to 5 clusters). Genomes in the asexual clustering also did not match as well within their designated clusters as compared to the sexual clustering (this was observed in all simulation runs). These results support our hypothesis that sexual reproduction maintains genetic homogeneity while asexual reproduction leads to genetic diversification.

4. BENEFIT OF GENETIC HOMOGENEITY AND CLUSTERING

So far we have argued that sexual reproduction leads to genetic homogeneity and clustering. That now begs the question of “What is the benefit of genetic homogeneity and clustering?” We hypothesize that when conditions are relatively stable, the strength of selective pressures is strong, and when organisms have become adapted to those selective pressures, it would be most beneficial to maintain those adaptations. As seen from our simulations, asexual reproduction results in diversification of genetic composition and sexual simulations resulted in the formation of tight clusters. These tight clusters are maintained by the sexual process and prevent the massive diversification (deleterious mutation accumulation) seen by asexual simulations.

To illustrate this benefit of genetic homogeneity and maintenance of adaptational advantages in a biological context, we take the example of a developed ecosystem that is near its “climax community.” Such systems can be considered relatively stable with high biodiversity. The high biodiversity means there is more competition for the same limited resources. In order for all these species to successfully live amongst each other, selective pressures have caused each species to
develop specific adaptations that allow it to occupy an exclusive niche (strong selective pressure) (Raven et al. 2005). It’s also a common observation that k-reproductive strategies typically dominate such systems (Raven et al. 2005). The practice of organisms in such systems to reproduce fewer, but more highly niche-adapted offspring are all adaptations due to selective pressures that allow these species to survive in this type of environment. Straying away from such adaptations, which means straying away from the species’ niche, results in competition with other species that occupy other niches. These other species are highly adapted for their niches; consequently, the ‘stray organism’ is unlikely to survive the competition. Sexual reproduction maintains adaptational advantages and minimizes the conversion of precious resources to the production of ‘stray organisms’ that are unlikely to survive.

The same reasoning applies to an opposite situation. It is a common observation that in harsh environments, many sexual organisms employ the r-reproductive strategy. In such environments, specific adaptations geared toward maximizing individual survival in the environment are not as useful. Instead, selective pressures have caused these organisms to develop adaptations that allow them to reproduce quickly and in prodigious numbers, as well as typically having low nutrient requirements, short maturation time, etc. (Raven et al. 2005). In order to maintain these adaptations, such organisms also reproduce sexually.

Some organisms (yeasts for example) are capable of switching between asexual and sexual reproduction according to environmental conditions (Ridley 1997). Yeasts reproduce sexually when under conditions of high stress (Zeyl and Bell 1997, Hoekstra 2005). Traditional thinking would conjecture that the sexual process either generates genetic diversity, which is beneficial in novel or changing environments, or that sex concentrates deleterious mutations for more effective removal by selection (Zeyl and Bell 1997). An alternative explanation has been proposed where difficult conditions induce high levels of genomic aberrations. Consequently, yeasts engage in sexual reproduction to retain and preserve the karyotype of the parents (Heng 2007). We believe the explanation that sexual reproduction retains and preserves parental karyotypes is on the right track. However, we feel the idea that sexual reproduction as the response of an organism to maintain its genomic integrity against environmentally induced genomic challenges is limited in scope. Not all organisms are unicellular; multicellular organisms won’t pass on altered genomes to its offspring unless the genomic aberrations occur at the germ line and we believe most environmental conditions are not severe enough to challenge genomic integrity of these germ cells. Instead, we believe that sexual reproduction is a species survival mechanism that fuses proven, environmentally adapted genomes from successful individuals to further maintain that successful genome. When conditions are good, selective pressures are weak, possibly weak enough that it would be better to produce asexually. However, when conditions worsen and selective pressures become very strong, it would be most beneficial to maintain a successful genome.

As another example of heterogonic organisms, we focus on the cyclically parthenogenetic monogonont rotifers. The product of sexual reproduction in monogononts is a thick-shelled resting egg capable of surviving adverse periods that occur during the typical annual cycle in temperate zone lakes (Serra et al. 2004). It’s been argued that the critical functional role of dormancy in these eggs is a strong enough selective factor to maintain sex in monogononts and a similar argument has been made for cyclically parthenogenetic aphids (Serra et al. 2004). However, we feel the production of resting eggs is a consequence of sexual reproduction, not the selective factor that maintains it. It’s already been suggested that it is possible to decouple sex and resting egg production (Serra et al. 2004).

However, researchers have reported a positive correlation between sexual reproduction and population density in monogononts. One explanation for this observation, the habitat deterioration hypothesis, conjectures that high density causes habitat deterioration so sex occurs when the population size peaks (Serra et al. 2004). We agree with the habitat deterioration hypothesis. Under high population density, each monogonont must compete for fewer resources. In this sense, selective pressures have now increased and become strong. Under our hypothesis, it is advantageous to maintain species identity when selective pressures are strong, hence the conversion to sexual reproduction.

Biologists have often noted that parthenogenetic organisms often live in marginal or transient environments while their sexual cousins occupy more suitable environments (Haag and Ebert 2004). Various hypotheses have been put forth to explain...
this puzzling geographic distribution. The reproductive assurance hypothesis asserts that asexual organisms may be better able to colonize marginal habitats because they don’t need to mate for reproduction. Another hypothesis states that asexuals do better in marginal habitats where they can avoid the many more biotic interactions (competition, parasites, predators, etc.) found in more suitable habitats (Haag and Ebert 2004). Our hypothesis fits both explanations: suitable habitats have strong and stable selective pressures (many biotic factors); therefore sexual reproduction is advantageous in such environments. Marginal habitats don’t have such strong and stable selective pressures. Consequently, sexual reproduction doesn’t have a significant benefit and asexual reproduction, with its simplicity and lower cost, becomes more dominant.

5. FURTHER COMPUTER SIMULATIONS

To further support our idea that sex promotes genetic homogeneity and maintains adaptational advantages, we have designed computer simulations that show that sexual reproduction is beneficial in stable conditions with strong selective pressures.

These simulations are identical to the previous simulations in the generation of sexual and asexual offspring, the differences lie at the end of program execution. In these simulations, we introduce a selective pressure factor (SPF) at the end of each run. The SPF determines what offspring are to be considered successful and kills off the unsuccessful offspring. In the beginning of the program when the asexual seed genome and sexual male and female seed genomes are declared, the SPF is defined as the exact same copy of the seed genome in the asexual simulation, and it is defined as the combination of the male and female seeds in the sexual simulation.

In Simulation 3, the SPF remains unchanged, what we change around is how similar the offspring must be to the SPF (e.g. offspring must be 70%, 80%, 90%, etc. similar to the SPF); this is our ‘selective pressure.’ The more similar offspring must be to the SPF, the stronger the selective pressure. The results are shown in Figure 3.

![Survival vs Strength of Selective Pressure](image_url)

**Figure 3:** Genome length mutation rate and gene mutation rate both set at 3%. 100 simulations were run. The selective pressure value is the percentage of similarity based off of hamming distance. A selective pressure of 10 on the graph corresponds to a required minimum of 90% similarity to SPF, a 20 corresponds to 80% similarity, etc. End population size of each run was about 600 individuals.
In Simulation 4, we test the survivability of sexual and asexual reproduction in unstable conditions. In this simulation, we keep the strength of the selective pressure constant. Instead, we change the values in the SPF. The more we change the SPF, the more unstable the conditions. The results are shown in Figure 4.

For computational convenience, SPF is factored only once with every simulation run of 100 generations. We ran 100 simulations for each change in SPF, which is mathematically equivalent to a simulation run of 10,000 generations with SPF factored every 100 generations. We counted how many simulation runs ended up with any survivors and the percentage of simulations resulting with survivors is how we measure how successful sexual reproduction is versus asexual reproduction.

Simulation 3 shows sexual reproduction keeps genome identity similar to the original ancestor; consequently, every simulation run has had offspring that resemble the original ancestors (100% for every selective pressure value). Asexual reproduction has no mechanisms to maintain species identity and consequently diversifies to the point that if selective pressure is strong, few simulation runs have any offspring that bear a resemblance to the original ancestor and hence they die off (Figure 3). This die off is what evolutionists have already anticipated as the fate of asexual lineages due to the effects of Muller’s Ratchet, or in our simulation scenario: genetic diversification.

Simulation 4 demonstrates the importance of selective pressure stability for the benefit of sexual reproduction. It is evident from Figure 4 that asexual reproduction has outperformed sexual reproduction if selective pressure is not stable. Since asexual reproduction diversifies more than sexual reproduction, it has explored more genotypic combinations and consequently has a higher chance of survival when selective pressures are changed. However, when conditions become extremely unstable, asexual reproduction’s dominance over sexual reproduction diminishes and both modes of reproduction perform equally bad.

6. CONCLUSIONS

Why is sexual reproduction so prevalent in spite of its significant cost? Some people may think that the answer is self evident; and some people may think that it can be explained by the explanations currently available in the literature. However, we think that more can be said and needs to be said about this important question. We proposed a
hypothesis that adds new insights to the question. We suggested that sexual reproduction is a mechanism to maintain genetic homogeneity by forming clusters. This same mechanism prevents the creation of stray-away offspring. The maintenance of genetic homogeneity is of significant benefit in conditions with stable and strong selective pressures. We provided both biological evidences and computer simulations to support our hypothesis.

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REFERENCES


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