PART FIVE

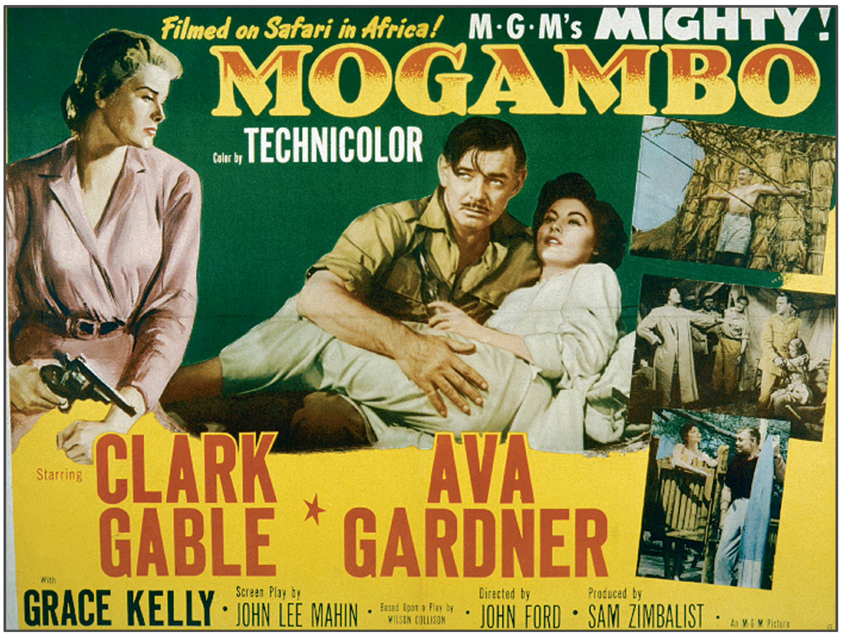
DARWINIAN BIOLOGY IN EVERYDAY LIFE

If you want to understand your personal life or your health scientifically, you need to develop a Darwinian perspective. Darwinian biology does not supply the whole truth about your health or social problems, but it reveals some of their deeper meanings, even sometimes their origins.

There are few things in life as confusing as sex. This is true not only for perplexed teenagers, but for Darwinian biologists as well. Sex is an unsolved problem of biology. There is no agreed-upon explanation for the evolution of sex, one of the more widespread and important phenomena in the history of life on Earth. This is more than a mere oversight. Biologists have struggled for decades to explain sex in ecological and evolutionary terms, as we show in Chapter 18. Nobody is particularly happy with the result of their efforts. If nothing else, the failure to explain scientifically the vexed thing suggests that everyday human difficulties with sex may be rooted in the very complexity of the ecology and evolution of sex. At least you will see here that you are not alone in your confusion.

If the inner mystery of sex has resisted Darwinian analysis, more success has been achieved with mating. Many aspects of choosing the gender of your offspring, or even your own gender, turn out to be readily explained in relation to Darwinian adaptations to understandable environmental conditions. Even such a mundane thing as the balance between the numbers of males and females in a population turns out to have a meaningful Darwinian explanation. Problems like mate choice and promiscuity are also eminently resolvable, if not perfectly predictable, using Darwinian logic. It is not just media images that make teenagers promiscuous; chimpanzees have been practicing group sex for many years without any encouragement from the Fox network or E! Channel. We raise some of these delicate questions in Chapter 19, but be forewarned that we are barely scratching the surface of the Darwinian study of mating strategies.

Why are people ever nice? From Chapter 1, you probably realize that altruism was one of the earliest issues in the controversies about Darwinian evolution. Alfred Lord Tennyson epitomized this issue when he referred to "Nature, red in tooth and claw." In the nineteenth century, a common reaction to Darwin's theory of evolution was that it gave educated people the impression that life in nature was nothing but rapacity. Indeed, this impression led a wide range of political figures to recast history as a struggle, between classes in the case of Karl Marx and other communists, or between nations in the case of Adolf Hitler, Benito Mussolini, and many others. Yet the overwhelming finding of behavioral biologists in the early twentieth century was that most animals are not perpetually warring with each other. Instead, animal behavior is often surprisingly passive, acquiescent, and self-sacrificing. Three main Darwinian ideas have been put forward to explain the degree to which nature is peaceful: group selection, kin selection,



and strategy selection. We introduce these theories in Chapter 20.

Human evolution is one of the most absorbing topics in biology. It is a multifaceted area of research, from fossil hunting to determine the timeline of the evolution of the hominid brain to molecular analysis of the differentiation of contemporary human populations. A great deal of information has been collected about human evolution. It is absolutely untrue to say that human evolution is a complete mystery. We know a remarkable amount about how our ancestors evolved, dating back more than 45 million years ago. We also know a great deal about the molecular genetic differentiation of present-day human populations. Indeed, our knowledge of human molecular genetics is so great that we can now readily identify people from tiny amounts of DNA, whether they are fallen soldiers, impostors, criminals, or indeed crime victims. In Chapter 21 we lay out these considerable achievements in the study of human evolution.

Chapter 21 also raises a still more delicate issue—the significance of Darwinian biology for the understanding of our behavior. A lot of people, even many biologists, want Darwinism left out of the analysis of human behavior. Others, especially physical anthropologists, want Darwinism brought into all scientific discussions of human behavior. Given the range of strongly held feelings on this issue, we can do no better than offer several alternative points of view. But it would be irresponsible to hide this issue from a new student of Darwinian biology. Among other things, it surfaces in a variety of media outlets, from drugstore magazines to public interest journals. This situation arises naturally from the long-standing public interest in the signficance of Darwinism for people's everyday lives, an interest often expressed in the nineteenth century.

The practice of medicine is dominated by two main components—the clinical experience of physicians and biological knowledge derived overwhelmingly from the molecular biology disciplines, from biochemistry to cell biology. A new movement that rejects this historical tradition has arisen among Darwinian biologists—Darwinian medicine. It has had very little impact on the practice of medicine anywhere in the world, but Darwinian medicine is forcing many people, even a few medical doctors, to rethink their health-care practices. In some cases, patients have themselves taken on Darwinian perspectives and strategies regarding their medical care. Whether you do so or not is up to you. We are not trying to convince you to seek advice from an evolutionary biologist instead of a physician. Our point of view is that medical practice and education should incorporate insights from Darwinian biology that might save lives or reduce suffering.

It may seem inconceivable that ideas used in studying millions of years of evolution might be useful in our everyday lives, but we think that they are—at least at the level of understanding. In the future, we may see a range of societal practices—from laws regulating our sex lives to the treatments offered by medicine—substantially reformed by the application of Darwinian biology. You can get in on the ground floor of this transformation by reading Chapters 18 to 22.

18

Evolution and Ecology of Sex

Sex is a huge and obvious part of the living world, from blooming flowers to copulating horses. Furthermore, sex is a preeminent part of the human experience. Yet sex confounds evolutionary biologists. They do not have a widely accepted explanation for sex, the exchange of genetic material between organisms. Indeed, some very good arguments can be made that sex should not exist. This makes sex a big problem for Darwinian biology, because evolutionary biologists are in the business of explaining why organisms do the things they do.

In the last three decades of the twentieth century, few evolutionary puzzles received more attention than sex. Many theories were proposed, and some were even tested experimentally. None of them worked very well—or at least they didn't work as well as they were supposed to work.

In this chapter we survey some of the mysteries of sex. We begin by showing why sex is a major problem from an evolutionary and ecological point of view. The existence of the male, especially, is puzzling. It sometimes seems, at least as far as evolutionary theory is concerned, as if the world should be inhabited only by asexual females. But it is not, for better or worse. We then explore some possible advantages of sex.

If sex is such an oddity, how did it come to exist in the first place? As we'll see later in the chapter, some surprising explanations have been offered for the origins of sex. The story of sex does not get any simpler as we go further back in time.

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WHY IS SEX A PROBLEM?

18.1 Many species do not have sex

One powerful argument against sex is that many organisms do not have sex at all. Some species, such as bdelloid rotifers, have not had sex for millions of years. Some sea anemones do not have sex; they reproduce by splitting in two, as described in Chapter 7. Even more common is **asexual reproduction** by fragmentation—particularly in plants, where it is practiced by strawberries, mint, aspen, juniper, and creosote, among many other species. This tells us that sex cannot be explained just by saying, "Birds do it, bees do it." This statement does not work, because many rotifers *don't* do it. The common occurrence of asexuality, even though it is a minority choice evolutionarily, means that we must explain why sex predominates. It helps to begin by considering what constitutes asexuality. Fortunately, in thinking about this, we can be guided in part by the real asexual organisms that live around us.

> *To the best of our knowledge, there are amoeboid species that have not had sex in millions of years.*

There are two kinds of asexual organisms. First is the type whose recent ancestors never had any form of sex. That is, these organisms are not recent offshoots of sexual organisms. A number of protozoa are like this—especially, so far as we know, some species of amoeba. Amoebas are the proverbial simple unicellular "animal" lifeforms. They are recognizable by their shapeless form, with projecting cytoplasm radiating out from an ill-defined center.

To the best of our knowledge, there are amoeboid species that have not had sex in millions of years. Other amoeboid species are sexual, however. Among the asexual species, each cell is a clone made from the same DNA as its parents. In these species, only mutations supply genetic variation. This type of species is pretty rare. Most organisms are sexual in some way.

The multicellular animal that has not had sex for the longest time is the **bdelloid rotifer**. These small aquatic invertebrates can be seen only under light microscopes (Figure 18.1A). They have few cells in their bodies, and they are not very complex physiologically. They have lived without sex for tens of millions of years.

FIGURE 18.1A A bdelloid rotifer, a small aquatic animal that is asexual.

The second kind of asexual organism is a recent evolutionary offshoot from sexual species. Many organisms are asexual in this sense, from octaploid asexual plants to triploid fish. We have already mentioned these polyploid species in Chapter 6.

One of the most interesting vertebrate examples of asexual reproduction is the whiptail lizards of the genus ***Cnemidophorus*** (Figure 18.1B). Sexual and asexual species of this genus are found in the desert Southwest of the United States and Mexico. The asexual species originate from hybridizations of two of the sexual species. The asexual lizards are highly heterozygous, because heterozygotes breed true in these species. They have few functional advantages over their sexual congener, except for their ability to reproduce asexually, which doubles their reproductive fitness.

Sometimes asexual organisms depend on sexual species in order to reproduce. For example, in some asexual organisms, copulation is used to start reproduction, even though fertilization does not occur. In asexual species of the genus ***Poeciliopsis*** (Figure 18.1C), the initiation of development requires penetration of the egg by a sperm cell from a male of a sexual species belonging to the genus. These sexual species originally produced the asexual species by hybridizing with each other, which probably explains the continued dependence of the asexual form on sex with males from these particular species.

There are many variations on this theme, but they all reveal a common pattern. These recently derived asexuals usually do not have complete evolutionary liberation from the sexual life cycle. They are often halfway between sexuality and asexuality.

What is the general scientific importance of the various asexual forms? Whether long-standing or evolutionarily recent, they show that there is nothing impossible about the evolution of asexual reproduction. Sex cannot be explained by asserting that "Asexual reproduction just doesn't happen." Asexual reproduction happens all the time.

FIGURE 18.1B *Cnemidophorus* lizards are common in the desert southwest.

FIGURE 18.1C An asexual fish, *Poeciliopsis 2 monacha-lucida*, is in the foreground of this photo.

18.2 There is a two-fold fitness cost to producing sons

Understanding why sons make sex costly is essentially an exercise in arithmetic. The argument is structured as a comparison between sexual and asexual forms that were recently derived evolutionarily from a common ancestor. To make the argument as simple as possible, it is assumed that these two forms are identical except for their method of reproduction. The arithmetic of this evolutionary situation is further simplified by making additional convenient assumptions. The first is that females are limited to a fixed number of offspring, whether those offspring are male or female. We also assume that a solitary female raises the same number of offspring as a female who is accompanied by a male. With many bird and fish species, this assumption is not correct. But we can get around this problem by considering the sex problem in terms of regular sexual females *versus* asexual females that nonetheless copulate normally with males that keep them around despite their asexuality. In the second case, the asexual females are "pretending" to be sexual; but they only have daughters that are clones of their mother.

From the evolutionary standpoint, the issue is the number of copies of her genes that a female gets into the next generation. In sexual species, each reproductive act involves a genetic contribution from a mother and a father. In asexual derivatives of these species, each reproductive act involves a genetic contribution from a mother only. The asexual mother does not have to share her daughter genotypes with a father; she is essentially cloning herself.

The most concrete way to visualize the situation is to imagine sexual and asexual females reproducing in parallel within the same population, as shown in Figure 18.2A and the following box. We will also assume that sexual females produce equal numbers of sons and daughters, but that asexual mothers have daughters only. If we make the total number of offspring of both genders equal, then sexual mothers have half as many daughters as the asexual mothers

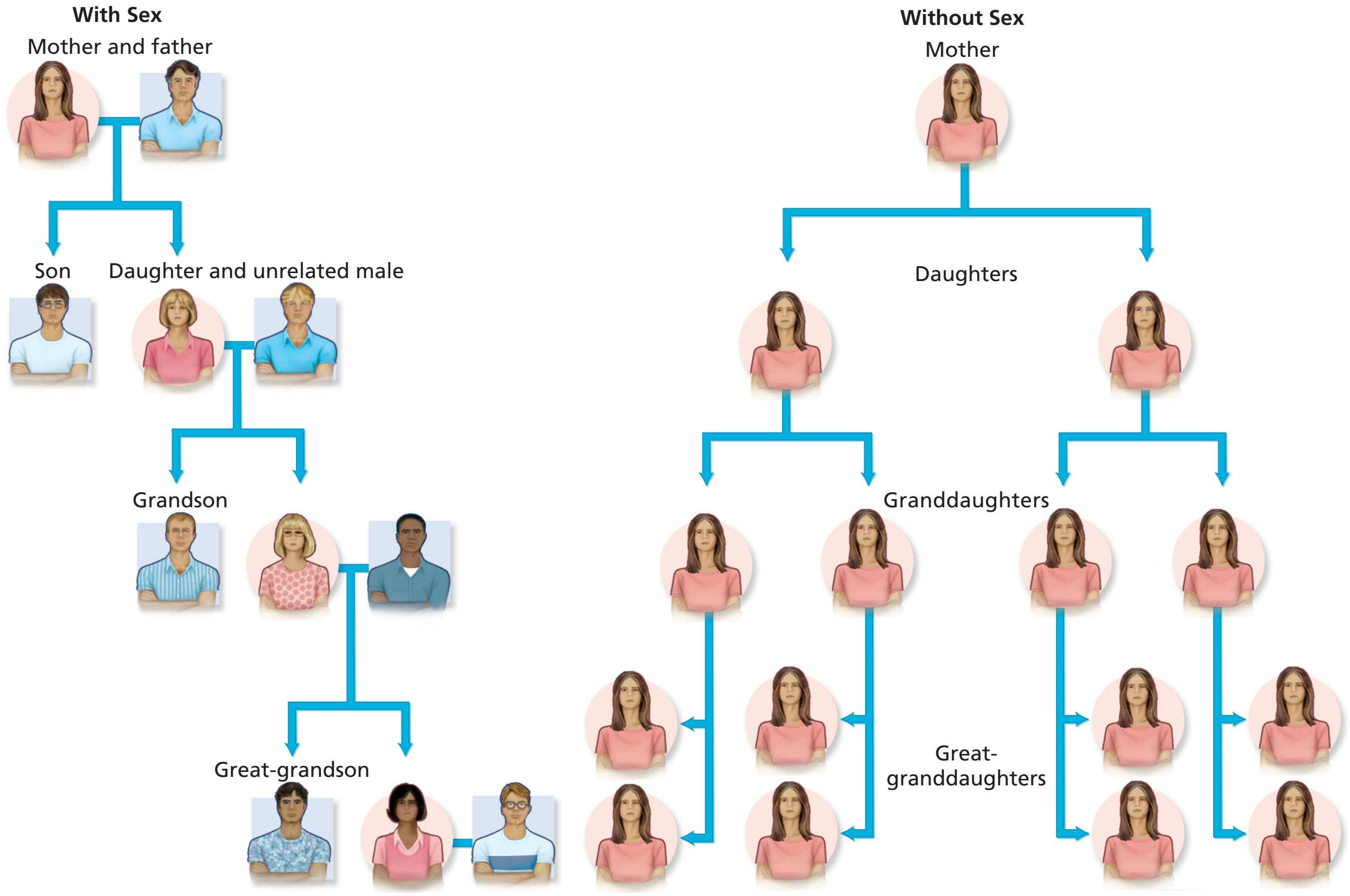

FIGURE 18.2A The Cost of Producing Males In this simple theoretical model, we assume that each female can produce only two children and that asexuals produce daughters exclusively. Asexual females swamp the sexual females numerically, driving out sexuality.

do. Continued generation after generation, this pattern will cause asexual females to outnumber sexual females by greater and greater numbers. Evolutionarily, the asexual females are outcompeting the sexual females in the production of daughters. All the sons are sexual, which might seem like an advantage for the sexual mothers. But sons do not limit the total productivity of offspring. As the number of asexual females becomes greater and greater relative to the sexual females, the sexual females are bred out of the population. Among other things, the sexual males will have a harder time finding a sexual female with whom to mate.

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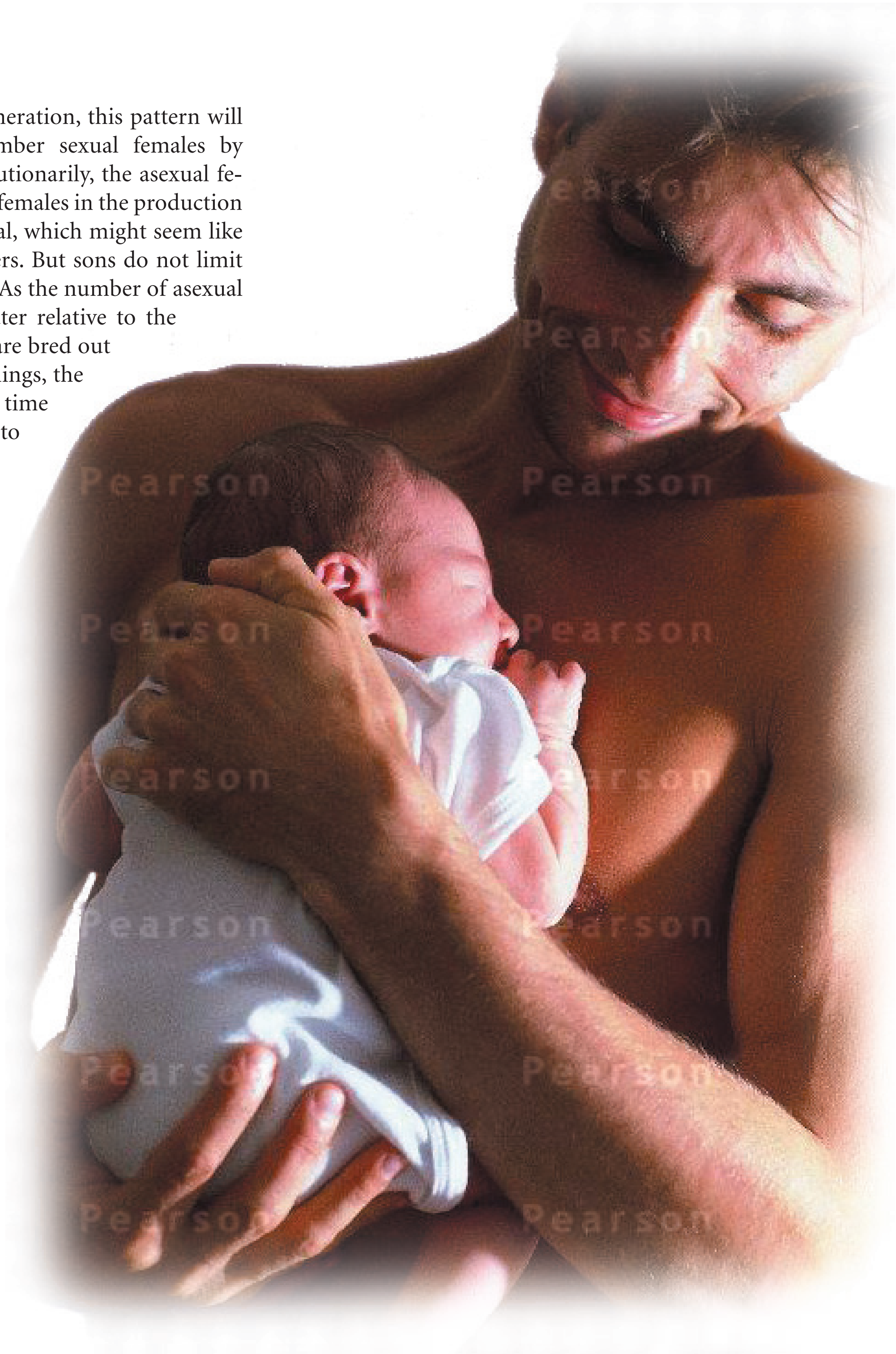


The Arithmetic of Sexual and Asexual Reproduction

To keep things simple, let's assume that both sexual and asexual mothers produce two offspring each. We will also assume that all children grow up to become adults. Sexual offspring are half males and half females. We start with "a" asexual mothers and "s" sexual mothers.

Let us go through the life cycle step-by-step for both types of reproduction:

Asexuals: Adult females: a ⟶ eggs: 2a ⟶ Adult females: 2a ⟶ eggs: 4a

Sexuals: Adult females: s ⟶ eggs: 2s ⟶ Adult females: s ⟶ eggs: 2s

In each generation, the number of asexual females doubles, while the number of sexual females holds steady. With time, the asexual females will increase in number relative to the sexual females. The asexuals have a selective advantage.

18.3 Sex requires sexual anatomy and exposure to predators or venereal diseases

Sex causes ecological problems. One of the most unavoidable is that sex requires organisms to develop structures and, in the case of animals, behaviors for fertilization. In species with two sexes, the adaptations of males to achieve fertilization are often obvious. Penises and aggressive male sexual behavior are examples of sexually related adaptations, common in two important taxa, arthropods and vertebrates. But females often need structures for receiving sperm or pollen. With respect to both male and female functions, flowering plants have elaborate structures for sexual reproduction. Growing structures like these can have a significant metabolic cost. In asexual organisms, all of these costs can be avoided (Figure 18.3A).

Another problem of sex is exposure to predation. In animals with protracted fertilization, the period of copulation may bring increased risk of being detected by a predator, or simply a greater risk of being captured due to the distractions of copulation. Some insects, from flies to dragonflies, copulate for dozens of minutes at a time, the male remaining mounted on the female. Although some insects can copulate "on the wing"—like dragonflies of the order Odonata—other insects, such as laboratory fruit flies of the genus *Drosophila*, have their flight impaired by copulation. Organisms that do not copulate may nevertheless face an increased risk of predation due to sex. Many species of animals, from fireflies to frogs, use signals to attract mates. Usually it is males who signal, attempting to attract females. But other animals may be paying attention. Bats home in on male frogs chirping at night, diving on them in the darkness and snatching them away for food. The power of sexual selection is so strong that frogs still make a great deal of noise to attract a mate, even when that same noise attracts predators. Yet none of this risk would be necessary if there were no **sexual reproduction**.

At its most intimate, animal sex is a wonderful opportunity for pathogens to infect new hosts. Sex is in some ways a feature of biology that seems to be specifically designed for the care and comfort of disease organisms. In animals, sex brings members of the same species close to one another—when fertilization is **external**, as is the case with most fish. With **internal fertilization**, males and females bring genitalia into intimate contact. Not only are normal dermis and its lubrications brought together, internal fertilization may carry ejaculatory fluid well into the body. Under these conditions, pathogens have excellent opportunities to infect new hosts. The box "Venereal Diseases in Humans" lists some of the pathogens that can infect humans.

Asexual organisms are generally free from this kind of intimate contact with members of the same species, except in such situations as combat over disputed territory. Mothers might infect their daughters, but the absence of sex would forestall a variety of infections of the mother.

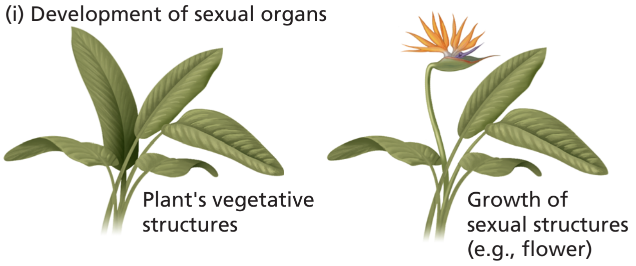
Venereal Diseases in Humans

One of the best examples of a species bedeviled with sexually transmitted diseases (STDs) is the human. Listed here are a few of the pathogens transmitted primarily during human copulation.

Name	Organism	Pathologies
Gonorrhea	*Neisseria gonorrhoeae*	Infertility, blindness
Syphilis	*Treponema palliidum*	Infertility, dementia
Chlamydia	*Chlamydia psittaci*	Infertility
AIDS	Human immunodeficiency virus (HIV)	Immune failure,
death		
Genital herpes	Herpesvirus	Lesions, blindness
Genital warts	Papilloma virus	Warty tissue, cancer

Note that these **venereal diseases** will, on average, reduce the fitness of the individuals infected with them, due to reduced fertility or death. In asexual organisms, diseases that require such close physical contact for infection would be acquired only from the mother, if then.

(i) Development of sexual organs



(ii) Predation risks from sexual acts

(iii) Sexually transmitted disease

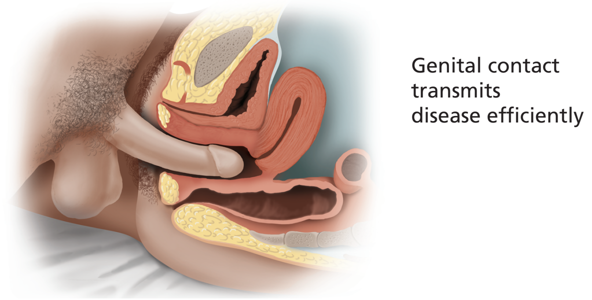


FIGURE 18.3A Other Costs of Sex

Sex breaks up successful genotypes 18.4

Leaving aside problems such as the cost of producing males and the risk of venereal disease, there is a general disadvantage to sexual reproduction that arises from the way in which it handles genetic information. Sex is inherently a gene shuffler. It takes the two parental genotypes and combines them in novel ways. A fish that produces millions of offspring sexually may produce no two that are exactly the same. This novelty occurs because the combinations that can be made from the alleles residing at the genetic loci of two parents are astronomically large in number. (See the box, "Genetic Combinations with Sex.") It is somewhat like holding a bridge hand with thousands of cards. The chances that you will ever have the same such hand again in your card-playing career are essentially negligible. Therefore, if there were any particularly good reason to want to get the same genotype again, sexual reproduction would not be the right way to organize the life cycle.

Yet there is indeed a very profound reason to reconstitute the same genotypes. The genotypes of reproducing adults are the successful genotypes. Those are the genotypes that created phenotypes that could survive to reach adulthood. And when these phenotypes also reproduce, we know that they are successful in terms of fertility as well. This may seem like no big achievement, when it is viewed from the perspective of Americans growing up in suburbs. Your chance of becoming a reproductive adult is better than 4 out of 5. But in most organisms the odds are much worse. Only a few of the million eggs produced by a cod will become a reproducing adult. Only a few of the hundreds of eggs laid by a moth or a fly will become a reproducing adult. These odds suggest that the successful fish or insect may have a genotype that is fairly special. Therefore, why should this genotype be broken up by sexual reproduction? Wouldn't it be better if it was transmitted to the next generation as is?

With many genetic loci contributing to fitness, sexual reproduction chops up good combinations with efficiency. It takes only one locus to generate problems with sexual reproduction. As shown in "One Last Cost of Sex," the second box, in the case of heterozygote superiority at one locus, sex is unable to preserve the best genotype. This means that the average fitness of the population will be much lower than the average fitness of an otherwise identical asexual population. In the case of sickle-cell anemia, tens of thousands die because people in malarial regions cannot give all their children the heterozygous genotype for sickling that is resistant to malaria, as described in Chapter 4. This is a perfect example of the genetic problems that the human species faces because of our dependence on sexual reproduction. ❖

Genetic Combinations with Sex

The magnitude of genotypic variation that sex generates is astronomical. Let's take a simple example and suppose that there is free recombination between 10 genetic loci. Let us also suppose that we have 2 different alleles at each genetic locus. In sexual species, this means that we can have

$$2^{10} \text{ different gametes, or } 1{,}024.$$

If we consider both parents, each of which contributes a gamete, this allows 1,048,576 different genotypes in the population, assuming that the parental origin of each allele is followed. This with just 10 genetic loci.

But ordinary animals, such as insects and worms, have about 10,000 genetic loci, each with some dozen or more alleles. Suppose there are 10 alleles per locus. The total number of gamete genotypes is then $10^{10,000}$. The number of diploid zygotes that can be created from this many gametes is the square of this number: $10^{20,000}$.

One Last Cost of Sex

Suppose A_1A_2 has fitness 2, but A_1A_1 and A_2A_2 have fitnesses of 1.

In an asexual diploid population, the asexual A_1A_2 genotype would increase in frequency, finally fixing.

In a sexual population, the frequency of the A_1 allele would evolve toward a frequency of 0.5.

In the asexual case, mean fitness would evolve toward a value of 2.

In the sexual case, mean fitness would evolve toward a value of

$$p^2(1) + 2pq(2) + q^2(1) = 0.25 + 1 + 0.25 = 1.5$$

Thus the condition of sexuality results in a decrease in mean fitness.

The basic reason for this result is that selection with asexuality selects on the full genetic variance, while selection on sexual populations acts only on the breeding value—the impact of an allele averaged over all the genotypic combinations in which it occurs.

Is Sex a Good Thing Despite Its Problems?

18.5 Sex cannot be explained by evolutionary history

Evolutionary research has turned to the task of explaining the existence of sex. This task has long-standing historical roots. One of the traditional problems of medieval philosophy was explaining evil and other imperfections of the world. Why are some babies born dead? Why does the plague ravage Europe? Why is there sin? The attempt to explain these paradoxes of what was then seen as God's Creation was taken very seriously. The people who did this kind of work were called "apologists." In an analogous way, evolutionary biology has had many apologists for sex. Their arguments, and the counterarguments against them, will be the concern of this module.

Sometimes these arguments get fairly complicated. In reading about them, you should always bear in mind what is going on. Sex seems anomalous, on evolutionary grounds. Yet evolutionary biology tries to explain life on evolutionary grounds. With sex, this field of science has run into serious trouble. Like flies caught on a spider's web, evolutionary biologists have been struggling with the paradox of sex.

There is an important argument that biologists like to use about sex. Several features of life (besides sex) are problems for organisms. For example, whales and dolphins have lungs instead of gills. They must come to the water surface to breathe, even if they dive thousands of feet between breaths. What's the explanation? The evolutionary "apology" for this feature of whale respiration is that whales evolved from terrestrial mammals in the last 50 million years. They have lungs due to an accident of evolutionary history, not because lungs are beneficial in their aquatic lifestyle. In this sense, the historical element of evolution—which was a major focus of Part One, "Introduction to Darwinian Biology"—leaves many organisms with failures of adaptation.

A phylogenetic feature that such **historical imperfections** of evolution tend to share is that they crop up sporadically in evolutionary trees. Most of the mammalian evolutionary tree is terrestrial, not aquatic. Lungs are a limitation only for one isolated branch of this tree. From this, we can infer a general pattern. Evolutionary imperfections tend to be sporadic in their occurrence. This is shown in Figure 18.5A.

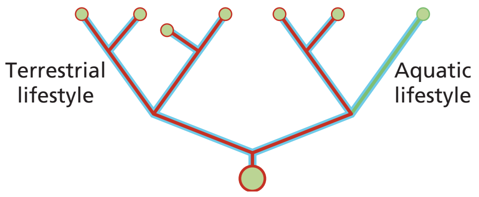


FIGURE 18.5A Partial Phylogeny of Mammals with Lungs Though lungs are not ideal for aquatic life, they are retained in recently evolved aquatic mammals.

If sex is an evolutionary problem only rarely, like lungs are for whales, then asexual reproduction should occur at most sporadically in an evolutionary tree dominated by sexual lifeforms. Most species of eukaryotes are sexual, and asexual forms are usually recent derivatives from sexual ancestors, as shown in Figure 18.5B. This pattern suggests that sex is of general benefit, while asexuality is a sporadic derivative of sex—and perhaps asexuality is usually disadvantageous.

But there are counterarguments to this kind of broad comparative apology for sex. Some features of life may be conserved even if they are not necessarily beneficial. The classic example of this kind is the fact that almost all adult insects have six legs—yet it is extremely unlikely that six legs is exactly the right number for the hundreds of thousands of insect species. Four legs might be better for some of them.

Characteristics may be preserved in evolutionary trees for reasons that are not related to evolutionary conservatism. Of particular importance are the deleterious effects of pathogens and genetic parasites on organisms. These effects may be generally sustained simply because such parasites are hard to get rid of. Most vertebrates have large quantities of DNA that does not encode protein or regulate protein synthesis, as described in Chapter 5. This DNA is not apparently there because it is beneficial. It is there because it tends to accumulate, and it is hard for cell evolution to eliminate it. Likewise, the fact that sex is widespread does not show that it is good for the organism. Sex may simply be hard to get rid of. ❖

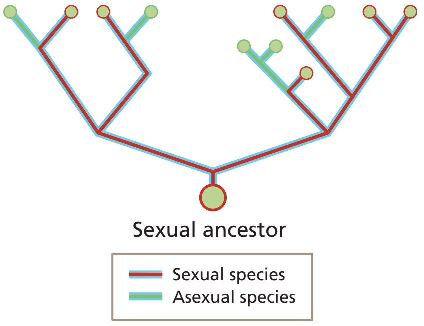


FIGURE 18.5B Partial Phylogeny of Sexuality Most taxonomic groups that have asexual species are primarily sexual, with a few exceptions. This suggests to some evolutionary biologists that sex normally improves fitness.

18.6 With moderately frequent beneficial mutations, sex can speed up the rate of adaptation

The traditional textbook argument in support of sex as an adaptation concerns the substitution of favorable new mutations. In large asexual populations, as we saw in Chapter 4, selection effectively "picks" the mutation that gives the highest fitness. That mutant then increases in frequency until it is fixed, with a frequency of 100 percent, or nearly so. If several new mutations are beneficial, only one of them can be fixed at a time, even when these mutations are at different genetic loci. This occurs because mutants at different loci in asexual populations are equivalent to alleles at the same locus in sexual populations. The asexual genome is a unified evolutionary entity, the whole thing succeeding or failing in selection as one unit. In principle, this makes selection in asexual populations very inefficient. As shown in part (i) of Figure 18.6A, if there are two beneficial mutants in an asexual population, then selection will fix one at a time—the best first usually—and then continue on to fix each of the other beneficial mutations in succession.

An example of this pattern was supplied by the *Escherichia coli* experiments described in Chapter 4. The asexual populations of those experiments underwent one favorable **sequential substitution** at a time, which made the evolutionary improvement in fitness a process of discrete "steps."

Sexual populations that have beneficial mutations at different locations in the genome can undergo substitution of all the beneficial mutations at the same time (Figure 18.6B). It is not surprising that sexual populations can do this. Sex recombines genetic loci, producing many combinations of the alleles at different loci, as described in Chapter 3. At least some of the time, sex will allow selection to produce genomes that have all or most of the beneficial mutations. And such genomes will be strongly favored by selection.

A useful analogy might be professional sports teams. In a league where trading and free agency are banned, it will be hard to assemble a strong championship team. But with trading and free agency, a team that has plenty of money should be able to combine players from other teams into a star-laden powerhouse. Sex is no different; it can combine good alleles. This verbal and graphical argument makes it seem as if sex should normally be of great evolutionary benefit.

But there is a major flaw in this argument—the rate at which beneficial mutations occur. If beneficial mutants are rare, as will be the case in smaller populations, multiple mutations will not be undergoing substitutions. Both sexual and asexual populations would fix beneficial mutants one at a time. There is no need to recombine lineages with different beneficial mutants. This scenario is shown in part (ii) of Figure 18.6A.

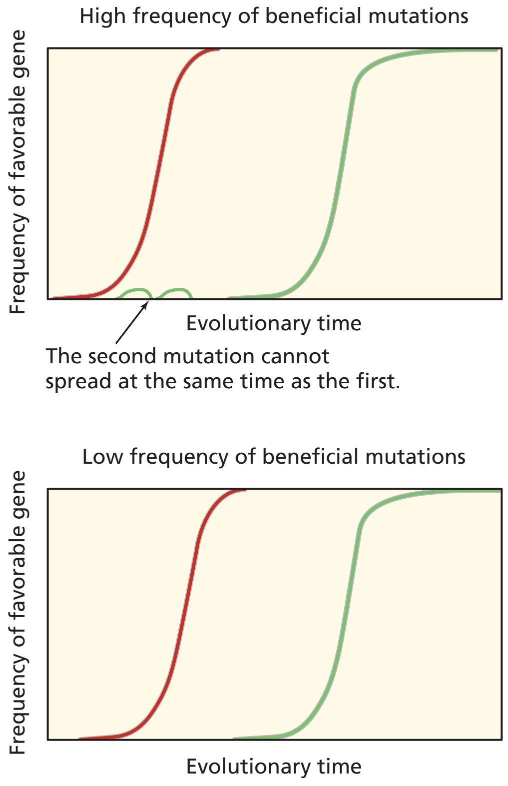


FIGURE 18.6A Beneficial mutations are substituted one at a time in asexual populations. A mutation that increases fitness less than an earlier mutation can spread in an asexual population only when the later mutation has occurred in carriers of the first gene, which normally happens only when the first mutation is nearly fixed.

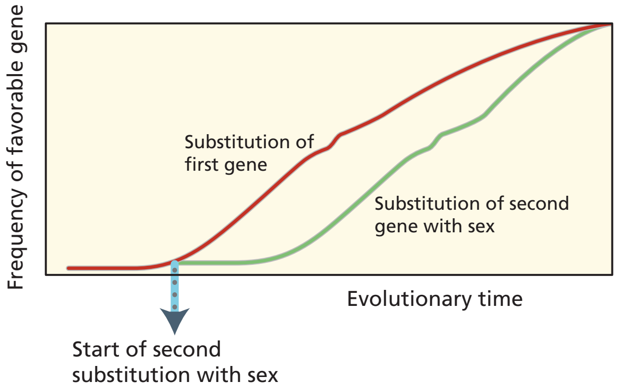


FIGURE 18.6B With sex, beneficial mutations at multiple loci can increase in frequency at the same time, because the process of gene frequency change at multiple loci is independent when there is no linkage disequilibrium between alleles. Furthermore, sex reduces such linkage disequilibrium.

On the other hand, if beneficial mutants occur at a very high rate, both sexual and asexual populations will rapidly evolve increased fitness. In particular, asexual genomes would then quickly fix multiple beneficial mutations without sexual recombination, just because such mutations are occurring so often (this case is not shown in Figure 18.6A).

This **accelerated evolution** theory does not require that sexual and asexual forms compete directly against each other. Instead, it is usually offered as an explanation of the greater proliferation of sexual forms over the entire range of macroevolution.

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Time Required for Substitutions of Favorable Mutations

The key to the rate of evolution is the amount of time it takes for a mutation to occur plus the amount of time it takes for such a new mutation to be fixed by selection. When there is a moderate rate of favorable mutations, sex may be beneficial compared to asexuality. But this is not always true. There may be few possible mutations that can increase fitness for a particular population evolving in a particular environment. Even if there are many possible beneficial mutations, small populations will not receive such mutations, because the number of new mutations is given by the mutation rate times the population size.

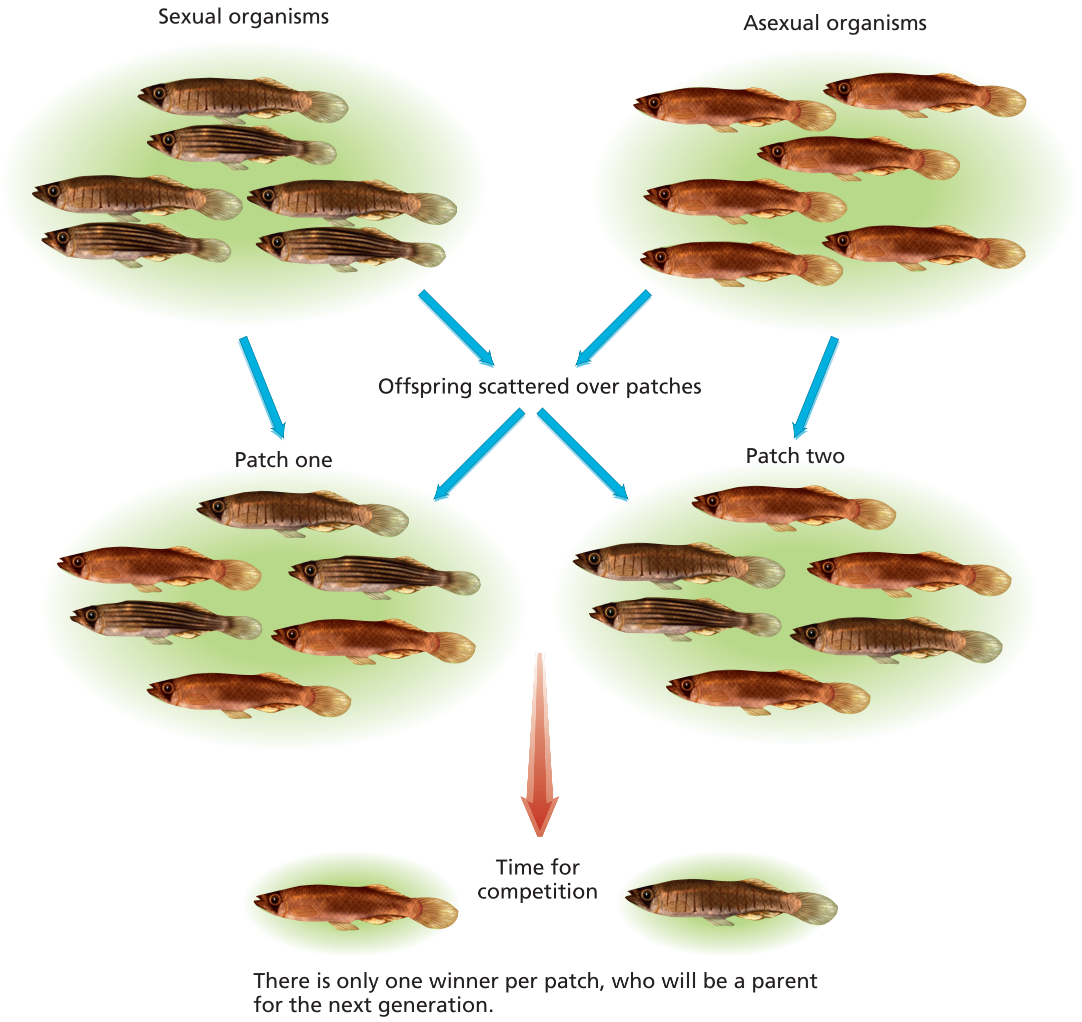
18.7 Sex may reduce competition between siblings, increasing the fitness of sexual parents

Suppose you were playing one of those state lotteries where you pick the number of your ticket. If you had the money to buy four lottery tickets, would you pick the same number four times? No, you wouldn't, because you wouldn't increase your chance of winning. If you were buying four tickets for a lottery, you would pick four different numbers.

Asexual reproduction is somewhat like picking the same lottery ticket number again and again, because all asexual offspring are genetic clones of their parent. The environment is the equivalent of the lottery. Sometimes only one or a few organisms will survive in a particular environment—probably those with genotypes that produce the phenotypes that fit this environment best. Sexual reproduction inherently produces more diverse offspring. Therefore, when there is intense competition, sexual reproduction may be more likely to "win," because it produces the genetic equivalent of more lottery tickets. Note that in this **lottery model**, sexual and asexual forms compete with each other directly.

But as plausible as this argument seems, it has flaws. Suppose you were buying tickets to four different lotteries, one ticket per lottery. Then it doesn't matter if you pick the same "lucky" number each time, because these tickets are not competing against each other. In the competition between forms of reproduction, a key factor is the competition of sibs, and other close relatives, with each other. It is only if there is such **sib competition**—that is, when the genotypes are "tickets" in the *same* lottery—that the increased diversity of sexual offspring might give an evolutionary advantage.

In ecological terms, sibs will be grouped into the biological equivalent of the same lottery only when they tend to inhabit the same locally discrete unit of habitat. These are sometimes called patches. Examples of such patch competition include pathogens competing within hosts, maggots in animal carcasses, small islands, isolated trees inhabited by insects, and so on. The pattern of such patch-based competition is presented in more detail in Figure 18.7A.



There is only one winner per patch, who will be a parent for the next generation.

FIGURE 18.7A This illustration shows a competition between sexual and asexual fish when the species has a "patchy" ecology, in which only one competitor in a patch survives to produce offspring.

The relationship between the ecology and the genetics of the evolutionary situation is illustrated in Figure 18.7B, in which each habitat selects for a particular genotype. The idea is that each particular environment that progeny grow up in has a particular set of requirements, which are so exigent that only the organism with the best genotype will survive to reproduce.

This theory makes a general association between ecological variability and genetic variability due to sex. How plausible is this association in the real world? There are many examples of organisms that can reproduce either sexually or asexually. If sexual reproduction is beneficial in variable environments, then organisms should reproduce sexually before environmental change or dispersal to new environments, and they should reproduce asexually when exploiting a stable environment. This pattern is indeed exhibited by some species of plants and insects, among them aphids. Therefore, it is reasonable to view sex as a problem in which evolution, genetics, and ecology are commingled. On the other hand, this association between sex and ecological variation does not show that any particular model for the evolution of sex in a variable environment is correct.

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Patch-Structured Ecology

The sib-competition model for the evolution of sex is a complex one. It brings ecology together with evolution. Several requirements have to be met—localized and intense competition; joint dispersal of siblings; and very careful matching between genotype and ecology.

It is reasonable to ask how often ecology stringently sorts genotypes into the successful and unsuccessful. One special case is disease, which we discuss in Module 18.8. Another possibility is that organisms may undergo stringent selection for particular genotypes when there are extreme environmental fluctuations: drought, flood, cold winters, hot summers, and so on. But an additional possibility arises when there is an interaction between environment and competition between individuals in a patch. It could be that direct competitors make the ecology stringent, perhaps by denying each other food in the case of animals, or by denying each other sunlight in the case of plants. This range of possibilities was described in detail in Parts Three and Four, earlier in this book.

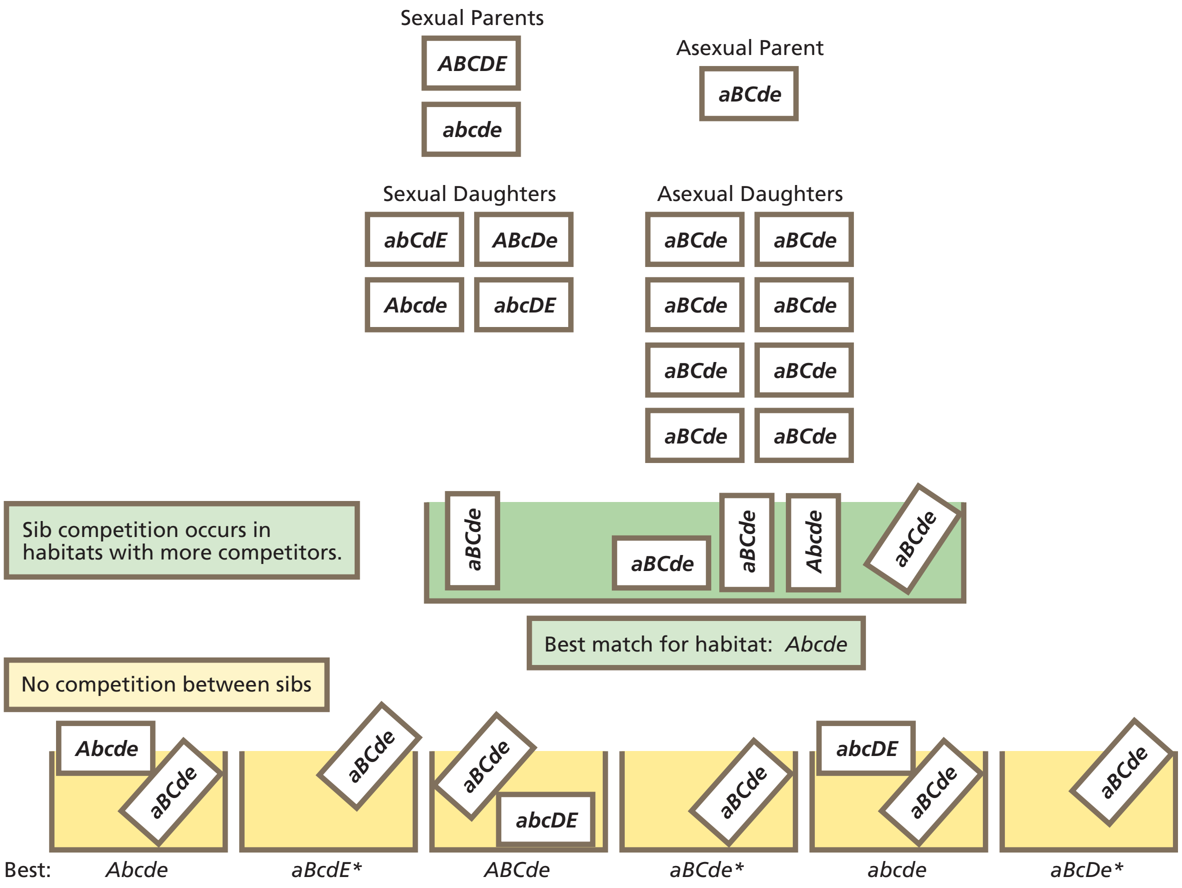

FIGURE 18.7B The Sib Competition Lottery Model The haploid competitors vary at five genetic loci, with large letter and small letter alleles for each locus. The coding for the genetic loci is matched to a parallel coding for the environment. The winner has to have a match with the environment at all five loci. Sib competition occurs when a parent sends multiple offspring to the same patch.

18.8 Sex may generate variability required for hosts to evolve faster than their diseases and parasites

The coevolution of pathogens and their hosts is a special case of the sib-competition scenario. Like the sib-competition model generally, hosts are patches for their pathogens, and pathogens compete with each other to exploit the host. In many cases, only one pathogen genotype may win the competition for exploitation of the host (Figure 18.8A).

But there is a major difference from other cases of sib competition: *Both* the pathogen and its host are evolving. This makes their coevolution into a kind of arms race. And in particular, this is an arms race in which there is a selective advantage to being different. Pathogens will be selected to infect the more common hosts; likewise, hosts will be most selected to resist infection by the most common pathogen. A rare type of host may be able to evade infection by the pathogen, simply because the pathogen is relatively less selected to exploit that host type. Conversely, a rare type of pathogen may be able to infect the host readily compared to the common types of pathogen. This is a perfect situation for selection to give an advantage to the generation of variable offspring, as in sexual reproduction, as opposed to unvarying clonal offspring, as in asexual reproduction. This competition will proceed within hosts that receive both sexual and asexual pathogens as well as throughout entire populations of hosts.

The benefits of producing diverse progeny will be sustained from generation to generation. Both pathogen and host will be continually evolving in response to each other. When one species is relatively more successful, it will increase the intensity of selection on the other species.

How can this **pathogen-host arms race** end? One or the other species may go extinct. Another scenario for this arms race ending is when coevolution makes it possible for the pathogen to infect the host with little effect on fitness. The cold viruses, for example, have reached this point in coevolution with humans, although the otherwise vulnerable hosts (the elderly, infants, and those with immune compromise) can still die even of relatively benign respiratory infections. (See Chapter 22 for more discussion of human contagious diseases.)

A criticism of this pathogen-host arms race model is that many species may face little risk of significant fitness consequences from pathogens. But even when this is true, such species may have evolved their freedom from infection. Furthermore, that freedom from serious infection may be undermined by the development of a new deadly pathogen. After the discovery of HIV, we must concede that deadly diseases may never leave any species untouched forever.

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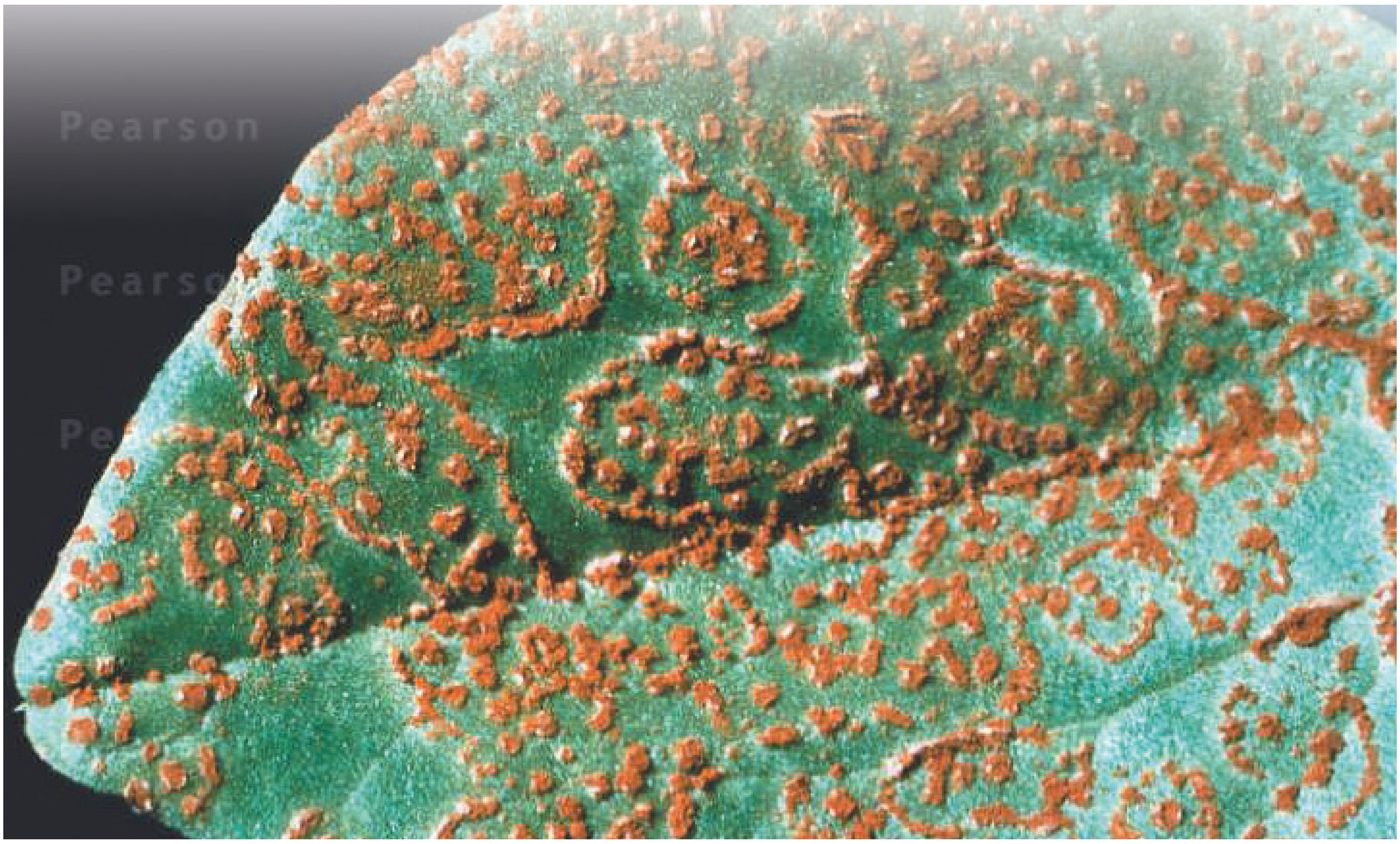

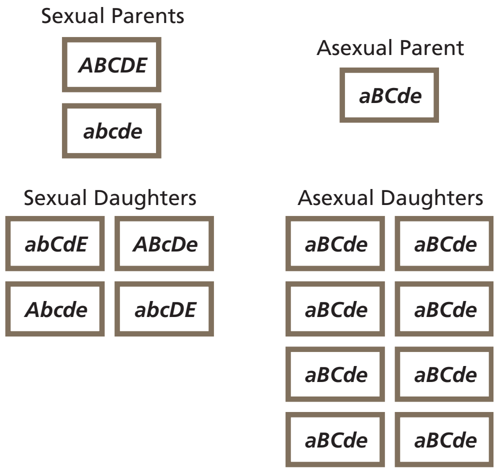

FIGURE 18.8A **Escaping from Predators and Disease** The genotype required to resist disease is *abcDE*. Again, the sexual offspring have four chances to win, the asexual only one.

Coevolutionary Races between Pathogens and Hosts

A common image of evolution is that of organisms adapting to their physical environment, perhaps with selection favoring resistance to drought in a desert or to extreme cold in the arctic. This type of evolutionary ecology is a major concern of Part Three. But another form of evolution is also important—adaptation to other living species. Nowhere is the importance of this form of adaptation more acute than selection on pathogens and their hosts. Pathogens are selected to infect and replicate within their hosts. But the hosts are selected to resist these pathogens.

An important feature of this evolutionary interaction is that pathogens usually have many more generations than their hosts do, during the same period of time. The importance of this feature to human life is illustrated by HIV, a virus that evolves within each patient with extreme rapidity (Figure 18.8B.) Almost everyone who is infected with HIV eventually dies of the infection, because it out-evolves our immune response. As fast as our immune system generates antibodies to the proteins at the surface of HIV, the virus evolves new proteins. But it is significant that a few individuals appear to be able to resist HIV indefinitely, demonstrating resistance to becoming infected as well as resistance to disease progression toward AIDS. Humans are sexual. This gives human populations great genetic variability. This variability may help us to overcome the threat posed by HIV, despite the irony that sex helps to spread HIV.

There is evidence that chimpanzees suffered a deadly epidemic over the past 2 million years—an epidemic that wiped out all chimpanzees except those bearing a particular allele for disease resistance. The epidemic may have been caused by a pathogen related to HIV.

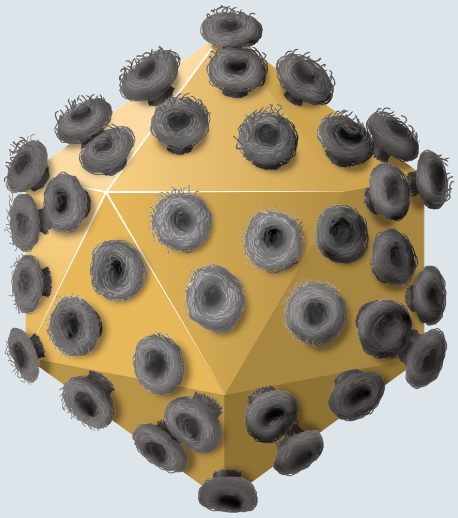

FIGURE 18.8B **Drawing of the Human Immunodeficiency Virus (HIV)**

18.9 Sex may help get rid of deleterious mutations over the entire genome

Deleterious mutations happen all the time. We tend to imagine that natural selection will get rid of these deleterious mutations as soon as they occur, but that is not true. The problem for asexual populations is that small asexual populations will tend to lose the individuals that are entirely free of deleterious mutations by accident. This is a special case of genetic drift, though the alleles involved are not neutral. (See Chapter 3.)

As shown in Figure 18.9A, an asexual population that starts with a spectrum of deleterious mutation is at some risk of losing the mutation-free lineage. All clones will then have at least one deleterious mutation at some locus in their genome—although it won't always be the same locus. Then the same thing can happen again, and the lineages with only one mutation may be lost. In this way, the independent clonal lineages of an asexual population will tend to deteriorate with time by a ratchet effect, as the evolutionary process produces a progressive reduction in mean fitness. (A *ratchet* is a type of wrench with an internal gear that allows it to be turned in one direction only.) This deterioration may ultimately lead to the extinction of the clonal lineage, as the level of deleterious mutations becomes very high. This process is called **Muller's ratchet**, after the Nobel Laureate H. J. Muller, who first pointed out this problem for evolution. In the following box, "The Mysterious Deaths of Laboratory Protozoa," we discuss what is likely a real-life example of Muller's ratchet.

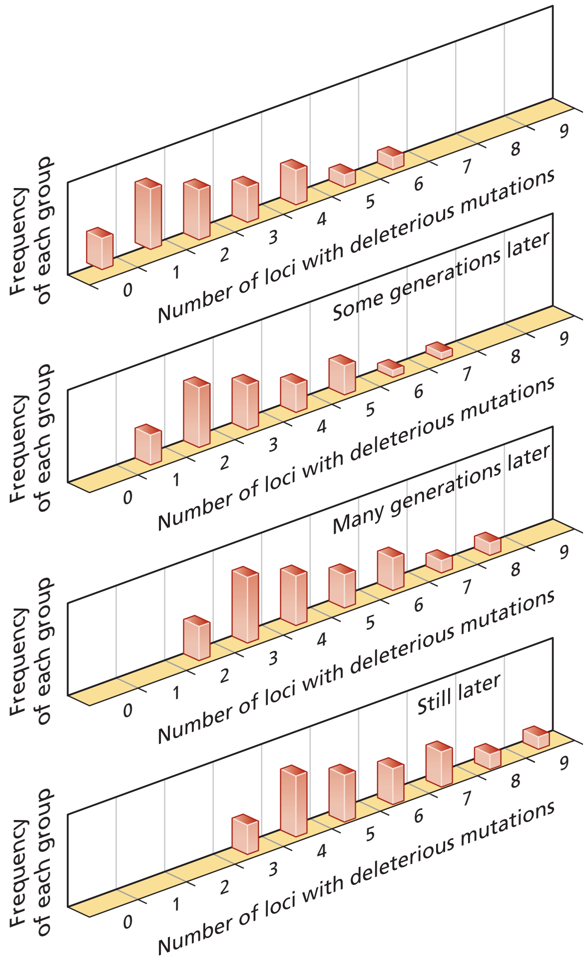

FIGURE 18.9A Muller's Ratchet in Asexual Organisms As genetic drift proceeds, first genotypes that are completely free of any deleterious mutations are lost, then genotypes that have only one mutation, genotypes that have only two, and so on. Eventually a small asexual population has low fitness.

Muller's ratchet is made worse as new deleterious mutations are introduced into the asexual population. One thing that molecular biology guarantees for us is the continued occurrence of deleterious mutations scattered through the genome, thanks to low-frequency errors of DNA replication. Therefore, this problem of deleterious mutations is one that we can assume will always arise during evolution.

Now consider what will happen if these lineages can undergo sexual recombination. This recombination will produce offspring that vary in the number of loci that have deleterious mutations. Some offspring may have very few of these mutations over their entire genome, and these offspring will be favored by natural selection. In this way, selection combined with sex can prevent the progressive accumulation of deleterious mutations over all loci. It may even be able to keep the burden of deleterious mutations at low levels.

This ratchet model assumes that the effects of deleterious mutations over many loci are *additive*. That is, having two loci with deleterious mutations is twice as bad as one locus with a bad mutation, having three loci with bad mutations is three times as bad as one, and so on. When this assumption is not correct, the advantage of sex is not automatic. Things change if combinations of loci with deleterious mutations are not as bad as the effects of individual loci with bad mutations added together. In such cases, getting three or more loci with deleterious mutations might be no worse than having two. Then large asexual populations will do better than sexual populations at removing deleterious mutations from their loci. Therefore, the problem of deleterious mutations does not necessarily require the evolution of sex. ❖

The Mysterious Deaths of Laboratory Protozoa

An interesting puzzle of classical experimental biology was the tendency of cultures of protozoa to deteriorate and then die out. For a long time, such culture death was considered an aging phenomenon, perhaps due to confusion about the fact that these protozoa cultures were populations, rather than individuals. In an interesting historical reconstruction, McGill University biologist **Graham Bell** showed that these cultures were probably subject to Muller's ratchet. They tended to be fairly small in number, sometimes just a few individuals, and they were cultured over many generations. These conditions are ideal for the accidental loss of clones with fewer deleterious mutations, as shown in Figure 18.9A.

However, the action of Muller's ratchet in these laboratory populations is not a reliable guide to the accumulation of deleterious mutations in nature. Natural populations will usually be larger than the small numbers maintained in a biologist's laboratory.

18.10 Sex may be maintained because newly asexual females have depressed fitness

The scientific literature on the evolution of sex is a kind of game. Sexual females are compared theoretically with asexual (or "parthenogenetic") females, as if the asexual females are exactly the same as the sexual females, other than the lack of sex in their lives. But this is not always true.

First, newly **parthenogenetic** females usually do not have a reproductive system as efficient as that of sexual females. This is not because asexual females necessarily must have inefficient reproduction. Instead, the evolution of any new structure or function is likely to be inefficient at first. Evolution usually takes many generations to shape adaptations so that they are highly efficient. It takes time for mutations that enhance adaptations to occur, and then each of these mutations takes some time to increase in frequency.

For example, recently evolved types of "flight," such as flight in gliding squirrels, are very awkward compared to flight in animals that have had it for millions of years, like bats and birds. Likewise, a newly asexual female usually has much reduced fertility. For example, when new asexual lineages of *Drosophila* species are isolated in the laboratory, they usually have fertility that is only a few percent of the normal sexual level. When this occurs, asexual forms will be selected against relative to sexual variants.

Second, newly asexual females face a particularly pertinent hazard that comes from the environment—males. Sexual males will continue to be attracted to asexual females, before these females have evolved distinctive morphology and behavior. Therefore, sexual males will attempt to mate with asexual females. If these females resist, they may be damaged. If they do not resist, then they face the problem of fertilization. As shown in Figure 18.10A, fertilization can have various consequences. One possibility is that the asexual female's egg may respond to the fertilization event and initiate meiosis, so that fertilization can proceed normally. In this event, asexual reproduction is terminated. This result occurs normally in new asexual females of *Drosophila*.

Another possibility is that the asexual female's diploid egg is fertilized by haploid sperm, producing triploid offspring. In most cases, triploid offspring will not be viable. And if they are viable, then they will have reduced fertility. Finally, triploid offspring may be viable, but reproductively isolated. This result can lead to a new triploid species (see the box, "Triploid Asexual Species Reveal the Struggles of Parthenogens").

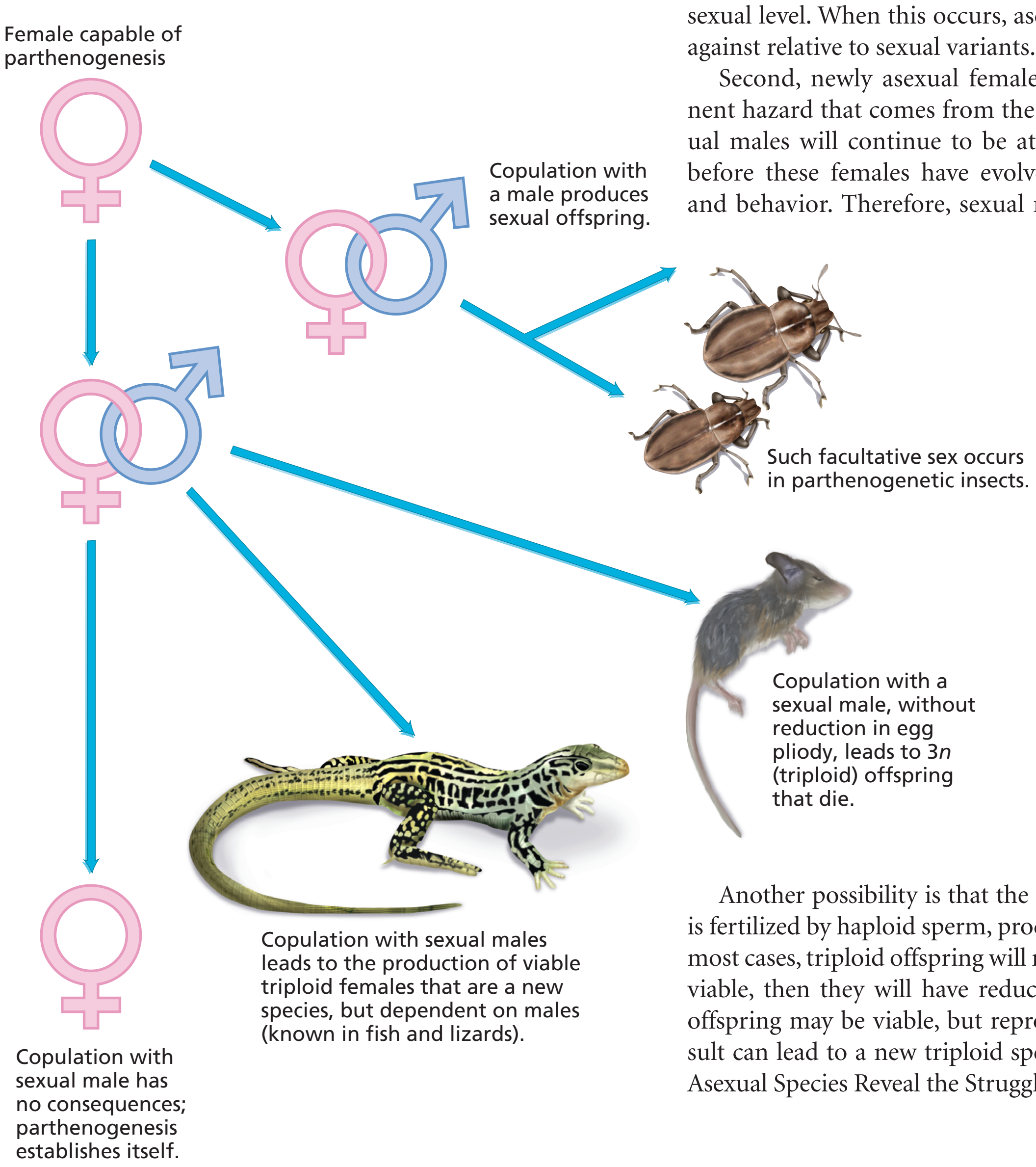

FIGURE 18.10A It may be hard for parthenogenetic females to escape from males. The problem is that females with the capacity to reproduce parthenogenetically often have sex anyway, resulting in the production of offspring that may be sexual, dead, or triploid. This prevents the establishment of parthenogenesis.

Triploid Asexual Species Reveal the Struggles of Parthenogens

Triploid asexual species have evolved a few times, suggesting that they originated from the sexual fertilization of asexual females that have diploid eggs. There are even vertebrate examples. The whiptail lizards of the genus *Cnemidophorus* (Figure 18.1B) have triploid asexual female races in the desert Southwest of the United States. Fish of the genus *Poeciliopsis* have races of triploid asexuals derived by hybridization of two sexual species, *P. lucida* and *P. monacha* (Figure 18.1C). Salamanders of the genus *Ambystoma* are triploid hybrids of two sexual species, *A. laterale* and *A. jeffersonianum*. See Module 6.10 for more on *Poeciliopsis* and *Ambystoma*.

On the other hand, it should not be assumed that these asexual triploids evolve very often. There are no known triploid asexual mammalian species, for example. What these rare examples show is that males continue trying to fertilize females that are evolving asexual methods of reproduction, possibly even with the cooperation of the females, who will not know that they have the evolutionary opportunity to escape from males. Only human females would know that.

In all these cases, the evolutionary dynamic is not one of "fair" competition between sexual and asexual females. There is nothing fair about sex. The difficulty of making the transition to asexuality, particularly in the potentially "hostile" environment of males, may prevent the females' escape from sex altogether. In this case, sex is maintained whether a population would do better without it or not.

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ORIGIN OF SEX

18.11 The origin of sex is even more complicated than its maintenance

It is not obvious that sex is beneficial in general. As we have seen throughout the discussion "Is Sex a Good thing Despite Its Problems?" beginning with Module 18.5, there are specific situations where sex might be selectively favored. But these situations are not universal. So why is sex so common?

One possible answer is that if sex is difficult to escape from, then sex may be evolutionarily "sticky." Once a group of organisms has it, then it may be hard to get rid of—because of males or because of lower fertility among parthenogenetic females. Therefore, sex could be as common as it is because it "sticks" evolutionarily, like tar to a shoe on a hot day.

But this leaves the problem of how groups of organisms evolve sexual reproduction in the first place. Sex is an elaborate reproductive adaptation. It is doubtful that it could be acquired in an accidental manner. Again, this seems to indicate that sex must be a beneficial adaptation produced by natural selection. But this argument is not quite as solid as it first appears.

It is important to compare sex with asexuality in a way that reflects evolutionary history. The evolution of sex does not depend on an abstract or perfect competition of sex with asexuality. Instead, it depends on the evolutionary forces that determine the **origin of sex** and its maintenance. We can think of sex as a giant box for evolution, as shown in Figure 18.11A . Sex originates when a population enters this box. Asexuality evolves from sex when a population leaves the box. Selection maintains sex when evolutionary pressures push populations back into the sex box.

What we have seen to this point is that sex might be maintained either by selection favoring sex or by selection evolutionarily punishing females that try to escape from sex. Given this ambiguity about the maintenance of sex, can we get a better understanding of sex by considering its origin?

In considering the origin of sex, note that there are at least three very different processes involved: syngamy, recombination, and gametogenesis. We will consider each of them in order.

Syngamy, or *fertilization*, is the fusion of genomes to produce a genome with doubled ploidy, as shown in Figure 18.11B. There are advantages to cells fusing and doubling gene number. For example, if either haploid genome has one or more recessive deleterious alleles, then syngamy would rescue the haploid genomes from the full deleterious effects of these alleles. But note that if this is the advantage of syngamy, then it is hard to understand the advantage of later reducing ploidy during gametogenesis.

Recombination occurs when strands of DNA that have similar sequences of nucleotides physically touch each other, unravel their helices, and create new combinations of DNA segments, as shown in Figure 18.11C. While the genome size is doubled, cells have the opportunity to recombine their chromosomes. One puzzle regarding the origin of recombination is that it is likely to be very inefficient in its early evolution, leading to the production of unbalanced combinations of genes. These might be chromosomes that have lost large sections, thanks to improper resolution of the physical crossing of DNA strands that occurs during recombination. Efficient structural resolution of strands of DNA is normal in present-day recombination. But during the evolutionary origins of recombination, it must have been haphazard. Why would cells undergo an initially inefficient process of recombination?

Gametogenesis occurs when sexual cells produce gametes with half the ploidy by some type of reductive division, as shown in Figure 18.11D. The famous instance of gametogenesis is meiosis, but the process can differ from the typical textbook formulation. From an evolutionary standpoint the question is, why would gametogenesis be useful? One scenario is that larger

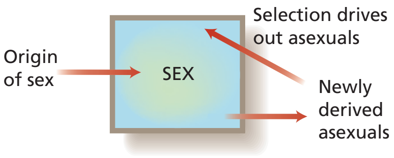


FIGURE 18.11A Sex can be thought of as an evolutionary box. When selection maintains sex, a species evolves into the box. When selection maintains sex, females that abandon sex are selected against by some process, such as sib competition or the effects of males.

genomes are too costly to maintain, compared to the haploid genomes of gametes. But if that is true, why would syngamy evolve in the first place?

In the following modules we will consider some facts of molecular and cell biology, to see if they can help to make sense of the origin—and perhaps the later evolution—of sex. One fundamental point is that in order to spread, sex does not have to benefit the cells that have it. Instead, it may spread as a result of selection for the spread of parasitic DNA sequences. Sex and recombination may also evolve as a result of selection for seemingly unrelated molecular adaptations, the foremost being DNA repair (see Module 18.13).

These additional possibilities for the evolution of sex underscore the likelihood that sex is not a simple evolutionary adaptation. It has been affected by many different selection pressures, not all of them consistent with each other. This makes understanding it something of a nuisance for evolutionary biologists—but still kind of fun. ❖

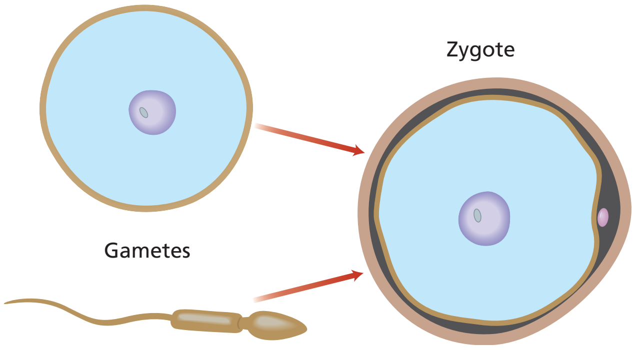


FIGURE 18.11B Syngamy takes place when gametes of two types meet to produce a zygote. Usually these gametes are an egg and a sperm.

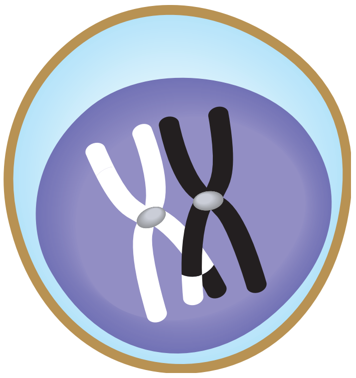


FIGURE 18.11C Recombination occurs when one chromosome swaps genetic material with another chromosome.

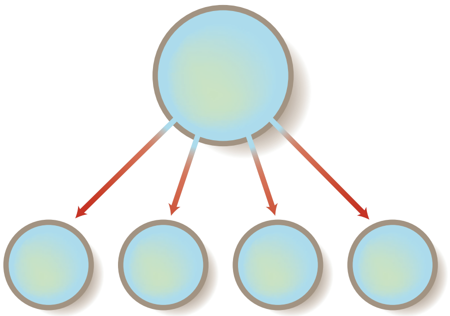


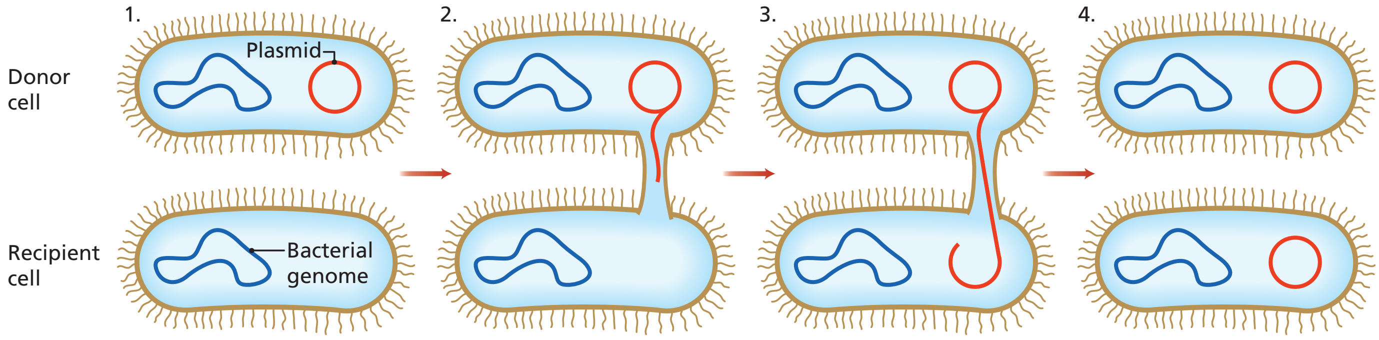
FIGURE 18.11D Reductive division produces gametes, which are usually haploid.

18.12 Simple forms of sex can originate from mobile genetic elements

At least one scenario for the origin of sex has no qualitatively difficult problems. This scenario supplies an origin that does not depend on sex being generally beneficial. Yet it does not preclude the possibility that sex is intermittently beneficial.

The general idea is as follows. We know that bacteria often have closed loops of DNA called **plasmids**. Plasmids replicate independently of the host genome. Sometimes plasmids encode the formation of *pili*, long bridging structures that enable plasmids to pass from one cell to another—as shown in Figure 18.12A, part (i)—by a process called **conjugation**. Plasmids are sometimes beneficial to their bacterial hosts. For example, some plasmids encode genes for resisting antibiotics such as ampicillin. The evolution of resistance to antibiotics in bacteria has come about in part from the spread of plasmids. But other plasmids spread despite measurably deleterious effects. An interesting thing about plasmids is that they can spread whether they are beneficial or not, because they establish a primitive form of genetic exchange that could be considered proto-sexual. Such genetic exchange does not necessarily result in chromosomal recombination, but it does mix plasmid-borne genes between cells.

(i) Bacterial conjugation



(ii) Transient cell fusion

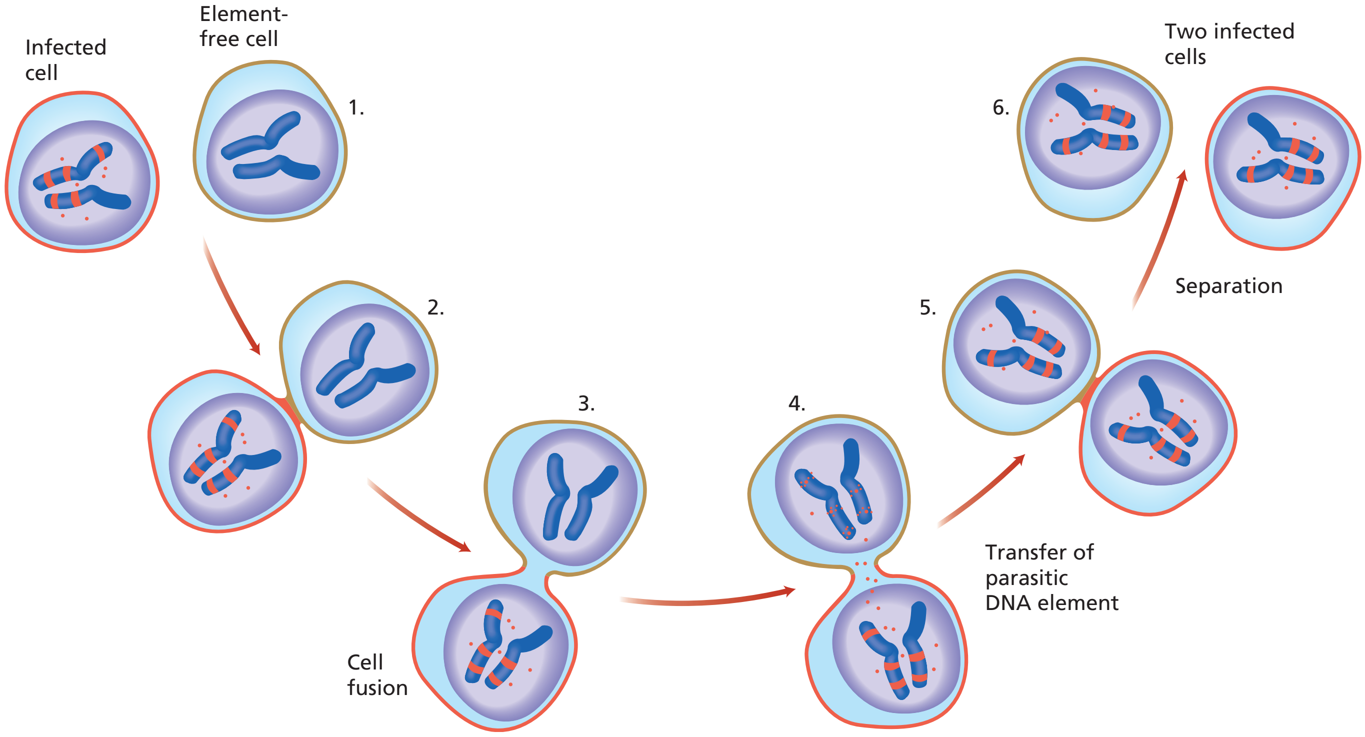


FIGURE 18.12A Primitive Transfer of Genes Plasmids are transmitted from bacterium to bacterium by a process known as conjugation. Conjugation is coded for by plasmids. **(ii)** Parasitic DNAs that float in the cell *and* foster transient cell fusion can foster their own transmission. They might also establish primitive sex.

Eukaryotic cells do not have conjugative plasmids like those in bacteria. But a somewhat similar process might have been involved in the origin of eukaryotic sex. Consider the cell fusion scenario in Figure 18.12A, part (ii). Suppose we have a mobile genetic element that can replicate in the host eukaryotic genome. If this mobile element contains a gene that alters the cell membrane so that cells are more likely to fuse on contact, and this element then "infects" any cells that undergo transient fusion with a carrier of the element, then it will transmit itself through a population of cells by such transient cell fusion. In this process, there is a substantial advantage to such elements from transient **cell fusion**. The transient cell fusion scenario easily explains both proto-syngamy and proto-gametogenesis, in that its spread requires a *cycle* of cell fusion and disconnection.

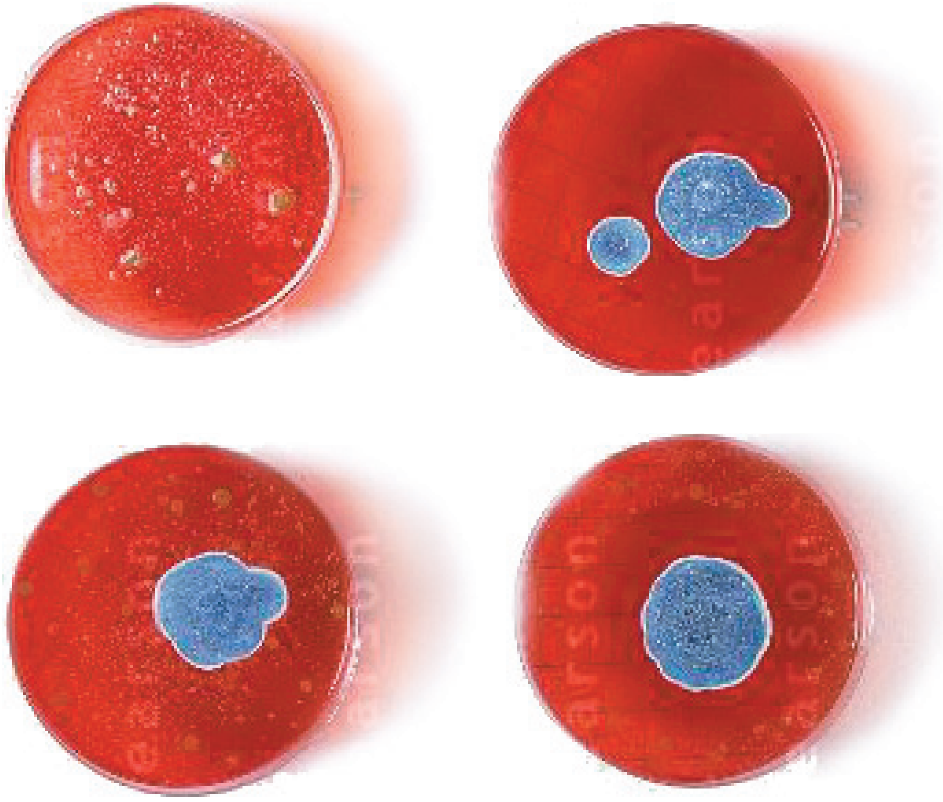
In addition, this type of mobile genetic element can spread through a population without being beneficial. It can do so as long as its deleterious effects are not too great relative to the probability that the element will soon infect a new cell host. In this model, sex spreads because it is contagious, not because it is beneficial. However, beneficial effects are not precluded, like those of antibiotic-resistant genes carried on conjugative plasmids.

This model for the origin of sex shows that sex does not have to be generally beneficial to spread. Sex might have been a by-product of a mobile genetic element, evolving only for the benefit of the element, but then retained because males or low asexual fertility made it difficult for females to escape sex evolutionarily. Or sex could have always been generally beneficial for some reason that is not now known.

Two More Forms of Sex

We have already considered two basically different forms of genetic exchange—conjugation and eukaryotic sex. There are two other forms of genetic exchange in the living world. The first is **transformation**, the genetic exchange that occurs when bacteria absorb the DNA secreted by other living cells or by dead cells, and then recombine it into their genome. It is not clear whether transformation is beneficial. One possibility is that the absorbed DNA is just a metabolically cheaper source of nucleotides. In other words, transformation could be DNA cannibalism.

The second additional type of genetic exchange is **transduction**, the genetic exchange that occurs when viral infection of bacteria produces viral capsules that bear host DNA and that then find their way to other bacterial cells. Transduction may be entirely accidental. It is not as well known as the other forms of genetic exchange.



18.13 Recombination may have evolved as a by-product of selection for DNA repair

A preeminent fact about the evolution of life is that DNA has to be repaired . Other types of molecules, such as protein, can be discarded when damaged. But DNA is the information repository of the organism, and it has to be repaired if at all possible. Thus all organisms have **DNA repair** machinery, the details of which are part of the study of molecular biology.

Recombination may not really be part of the evolution of sex, as such.

One form of DNA damage is potentially grievous—double-strand breaks. Such breaks completely interrupt the DNA sequence of a chromosome. In diploid eukaryotic cells, however, double-strand breaks can be repaired using the DNA template provided by the homologous DNA sequence on the matching homologous chromosome. A side effect of such double-stranded break repair is that the resolution of the repair process can lead to recombination, in which the sequence from one chromosome is swapped with the sequence of the other chromosome, as shown in Figure 18.13A.

This side effect raises the important possibility that recombination is not itself favored by natural selection. Instead, it might be an incidental by-product of a process that must be favored very strongly—DNA repair. This model is somewhat controversial, and the details of DNA repair do not always fit this theory. For example, male *Drosophila* do not undergo recombination, yet their DNA repair processes seem to work just fine. The DNA repair theory of recombination supplies a completely different perspective on the evolution of recombination. Recombination may not really be part of the evolution of sex, as such. It may instead be a by-product of the evolution of DNA repair.

To put this discussion in perspective, consider that evolutionary geneticists often construct theoretical models in which selection favors, or opposes, recombination. Often these models depend on such ecological patterns as the correlations between different environmental features (e.g., temperature, humidity, etc.), environmental variation through time, and so on. Under some conditions, these models predict the evolution of frequent recombination. Under other conditions, they don't.

Yet recombination, while not quite universal, is one of the most widespread features of genetic systems. How could something so close to universal depend on ecological particulars? The generation of recombination by DNA repair seems to be a more appropriate solution to the occurrence of recombination, because DNA repair is a nearly universal molecular process, vital to the continuation of life. The DNA repair theory for recombination may not be correct. Cells could be doing something else that leads to the evolution of recombination. But this theory does seem to be at the right level of generality.

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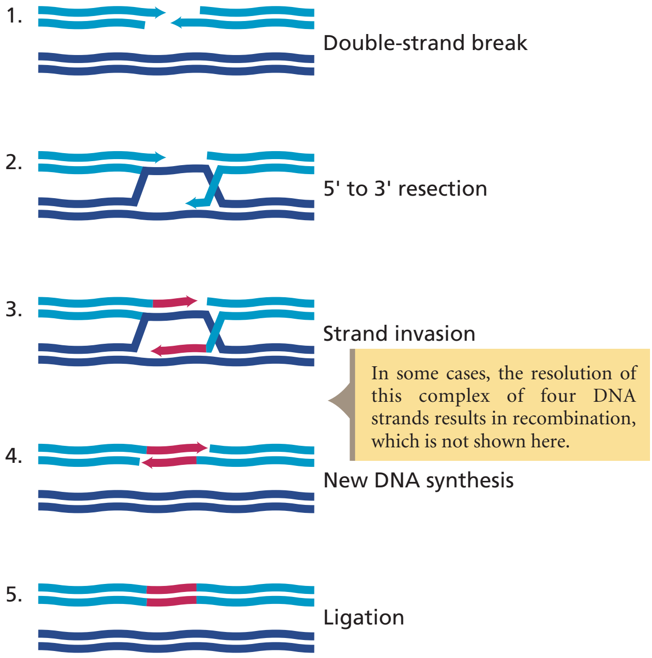

In some cases, the resolution of this complex of four DNA strands results in recombination, which is not shown here.

FIGURE 18.13A Double-strand break repair is a process that maintains the integrity of DNA. At the same time, under some conditions, it may foster recombination.

Resolving Contradictions in the Evolution of Sex

Now that we have gone through the range of theories about the evolution of sex, one obvious pattern is that many aspects of biology have been connected to the evolution of sex: ecological advantage, accumulation of mutations, mobile genetic elements, destructive males, and DNA repair. Still, it may not be too early to come to some general conclusions .

First, it is doubtful that sex evolved and is maintained because of a single universal evolutionary mechanism. Second, the evolution of sex has probably been driven by multiple evolutionary mechanisms acting at different levels—molecular, organismal, and population. It would be convenient if the entire phenomenon of sex could be explained by just one hypothesis, but that now seems very unlikely .

SUMMARY

1. Sex is the most challenging mystery in biological evolution. At the core of this mystery is the evolutionary advantage that females would derive from reproducing without a male genetic contribution. Such "parthenogenetic" reproduction would free females from a 50 percent dilution of their genes. Sex also breaks up successful genotypes, which the genotypes of all parents must be. There are other problems with sex, problems less related to genetics. The growth and operation of sex organs imposes a physiological burden, as does mating behavior in animals. Sex exposes organisms to greater risks of predation and disease, especially diseases spread during fertilization.

2. There are many adaptive theories of sex, proposing that sexual organisms have an advantage over asexual organisms under certain environmental conditions. Most of these advantages turn out to be dependent on specific ecological conditions that are unlikely to be universal, or even common. When populations are large, but not too large, and beneficial mutations common, but not too common, sex can accelerate evolution. When sibs commonly compete against each other in harsh, changing environments, sexual mothers may have more reproductive success. Contagious disease may eliminate all but rare genotypes that only sexual parents can produce.

3. A different kind of theory is based on the problem of deleterious mutations. Under some conditions, asexual lineages go extinct from the ratchet-like accumulation of such mutations. Under other conditions, large asexual populations may endure deleterious mutations better than sexual populations do. It is not clear whether deleterious mutations help explain sex.

4. One way to explain the maintenance of sex involves the difficulties faced by newly asexual females: (a) They have been observed to have low fertility, and (b) fertilization by males may terminate asexual reproduction or produce triploid offspring of low viability.

5. It is difficult to explain the origin of sex using adaptive models. The origin of sex can be explained instead in relation to the advantage that mobile genetic elements would receive from transient, inefficient cell fusion. Recombination might be explicable as an incidental side effect of DNA repair, one of the most important molecular adaptations of the cell, though there are some problems with this theory.

REVIEW QUESTIONS

1. Why don't males evolve asexual reproduction, as females do?
2. Explain how sex can be viewed as a generator of variation to help organisms adapt to a varying environment.
3. Explain how molecular recombination might help fitness in organisms with damaged DNA.
4. Describe the scenarios that might arise if a fertile male found his way to a newly established colony of asexual females.
5. Why is sex most useful if siblings compete against siblings in small ecological patches?
6. Construct a scenario in which sex is useful to humans in our reproduction.
7. Why do bacteria have sex without syngamy, while sex in plants and animals involves syngamy?
8. Which ecologies favor the evolution of sex, and which favor the evolution of asexuality?
9. Over the entire period of evolution, do you think that sex has generally increased fitness or decreased it?

KEY TERMS

accelerated evolution
asexual reproduction
Bell, Graham
cell fusion
chlamydia
conjugation
Cnemidophorous
DNA repair
external fertilization
gametogenesis
genital herpes
genital warts
gonorrhea
historical imperfections
internal fertilization
lottery model
Muller's ratchet
origin of sex
parthenogenesis
pathogen-host arms race
plasmids
Poeciliopsis
recombination
reductive division
rotifer, bdelloid
sexual reproduction
sequential substitution
sib competition
syngamy
syphilis
transduction
transformation
triploid asexuals
twofold cost of males
venereal disease
zygote

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