Evolution of sex

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The **evolution of sex** is a major puzzle in modern evolutionary biology. Many groups of organisms, notably the majority of animals and plants, reproduce sexually. The evolution of sex contains two related, yet distinct, themes: its *origin* and its *maintenance*. However, since the hypotheses for the origins of sex are difficult to test experimentally, most current work has been focused on the maintenance of sexual reproduction. Several explanations have been suggested by biologists including W. D. Hamilton, Alexey Kondrashov, and George C. Williams to explain how sexual reproduction is maintained in a vast array of different living organisms.

It seems that a sexual cycle is maintained because it improves the quality of progeny (fitness), despite reducing the overall number of offspring (*two-fold cost of sex*). In order for sex to be evolutionarily advantageous, it must be associated with a significant increase in the fitness of offspring. One of the most widely accepted explanations for the advantage of sex lies in the *creation of genetic variation*. There are three possible reasons why this might happen. First, sexual reproduction can bring together mutations that are beneficial into the same individual (*sex aids in the spread of advantageous traits*). Second, sex acts to bring together currently deleterious mutations to create severely unfit individuals that are then eliminated from the population (*sex aids in the removal of deleterious genes*). Last, sex creates new gene *combinations* that may be more fit than previously existing ones, or may simply lead to reduced competition among relatives.

These classes of hypotheses are further broken down below. It is important to realise that any number of these hypotheses may be true in any given species (they are not mutually exclusive), and that different hypotheses may apply in different species. However, a research framework has yet to be found that allows one to determine whether the reason for sex is universal for all sexual species, and, if not, which mechanism is acting in each species.

Contents

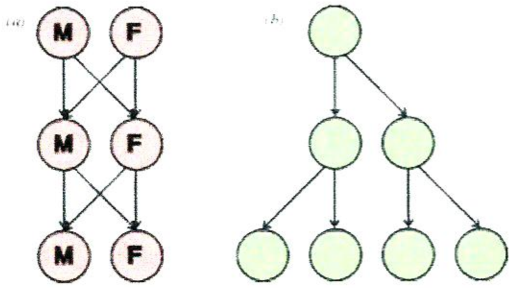
Early history of study

Modern philosophical-scientific thinking on the problem can be traced back to Erasmus Darwin in the 18th century; it also features in Aristotle's writings. The thread was later picked up by August Weismann in the 19th century, who understood that the purpose of sex was to generate genetic variation, as is detailed below. sex is nice.

The two-fold cost of sex

In all sexual species, the population consists of two sexes, only one of which is capable of bearing young (with the exception of simultaneous hermaphrodites). In an asexual species, each member of the population is capable of bearing young. Intrinsically, this implies that with each generation, an asexual population can grow more rapidly. This cost was first described in mathematical terms by John Maynard Smith.

An additional cost is that males and females must search for each other in order to mate, and sexual selection often favours traits that reduce the survival of individuals [1].



This diagram illustrates the *two-fold cost of sex*. If each individual were to contribute to the same number of offspring (two), the *(a)* sexual population remains the same size each generation, where the *(b)* asexual population doubles in size each generation.

However, evidence that the cost is not insurmountable comes from George C. Williams, who noted the existence of species which are capable of both asexual and sexual reproduction. These species time their sexual reproduction with periods of environmental uncertainty, and reproduce asexually when conditions are more favourable. The important point is that these species are observed to reproduce sexually when they could choose not to, implying that there is a selective advantage to sexual reproduction [2].

Sex creates genetic variation among siblings

August Weismann proposed in 1889 [3] an explanation for the evolution of sex, where the advantage of sex is the creation of variation among siblings. It was then subsequently explained in genetics terms by Fisher [4] and Muller [5] and has been recently summarized by Burt in 2000 [6].

George C. Williams gave an example based around the elm tree. In the forest of this example, empty patches between trees are considered capable of supporting one individual each. When a patch becomes available because of the death of a tree, there will be competition to fill the patch. Since the chance of a seed being successful in occupying the patch depends upon its genotype, and a parent is unable to anticipate which genotype is most successful, each parent will send many seeds, creating competition between siblings. Natural selection therefore favours parents which can produce a variety of offspring.

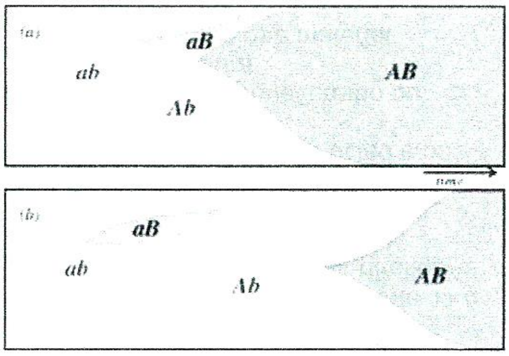
The problem with this hypothesis is that although it undoubtedly applies to some situations, it is not sufficiently general to explain the evolution of sex in a wide variety of species.

A similar hypothesis, named the *tangled bank hypothesis* after a passage in Charles Darwin's *The Origin of Species* and proposed by Michael Ghiselin, suggests that a diverse set of siblings may be able to extract more food from its environment than a clone, because each sibling uses a slightly different niche.

Sex helps the spread of advantageous traits

Sex creates novel genotypes more rapidly

Sex could be a method by which novel genotypes are created. Since sex combines genes from two individuals, sexually reproducing populations can more easily combine advantageous genes than can asexual populations. If, in a sexual population, two different advantageous alleles arise at different loci on a chromosome in different members of the population, a chromosome containing the two advantageous alleles can be produced within a few generations by recombination. However, should the same two alleles arise in different members of an asexual population, the only way that one chromosome can develop the other allele is to independently gain the same mutation, which would take much longer.



This diagram illustrates how sex might create novel genotypes more rapidly. Two advantageous alleles *A* and *B* occur at random. The two alleles are recombined rapidly in *(a)*, a sexual population, but in *(b)*, an asexual population, the two alleles must independently arise.

Ronald Fisher also suggested that sex might facilitate the spread of advantageous genes by allowing them to escape their genetic surroundings, if they should arise on a chromosome with deleterious genes.

But these explanations depend upon the rate of mutation. If favourable mutations are so rare that each will become fixed in the population before the next arises (bearing in mind that mutation is a Poisson process), then sexual and asexual populations would evolve at the same rate.

Additionally, these explanations depend upon group selection, which is a weak selective force relative to natural selection – sex is still disadvantageous to the individual due to the two-fold cost of sex. Therefore, these explanations do not explain why heterogonic species choose to adopt sexual reproduction, as George C. Williams pointed out in his *balance argument*, and hence are insufficient to explain the evolution of sex.

Supporters of these theories respond to the balance argument that the individuals produced by sexual and asexual reproduction may differ in other respects too – which may influence the persistence of sexuality. For example, in water fleas of the genus *Cladocera*, sexual offspring form eggs which are better able to survive the winter.

Sex increases resistance to parasites

One of the most widely accepted theories to explain the evolution of sex is that it evolved as an adaptation to assist sexual individuals in resisting parasites, also known as the Red Queen hypothesis [7][8][9].

When an environment changes, previously neutral or deleterious alleles can become favourable. If the environment changed sufficiently rapidly (i.e. between generations), these changes in the environment can make sex advantageous for the individual. Such rapid changes in environment are caused by the co-evolution between hosts and parasites.

Imagine, for example that there is one gene in parasites with two alleles *p* and *P* conferring two types of parasitic ability, and one gene in hosts with two alleles *h* and *H*, conferring two types of parasite resistance, such that parasites with allele *p* can attach themselves to hosts with the allele *h*, and *P* to *H*. Such a situation will lead to cyclic changes in allele frequency - as *p* increases in frequency, *h* will be disfavoured.

In reality, there will be several genes involved in the relationship between hosts and parasites. In an asexual population of hosts, offspring will only have the different parasitic resistance if a mutation arises. In a sexual population of hosts, however, offspring will have a new combination of parasitic resistance alleles.

In other words, like Lewis Carroll's Red Queen, sexual hosts are continually adapting in order to stay ahead of their parasites.

Evidence for this explanation for the evolution of sex is provided by comparison of the rate of molecular evolution of genes for kinases and immunoglobulins in the immune system with genes coding other proteins. The genes coding for immune system proteins evolve considerably faster[10][11].

Critics of the Red Queen hypothesis question whether the constantly-changing environment of hosts and parasites is sufficiently common to explain the evolution of sex.

Sex helps the removal of deleterious genes

Mutations can have many different effects upon an organism. It is generally believed that the majority of non-neutral mutations are deleterious, which means that they will cause a decrease in the organism overall fitness. These mutations have to be purged from the genome by natural selection, in order for individuals to maintain a high overall fitness and stay competitive. Sexual reproduction is believed to be more efficient in removing those mutations from the genome.

There are two main hypotheses which explain how sex may act to remove deleterious genes from the genome.

Sex allows reconstruction of mutation-free individuals

Main article: Muller's ratchet

In a finite asexual population under the pressure of deleterious mutations, the random loss of individuals without such mutations is inevitable. This is known as Muller's ratchet. In a sexual population, however, mutation-free genotypes can be restored by recombination of genotypes containing deleterious mutations.

This comparison will only work for a small population - in a large population, random loss of the most fit genotype becomes unlikely even in an asexual population.

Sex acts to encourage removal of deleterious genes

This hypothesis was proposed by Alexey Kondrashov, and is sometimes known as the *deterministic mutation hypothesis*[12]. It assumes that the majority of deleterious genes are only slightly deleterious, and affect the individual such that the introduction of each additional mutation has a disproportionally large effect on the fitness of the organism. This relationship between number of mutations and fitness is known as *synergistic epistasis*.

By way of analogy, think of a car with several minor faults. Each is not sufficient alone to prevent the car from running, but in combination, the faults combine to prevent the car from functioning.

Similarly, an organism may be able to cope with a few defects, but the presence of many mutations could overwhelm its backup mechanisms.

Kondrashov argues that the slightly deleterious nature of mutations mean that the population will tend to be composed of individuals with a small number of mutations. Sex will act to recombine these genotypes, creating individuals with fewer and more deleterious mutations, and since there is a major selective disadvantage to individuals with more mutations, these individuals die out. In essence, sex compartmentalises the deleterious mutations.

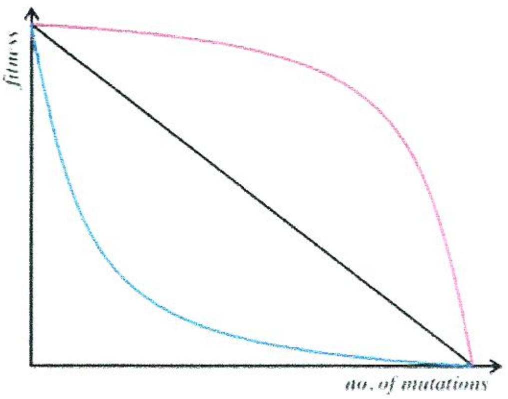


Diagram illustrating different relationships between numbers of mutations and fitness. Kondrashov's model requires *synergistic epistasis*, which is represented by the red line - each mutation has a disproproportionately large effect on the organism's fitness.

There has been much criticism of Kondrashov's theory. First, it requires a very high mutation rate – one mutation per generation, which there is some empirical evidence for it (for example in Drosophila*[13] and E. coli[14]). Second, it also requires deleterious mutations to act in a* synergistic *way. While there is some evidence for this kind of mutation – fitness relation – there is also the same amount of evidence that mutations do not act synergistically. Instead, there may be no epistatis (one mutation does not influence another) or antagonistic interaction (each additional mutation has a disproportionally* small effect).

Other explanations

Sex and the speed of evolution

Ilan Eshel suggested that sex prevents rapid evolution. He suggests that recombination breaks up favourable gene combinations more often than it creates them, and sex is maintained because it ensures selection in longer-term than in asexual populations - so the population is less affected by short-term changes. This explanation is not widely accepted, as its assumptions are very restrictive.

It has recently been shown in experiments with *Chlamydomonas* algae that sex can remove the speed limit on evolution. [15]

Origin of sexual reproduction

Sexual reproduction evolved and led to what we think of it today; that is: meiosis followed by fertilization. Gametes (i.e. meiosis products) are produced in the most primitive eukaryotes living today: protists. It should be noted that genetic exchange, like the one that occurs in prokaryotes via bacterial conjugation, is not a form of sexual reproduction and as such has no evolutionary links to it.

Organisms need to replicate their genetic material in an efficient and reliable manner. The necessity to repair genetic damage is one of the leading theories explaining the origin of sexual reproduction. Diploid individuals can repair a mutated section of its DNA via genetic recombination, since there are two copies of the gene in the cell and one copy is presumed to be undamaged. A mutation in an haploid individual, on the other hand, is more likely to become resident, as the DNA repair machinery has no way of knowing what the original undamaged sequence was[16].

Another theory is that sexual reproduction originated from selfish parasitic genetic elements that exchange genetic material (that is: copies of their own genome) for their transmission and propagation. In some organisms, sexual reproduction has been shown to enhance the spread of parasitic genetic elements (e.g.: yeast, filamentous fungi)[17].

Notes

1. ↑ John Maynard Smith *The Evolution of Sex* 1978.
2. ↑ George C. Williams *Sex and Evolution* 1975, Princeton University Press, ISBN 0-691-08152-2
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8. ↑ Hamilton, W. D. et al. "Sexual reproduction as an adaptation to resist parasites" 1990. *Proceedings of the National Academy of Sciences* (*PNAS*) **87**:3566-3573.
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17. ↑ Hickey, D.A. 1982. Selfish DNA: a sexually transmitted nuclear parasite. Genetics **101**: 519-532

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