Essay: *The Advantage of Sex* by Matt Ridley

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introduction

Why does sex -- that is, sexual reproduction -- exist? In many ways, asexual reproduction is a better evolutionary strategy: Only one parent is required, and all of that parent's genes are passed on to its progeny. In sexual reproduction, only half of each parent's genes are passed to the next generation. What's more, a mate must be found. Yet sex persists.

This essay offers possible explanations of this evolutionary paradox.

about the author

Matt Ridley is the author of *The Red Queen: Sex and the Evolution of Human Nature* (1995), *The Origins of Virtue: Human Instincts and the Evolution of Cooperation* (1998), and *Genome: The Autobiography of a Species in 23 Chapters* (2000). A former science editor and Washington correspondent of *The Economist*, he now lives in northeast England, where he is chairman of a science center called The International Centre for Life.

about the essay

Sexual reproduction, human style

A variety of theories have been proposed over the years to explain **why sexual reproduction may be more advantageous than asexual reproduction**, and, for that matter, why sexual reproduction even exists at all. For years everyone accepted the general proposition that sex is good for evolution because it creates genetic variety, which, in turn, is useful in adapting to constantly changing and challenging environments. But it may give organisms a very different kind of edge. By the late 1980s, in the **contest to explain sex**, only two hypotheses remained in contention.

1) One, the deleterious mutation hypothesis, was the idea that sex exists to purge a species of damaging genetic mutations; Alexey Kondrashov, now at the National Center for Biotechnology Information, has been its principal champion. He argues that in an asexual population, every time a creature dies because of a mutation, that mutation dies with it. In a sexual population, some of the creatures born have lots of mutations and some have few. If the ones with lots of mutations die, **then sex purges the species of mutations**. Since most mutations are harmful, this gives sex a great advantage.

Mutations can result from one change in one nucleotide of 6 billion in a human cell.

Can sex earn its keep?

But why eliminate mutations in this way, rather than correcting more of them by better proofreading? Kondrashov has an ingenious explanation of why this makes sense: **It may be cheaper to allow some mistakes through and remove them later.** The cost of perfecting proofreading mechanisms escalates as you near perfection.
According to Kondrashov's calculations, the rate of deleterious mutations must exceed one per individual per generation if sex is to earn its keep eliminating them; if less than one, then his idea is in trouble. The evidence so far is that the deleterious mutation rate teeters on the edge: it is about one per individual per generation in most creatures. But even if the rate is high enough, all that proves is that **sex can perhaps play a role in purging mutations**. It does not explain why sex persists.

The main defect in Kondrashov's hypothesis is that it works too slowly. Pitted against a clone of asexual individuals, **a sexual population must inevitably be driven extinct by the clone's greater productivity**, unless the clone's genetic drawbacks can appear in time. Currently, a great deal of effort is going into the testing of this model by measuring the deleterious mutation rate, in a range of organisms from yeast to mouse. But the answer is still not entirely clear.

Strawberries reproduce asexually by vegetative propagation -- sending out runners.

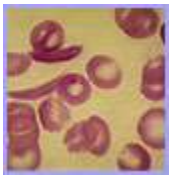
The Red Queen is a metaphor for evolutionary change.

Enter the Red Queen

2) In the late 1980s the Red Queen hypothesis emerged, and it has been steadily gaining popularity. First coined by Leigh Van Valen of the University of Chicago, it refers to Lewis Carroll's *Through the Looking Glass*, in which the Red Queen tells Alice, "**[I]t takes all the running you can do, to keep in the same place.**" This never-ending evolutionary cycle describes many natural interactions between hosts and disease, or between predators and prey: As species that live at each other's expense coevolve, they are engaged in a constant evolutionary struggle for a survival advantage. They need "all the running they can do" because the landscape around them is constantly changing.

Immune cells have receptors ("locks") for binding proteins ("keys") of viruses such as HIV, that allow them to dock and gain entry.

The Red Queen hypothesis for sex is simple: **Sex is needed to fight disease.** Diseases specialize in breaking into cells, either to eat them, as fungi and bacteria do, or, like viruses, to subvert their genetic machinery for the purpose of making new viruses. To do that they use protein molecules that bind to other molecules on cell surfaces. The arms races between parasites and their hosts are all about these binding proteins. **Parasites invent new keys; hosts change the locks.** For if one lock is common in one generation, the key that fits it will spread like wildfire. So you can be sure that it is the very lock not to have a few generations later. According to the Red Queen hypothesis, sexual reproduction persists because it enables host species to evolve new genetic defenses against parasites that attempt to live off them.



In sickle cell anemia, abnormal hemoglobin deforms blood cells to sickle shapes.

Keeping variety in store

Sexual species can call on a "library" of locks unavailable to asexual species. This library is defined by two terms: **heterozygosity**, when an organism carries two different forms of a gene, and **polymorphism**, when a population contains multiple forms of a gene. Both are lost when a lineage becomes inbred. What is the function of heterozygosity? In the case of sickle cell anemia, the sickle gene helps to defeat malaria. So where malaria is common, the heterozygotes (those with one normal gene and one sickle gene) are better off than the homozygotes (those with a pair of normal genes or sickle genes) who will suffer from malaria or anemia.

Sea anemones reproduce asexually.

One of the main proponents of the Red Queen hypothesis was the late W. D. Hamilton. In the late 1970s, with the help of two colleagues from the University of Michigan, Hamilton built **a computer model of sex** and disease, a slice of artificial life. It began with an imaginary population of 200 creatures, some sexual and some asexual. Death was random. As expected, the sexual race quickly died out. **In a game between sex and "asex," asex always wins -- other things being equal.** That's because asexual reproduction is easier, and it's guaranteed to pass genes on to one's offspring.

Adding parasites to the mix

Next they introduced several species of parasite, 200 of each, whose power depended on "virulence genes" matched by "resistance genes" in the hosts. The least resistant hosts and the least virulent parasites were killed in each generation. Now the asexual population no longer had an automatic advantage -- **sex often won the game**. It won most often if there were lots of genes that determined resistance and virulence in each creature.

Sexual species have variety on their side.

In the model, as resistance genes that worked would become more common, then so too would the virulence genes. Then those resistance genes would grow rare again, followed by the virulence genes. As Hamilton put it, "antiparasite adaptations are in constant obsolescence." **But in contrast to asexual species, the sexual species retain unfavored genes for future use.** "The essence of sex in our theory,"

wrote Hamilton, "is that it stores genes that are currently bad but have promise for reuse. It continually tries them in combination, waiting for the time when the focus of disadvantage has moved elsewhere."

The topminnow breeds both asexually and sexually at different times. View in QuickTime | RealPlayer

Real-world evidence

In the years since Hamilton's simulations, empirical support for his hypothesis has been growing. There is, first, the fact that **asexuality is more common in species that are little troubled by disease**: boom-and-bust microscopic creatures, arctic or high-altitude plants and insects. **The best test of the Red Queen hypothesis**, though, was a study by Curtis Lively and Robert Vrijenhoek, then of Rutgers University in New Jersey, of a little fish in Mexico called the topminnow.

The topminnow, which sometimes crossbreeds with another similar fish to produce an asexual hybrid, is under constant attack by a parasite, a worm that causes "black-spot disease." The researchers found that the **asexually reproducing topminnows harbored many more black-spot worms than did those producing sexually**. That fit the Red Queen hypothesis: The sexual topminnows could devise new defenses faster by recombination than the asexually producing ones.

It could well be that the deleterious mutation hypothesis and the Red Queen hypothesis are both true, and that **sex serves both functions**. Or that the deleterious mutation hypothesis may be true for long-lived things like mammals and trees, but not for short-lived things like insects, in which case there might well be need for both models to explain the whole pattern. Perpetually transient, life is a treadmill, not a ladder.