18 MICROEVOLUTIONARY PROCESSES

Rise of the Super Rats

Slipping in and out of the pages of human history are rats—*Rattus*—the most notorious of mammalian pests. One kind of rat or another has distributed pathogens and parasites that cause bubonic plague, typhus, and other deadly infectious diseases (Figure 18.1). The death toll from fleas that bit infected rats and then bit people has exceeded the dying in all wars combined.

The rats themselves are far more successful. By one estimate, there is one rat for every person in urban and suburban centers of the United States. Besides spreading diseases, rats chew their way through walls and wires of homes and cities. In any given year, they cause economic losses approaching 19 billion dollars.

For years, people have been fighting back with traps, ratproof storage facilities, and poisons, including arsenic

and cyanide. During the 1950s, they used baits laced with warfarin. This synthetic organic compound interferes with blood clotting. Rats ate the baits. They died within days after bleeding internally or losing blood through cuts or scrapes.

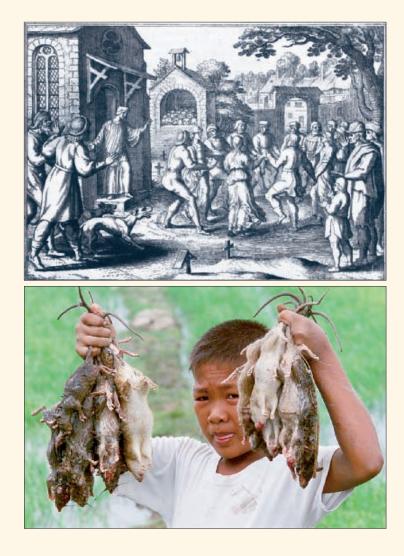
Warfarin was extremely effective. Compared to other rat poisons, it had a lot less impact on harmless species. It quickly became the rodenticide of choice.

In 1958, however, a Scottish researcher reported that warfarin did not work against some rats. Similar reports from other European countries followed. About twenty years later, 10 percent of the urban rats caught in the United States were warfarin resistant. *What happened*? To find out, researchers compared warfarin-resistant rat populations with still-vulnerable rats. They traced the difference to a gene on one of the rat chromosomes.

Figure 18.1 *Above*, medieval attempts to deal with a bubonic plague pandemic—the Black Death—that may have killed half the people in Europe alone. Not knowing that the disease agent hitches rides on rats, Europeans tried to protect themselves by praying and dancing until they dropped. Physicians wore bird masks, such as the mask shown on the facing page. They filled the "beak" with herbs that supposedly purified the air that plague victims had breathed. For the next 300 years, anyone accused of causing an outbreak of the plague, no matter how absurd the evidence, was burned alive.

Below, example of rats in this century. Rats infest 80,000 hectares of the rice fields in the Philippine Islands. They ruin more than 20 percent of the annual crops. Rice is the main food source for people in Southeast Asia.

Today we douse agricultural land and buildings with ever more potent rat poisons. By doing so, we have unwittingly promoted the rise of super rats. Three centuries from now, how will people be viewing *our* actions?



IMPACTS, ISSUES

At that gene locus, a dominant allele was common among the warfarin-resistant rat populations but rare among the vulnerable ones. The dominant allele's product actually neutralizes warfarin's effect on blood clotting.

"What happened" was evolution by natural selection. As warfarin started to exert pressure on rat populations, the rat populations changed. The previously rare dominant allele suddenly proved to be adaptive. The lucky rats that inherited the allele survived and produced more offspring. The unlucky ones that inherited the recessive allele had no built-in defense, and they died. Over time, the dominant allele's frequency increased in all rat populations exposed to the poison.

Selection pressures can and often do change. When warfarin resistance increased in rat populations, people stopped using warfarin. Not surprisingly, the frequency of the dominant allele declined. Now the latest worry is the evolution of "super rats," which the newer and even more potent rodenticides cannot seem to kill.

The point is, when you hear someone question whether life evolves, remember this: With respect to life, **evolution** simply means heritable change is occurring in some line of descent. The actual mechanisms

that can bring about such change are the focus of this chapter. Later chapters highlight how these mechanisms have contributed to the evolution of new species.



Watch the video online!

How Would You Vote?

Antibiotic-resistant strains of bacteria are becoming dangerously pervasive. Standard animal husbandry practice includes continually dosing healthy animals with antibiotics—the same antibiotics prescribed for people. Should this practice stop? See BiologyNow for details, then vote online.

Key Concepts

WHAT IS MICROEVOLUTION?

Individuals of all natural populations share a gene pool but differ in which alleles they inherit. As a result, they show variations in phenotypes.

An individual does not evolve. Rather, a *population* evolves, which means its shared pool of alleles is changing. Over the generations, any allele may increase in its frequency among individuals, or it may become rare or lost.

Microevolution refers to changes in allele frequencies as an outcome of mutation, natural selection, genetic drift, and gene flow. Sections 18.1, 18.2

NATURAL SELECTION

Natural selection is the outcome of variation in heritable traits that influence which individuals of a population survive and reproduce in each generation. Selective agents operating in the environment can stabilize, disrupt, or cause directional shifts in the range of variation. Sections 18.3–18.6

GENETIC DRIFT

Sometimes chance events bring about random changes in allele frequencies over time. The magnitude of this genetic drift is greatest in small populations, where it can lead to a loss of genetic diversity. Section 18.7

GENE FLOW

Gene flow is the physical movement of alleles into and out of a population. It tends to oppose the effects of mutation, natural selection, and genetic drift; it keeps populations of a species similar to one another. Section 18.8

ADAPTATION AND THE ENVIRONMENT

An evolutionary adaptation is a heritable aspect of form, function, behavior, or development that contributes to the fit between an individual and its environment. The challenge is to identify environmental conditions to which a given trait is presumably adapted. Section 18.9



Before starting this chapter, review the premises of the theory of natural selection as outlined in Sections 1.4 and 17.3 as well as the definitions of basic terms in genetics (11.1).

You will be drawing upon your knowledge of mutation (14.5) and the chromosomal basis of inheritance (12.5 especially). We urge you to scan earlier sections on causes of continuous variation in populations (11.5) and on how the environment can modify gene expression (11.6).

18.1

WHAT IS MICROEVOLUTION?

LINKS TO SECTIONS 11.4, 11.6, 11.7, 14.5, 17.9

As Charles Darwin and Alfred Wallace perceived long ago, individuals don't evolve; populations do. Each **population** is a group of individuals of the same species in a specified area. To understand how it evolves, start with variation in the traits that characterize it.

Individuals Don't Evolve, Populations Do

VARIATION IN POPULATIONS

The individuals of a population share certain features. Pigeons have two feathered wings, three toes forward, one toe back, and so on. These are *morphological* traits (*morpho*–, form). The individuals share *physiological* traits, including metabolic activities that help the body function in the environment. They respond the same way to certain basic stimuli, as when babies imitate adult facial expressions. These are *behavioral* traits.

However, the individuals of a population also show variation in the details of the shared traits. You know this just by thinking about the variations in the color and patterning of pigeon feathers or butterfly wings or snail shells. Figure 18.2 only hints at the range of variations in human skin color and distribution, color, texture, and amount of hair. Almost every trait of any species may vary, but variation can be dramatic among sexual reproducers.

For sexually reproducing species, at least, we may define the population as a group of individuals that are interbreeding, that are reproductively isolated from other species, and that produce fertile offspring. The offspring typically have two parents, and they have mixes of the parental forms of traits.

Many traits show *qualitative* differences; they have two or more distinct forms, or morphs. Remember the purple or white pea plant flowers that Gregor Mendel studied? The persistence of two forms of a trait in a population is a case of **dimorphism**. The persistence of three or more forms is **polymorphism**. In addition, for many traits, the individuals of a population show *quantitative* differences, a range of incrementally small variations in a specified trait (Section 11.7).

THE GENE POOL

Genes encode information about heritable traits. The individuals of a population inherit the same number and kind of genes (except for a pair of nonidentical sex chromosomes). Together, they and their offspring represent a **gene pool**—a pool of genetic resources.

For sexual reproducers, nearly all genes available in the shared pool have two or more slightly different molecular forms, or **alleles**. Any individual might or might not inherit identical alleles for any trait. This is the source of variations in *phenotype*, or differences in the details of shared traits. Whether you have black, brown, red, or blond hair depends upon which alleles you inherited from your two parents.

You read about the inheritance of alleles in earlier chapters. Here we summarize the key events involved:

Gene mutation

Crossing over at meiosis I (puts novel combinations of alleles in chromosomes)

Independent assortment at meiosis I (puts mixes of maternal and paternal chromosomes in gametes)

Fertilization (combines alleles from two parents)

Change in chromosome number or structure (loss, duplication, or repositioning of genes)

Only mutation creates new alleles. The other events shuffle existing alleles into different combinations, but what a shuffle! Each gamete gets one of many millions of possible combinations of maternal and paternal chromosomes that may or may not be identical at each locus. Unless you are an identical twin, it is extremely unlikely that another person with your precise genetic makeup has ever lived or ever will.

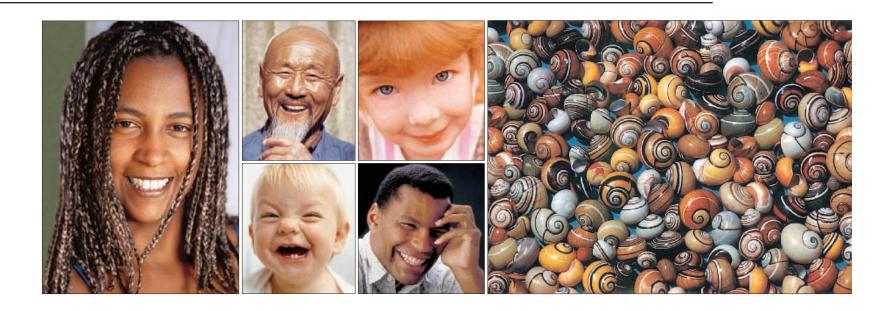
One other point about the nature of the gene pool: Offspring do not inherit phenotypes; they inherit *genes*. Section 11.6 describes how environmental conditions, too, bring about variation in the range of phenotypes, but the effects last no longer than the individual.

MUTATION REVISITED

Being the original source of new alleles, mutations are worth another look—this time in the context of their impact on populations. Usually, gene mutations that have beneficial or neutral effects are transmitted to a new generation. We cannot predict precisely when or in which individual a particular gene will mutate. We *can* predict rates of mutation, or the probability that a mutation will happen in a specified interval (Section 14.5). For instance, one estimated rate for mammalian genomes is 2.2^{-9} mutations per base pair per year.

Many mutations give rise to structural, functional, or behavioral alterations that reduce an individual's chances of surviving and reproducing. Even a single biochemical change may be devastating. For instance, skin, bones, tendons, lungs, blood vessels, and many other vertebrate organs incorporate collagen. Thus, when the collagen gene has mutated, drastic problems may ripple all through the body. Compare Section 11.4.

Any mutation that results in severe disruptions in phenotype usually causes death. It is a **lethal mutation**.



A **neutral mutation**, recall, alters the base sequence in DNA, but the change has no discernible effect on survival or reproduction (Section 17.9). It neither helps nor hurts the individual. For instance, if you carry a mutant gene that keeps your earlobes attached to the head instead of swinging freely, this in itself should not stop you from surviving and reproducing as well as anybody else. Therefore, natural selection does not affect the frequency of the trait in the population.

Every so often, a mutation proves useful. A mutant gene product that affects growth might make a corn plant grow larger or faster and thereby give it the best access to sunlight and nutrients. A neutral mutation might prove helpful if conditions in the environment change. Even if a mutant gene bestows only a slight advantage, natural selection or a chance event might favor its preservation in DNA and its transmission to the next generation.

Mutations are rare, so they usually have little or no immediate effect on a population's allele frequencies. But they have been slipping into genomes for billions of years. Cumulatively, they have served as reservoirs for change, for biodiversity that is staggering in its breadth. Think of it. The reason you don't look like a bacterium or an avocado or earthworm or even your neighbors down the street began with mutations that arose at different times, in different lines of descent.

STABILITY AND CHANGE IN ALLELE FREQUENCIES

Researchers typically track **allele frequencies**, or the relative abundances of alleles of a given gene among all individuals of a population. They can start from a theoretical reference point, **genetic equilibrium**, when a population is *not* evolving with respect to that locus.

Genetic equilibrium can only occur if five conditions are being met: There is no mutation, the population is infinitely large, the population is isolated from other populations of the same species, individuals mate at random, and all individuals survive and produce the same number of offspring.

Figure 18.2

A sampling of the phenotypic variation in populations of humans and snails, the outcome of variations in frequencies of alleles.

If you are interested, the following section offers a closer look at the nature of genetic equilibrium—the point at which a population is not evolving.

As it happens, genetic equilibrium is exceedingly rare in nature. Why? Mutations are rare but inevitable, and they might throw a wild card in the game of who survives and reproduces. Also, three processes—called *natural selection, genetic drift,* and *gene flow*—can drive populations out of equilibrium. **Microevolution** refers to small-scale changes in allele frequencies that arise as an outcome of mutation, natural selection, genetic drift or gene flow, or some combination of these.

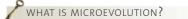
We partly characterize a natural population or species by morphological, physiological, and behavioral traits, most of which are heritable.

At any gene locus, different alleles give rise to variations in individual phenotypes—to differences in the details of shared structural, functional, and behavioral traits.

The individuals of a population share a pool of genetic resources—that is, a pool of alleles.

Only mutation creates new alleles. Natural selection, genetic drift, and gene flow affect only the frequencies of various alleles at a given gene locus in the population.

Most populations are slowly evolving, which simply means that the frequencies of the alleles for a specified trait are changing from one generation to the next.



8.2 When Is A Population *Not* Evolving?

How do researchers know whether or not a population is evolving? They can start by tracking deviations from the baseline of genetic equilibrium.

The Hardy–Weinberg Formula Early in the twentieth century, Godfrey Hardy (a mathematician) and Wilhelm Weinberg (a physician) independently applied the rules of probability to sexually reproducing populations. Like the geneticists who came after them, they perceived that gene pools can remain stable only when five conditions are being met:

- 1. There is no mutation.
- 2. The population is infinitely large.
- 3. The population is isolated from all other populations of the species (no gene flow).
- 4. Mating is random.
- 5. All individuals survive and produce the same number of offspring.





490 AA butterflies dark-blue wings



420 Aa butterflies medium-blue wings



90 *aa* butterflies white wings

Starting Population





420 Aa butterflies medium-blue wings



90 *aa* butterflies white wings



In other words, allele frequencies for any gene in the shared pool will remain stable unless the population is evolving. Hardy and Weinberg developed a simple formula that can be used to track whether a population of any sexually reproducing species is slipping out of that state of genetic equilibrium.

Consider tracking a hypothetical pair of alleles that affect butterfly wing color. A protein pigment is specified by dominant allele A. If a butterfly inherits two AA alleles, it will have dark-blue wings. If it inherits two recessive alleles *aa*, it will have white wings. If it inherits one of each (*Aa*), the wings will be medium-blue (Figure 18.3).

At genetic equilibrium, the proportions of the wingcolor genotypes are

$$p^{2}(AA) + 2pq(Aa) + q^{2}(aa) = 1.0$$

where *p* and *q* are the frequencies of alleles *A* and *a*. This is what became known as the *Hardy–Weinberg equilibrium equation*. It defines the frequency of a dominant and a recessive allele for a gene that controls a particular trait in a population.

The frequencies of *A* and *a* must add up to 1.0. To give a specific example, if *A* occupies half of all the loci for this gene in the population, then *a* must occupy the other half (0.5 + 0.5 = 1.0). If *A* occupies 90 percent of all the loci, then *a* must occupy 10 percent (0.9 + 0.1 = 1.0). No matter what the proportions,

p + q = 1.0

At meiosis, recall, paired alleles segregate and end up in different gametes. So the proportion of gametes having the *A* allele is *p*. The proportion having the *a* allele is *q*. The Punnett square on the next page reveals the genotypes possible in the next generation (*AA*, *Aa*, and *aa*).



490 AA butterflies dark-blue wings



420 Aa butterflies medium-blue wings



90 *aa* butterflies white wings

Next Generation

Figure 18.3 *Animated!* How to determine whether a population is evolving. The frequencies of wingcolor alleles among all individuals in this hypothetical population of morpho butterflies have not changed because all five assumptions upon which the Hardy–Weinberg rule is based are being met. FOCUS ON SCIENCE



The frequencies add up to 1.0: $p^2 + 2pq + q^2 = 1.0$. Suppose that the population has 1,000 individuals and that each one produces two gametes:

490 AA individuals make 980 A gametes420 Aa individuals make 420 A and 420 a gametes90 aa individuals make 180 a gametes

The frequency of alleles A and a among 2,000 gametes is

$$A = \frac{980 + 420}{2,000 \text{ alleles}} = \frac{1,400}{2,000} = 0.7 = p$$

$$a = \frac{180 + 420}{2,000} = \frac{600}{2,000} = 0.2 = a$$

2,000

2,000 alleles

At fertilization, gametes combine at random and start a new generation. If the population size is still 1,000, you will find 490 AA, 420 Aa, and 90 aa individuals. Because the allele frequencies for dark-blue, medium-blue, and white wings are the same as they were in the original gametes, they will give rise to the same phenotypic frequencies that occurred in the preceding generation.

As long as the assumptions that Hardy and Weinberg identified continue to hold, the pattern will persist. If traits show up in different proportions from one generation to the next, however, then one or more of the five assumptions is not being met. The hunt can begin for one or more of the evolutionary forces driving the change.

Applying the Rule So how does the Hardy–Weinberg formula work in the real world? For one thing, researchers use it to estimate the frequency of carriers of alleles that cause genetic traits and disorders.

For example, about 1 percent of people of Irish ancestry are affected by *hemochromatosis*. They absorb too much iron from their food. Symptoms of this autosomal recessive disorder include liver problems, fatigue, and arthritis. We can use the number to estimate the frequency of carriers of the recessive allele. If $p^2 = 0.01$, then p is 0.1, q is 0.9, and the carrier frequency (2pq) must be 0.18 among Irish populations. Such information is useful to doctors and public health professionals.

Another example: A deviation from the frequencies predicted by the Hardy–Weinberg formula suggests that a mutant allele for *BRCA2* may be lethal to female embryos. The allele also has been linked to breast cancer. For one study, researchers tracked the frequency of the mutant allele among newborn girls. There were fewer homozygotes than expected, based on the number of heterozygotes and the Hardy–Weinberg formula. By itself or in combination with other alleles, a pair of mutant *BRCA2* alleles may cause the spontaneous abortion of the early embryo.

NATURAL SELECTION

18.3 Natural Selection Revisited

Natural selection, again, is the outcome of differences in reproduction among individuals of a population that vary in their shared traits, some of which prove more adaptive than others under prevailing environmental conditions.

Natural selection may be the most influential process of microevolution. Its impact shows up at all levels of biological organization, which is the reason you were introduced to it early on, in Chapter 1. You also came across simple examples in other chapters, and Sections 17.2 and 17.3 offered you a glimpse of the history that preceded its discovery. Turn now to major categories of selection, as sketched out in Figure 18.4.

With *directional* selection, the range of variation for a trait shifts in a consistent direction; individuals at one end of the range of variation are selected against and those at the other end are favored. With *stabilizing* selection, the forms at one or both ends of the range are selected against. With *disruptive* selection, forms at one or both ends are favored and intermediate forms are selected against.

Diverse selection pressures acting on a population might favor forms at one end in the range of variation for a trait, or intermediate forms within that range, or extreme forms at both ends of the range.

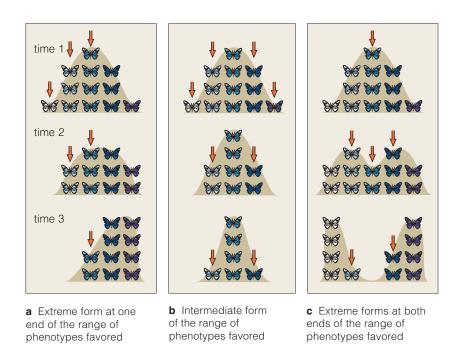


Figure 18.4 Overview of the outcomes of three modes of natural selection: (**a**) directional, (**b**) stabilizing, and (**c**) disruptive.



NATURAL SELECTION

LINKS TO SECTIONS

1.4, 16.7

18.4 Directional Selection

With **directional selection**, allele frequencies shift in a consistent direction, so forms at one end of a phenotypic range become more common than midrange forms, as in Figure 18.5. Directional change in the environment or novel conditions can cause the shift.

RESPONSES TO PREDATION

The Peppered Moth Populations of peppered moths (*Biston betularia*) offer us a classic case of directional selection. The moths feed and mate at night and rest motionless on trees during the day. Their behavior and coloration (mottled gray to nearly black) camouflage them from day-flying, moth-eating birds.

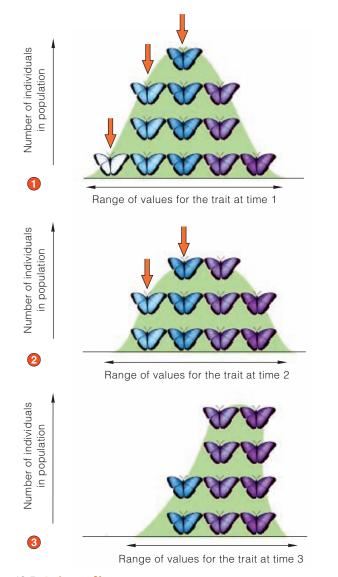


Figure 18.5 *Animated!* Directional selection. These bell-shaped curves signify a range of continuous variation in a butterfly wing-color trait. *Mediumblue* is between two phenotypic extremes—*white* and *dark purple. Orange* arrows signify which forms are being selected against over time.

In the 1850s, the industrial revolution started in England, and factory smoke altered conditions in much of the countryside. Before then, light moths were the most common form, and a dark form was rare. Also, light-gray speckled lichens had grown thickly on tree trunks. Light moths but not dark moths that rested on the lichens were camouflaged (Figure 18.6*a*).

Lichens are sensitive to air pollution. Between 1848 and 1898, soot and other pollutants started to kill the lichens and darken tree trunks. The dark moth form was better camouflaged (Figure 18.6*b*). Researchers hypothesized: If the original conditions favored light moths, then the *changed* conditions favored dark ones.

In the 1950s, H. B. Kettlewell used a *mark–release– recapture method* to test the possibility. He bred both moth forms in captivity and marked hundreds so that they could be easily identified after being released in the wild. He released them near highly industrialized areas around Birmingham and near an unpolluted part of Dorset. His team recaptured more dark moths in the polluted area and more light ones near Dorset:

	Near Birmingham (pollution high)		Near Dorset (pollution low)	
Light-Gray Moths				
Released	64		393	
Recaptured	16	(25%)	54	(13.7%)
Dark-Gray Moths				
Released	154		406	
Recaptured	82	(53%)	19	(4.7%)

Observers also hid in blinds near moths that had been tethered to trees. They observed birds capturing more light moths around Birmingham and more dark ones around Dorset. Directional selection was in play.

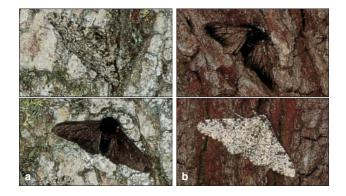


Figure 18.6 Natural selection of two forms of the same trait, body surface coloration, in two settings. (a) Light moths (*Biston betularia*) on a nonsooty tree trunk are hidden from predators. Dark ones stand out. (b) The dark color is more adaptive in places where soot darkens tree trunks.

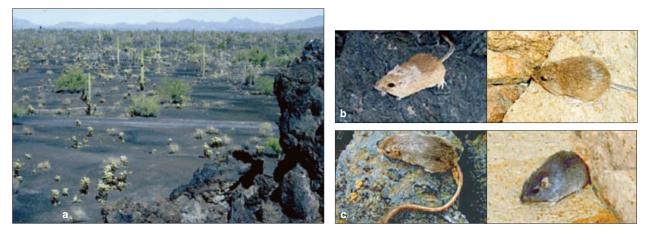


Figure 18.7 Visible evidence of directional selection in a population of rock pocket mice relative to a neighboring population, as documented by Michael Nachman, Hoi Hoekstra, and Susan D'Agostino. (a) Lava basalt flow at the study site. The two color morphs of rock pocket mice, each posed on two different backgrounds: (b) tawny fur and (c) dark fur.

Pollution controls went into effect in 1952. Lichens made a comeback, and tree trunks became largely free from soot. Phenotypes shifted in the reverse direction. Where pollution has decreased, the frequency of dark moths has been decreasing as well.

Pocket Mice Directional selection is at work among rock pocket mice (*Chaetodipus intermedius*) of Arizona's Sonoran Desert. Of more than eighty genes known to affect coat color in mice, researchers found a gene that governs a difference between two populations of this mouse species (Figure 18.7).

Rock pocket mice are small mammals that spend the day in underground burrows and forage for seeds at night. Some live in tawny-colored outcroppings of granite. In this habitat, individuals with tawny fur are camouflaged from predators (Figure 18.7*b*).

A smaller population of pocket mice lives in the same region, but these mice scamper over dark basalt of ancient lava flows. They have dark coats, so they, too, are camouflaged from predators (Figure 18.7*c*).

We can expect that night-flying predatory birds are selective agents that affect fur color. For instance, owls have an easier time seeing mice with fur that does not match the rocks.

Michael Nachman used genetic data on laboratory mice to formulate a hypothesis on differences in coat color in the two wild populations of pocket mice. He predicted that a mutation of either the *Mclr* gene or *agouti* gene could cause the difference. He collected DNA from dark pocket mice at a lava flow and from light mice at adjacent granite outcroppings.

DNA analysis showed that the *Mclr* gene sequence for all dark mice differed by four nucleotides from that of their light-furred neighbors. In the population of dark mice, the allele frequencies had evolved in a consistent direction as a result of selection pressure, so dark fur became more common.

RESISTANCE TO PESTICIDES AND ANTIBIOTICS

Pesticides can cause directional selection, as they did for the super rats. Typically, a heritable aspect of body form, physiology, or behavior helps a few individuals survive the first pesticide doses. As the most resistant ones are favored, resistance becomes more common. About 450 species of pests are now resistant to one or more types of pesticides. Also, some pesticides kill off the natural predators. Freed from natural constraints, resistant populations flourish and inflict more damage. This result of directional selection is *pest resurgence*. Some genetically engineered crop plants resist pests. In time, they too may exert selection pressure.

Antibiotics also can result in directional selection. Certain microbes produce natural antibiotics that can kill bacterial competitors for nutrients. We use natural and synthetic antibiotics to fight pathogenic bacteria. Streptomycins, for example, inhibit protein synthesis in bacterial cells. The penicillins disrupt covalent bonds that hold a bacterial cell wall together.

Yet antibiotics have been overprescribed, often for simple infections that would clear up on their own. Genetic variation in bacterial gene pools allows some cells with certain genotypes to survive as others die. So overuse of antibiotics favors the resistant bacterial populations, which will be harder to eradicate in the millions of people who contract cholera, tuberculosis, and other bacterial diseases each year. Also, healthy farm animals are routinely dosed with antibiotics to prevent infection. Consider: In eggs that look slightly fluorescent green, tetracycline is showing through.

With directional selection, allele frequencies underlying a range of variation tend to shift in a consistent direction in response to some change in the environment. NATURAL SELECTION

18.5 Selection Against Or in Favor of Extreme Phenotypes

LINK TO SECTION 17.3 Consider now two more categories of natural selection. One works against phenotypes at the fringes of a range of variation; the other favors them.

STABILIZING SELECTION

With **stabilizing selection**, intermediate forms of a trait in a population are favored, and extreme forms are not. This mode of selection can counter mutation, genetic drift, and gene flow. It tends to preserve intermediate phenotypes in the population (Figure 18.8*a*). As an example, prospects are not good for human babies who weigh far more or far less than average at birth. Also, pre-term instead of full-term pregnancies increase the risk, as Figure 18.9 indicates.

Newborns weighing less than 5.51 pounds or born before thirty-eight weeks of pregnancy are completed tend to develop high blood pressure, diabetes, and heart disease when they are adults. Researchers now suspect that the mother's blood concentration of a stress hormone, cortisol, is linked to low birth weight and illnesses that develop later in life.

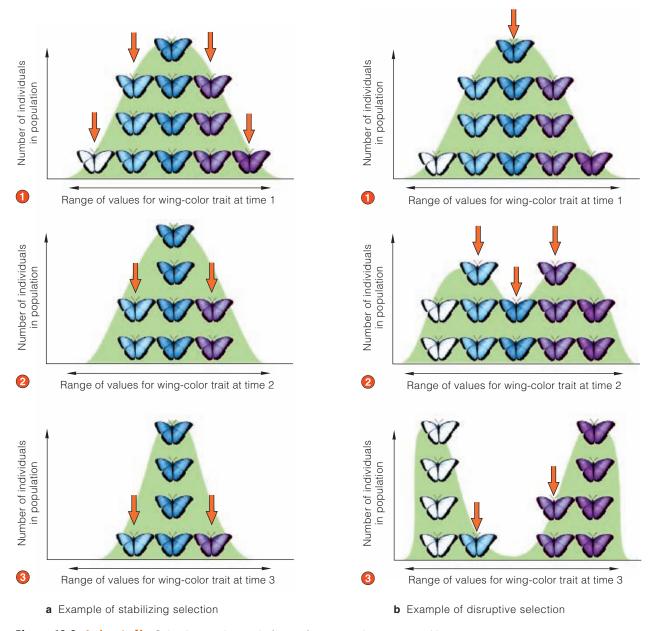


Figure 18.8 *Animated!* Selection against or in favor of extreme phenotypes, with a population of butterflies as the example. (a) stabilizing selection and (b) disruptive selection. The *orange* arrows show forms of the trait being selected against.

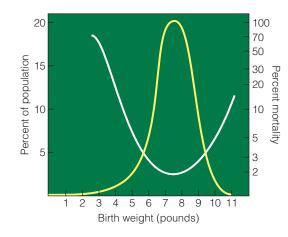


Figure 18.9 Weight distribution for 13,730 human newborns (*yellow* curve) correlated with death rate (*white* curve).

Rita Covas and her colleagues gathered evidence of stabilizing selection on the body mass of juvenile and adult sociable weavers (*Philetairus socius*), as in Figure 18.10. Between 1993 and 2000, they captured, measured, tagged, released, and recaptured 70 to 100 percent of the birds living in communal nests during the breeding season. Their field studies supported a prediction that body mass is a trade-off between risks of starvation and predation. Intermediate-mass birds have the selective advantage. Foraging is not easy in this habitat, and lean birds do not store enough fat to avoid starvation. We can expect that fat ones are more attractive to predators and not as good at escaping.

DISRUPTIVE SELECTION

With **disruptive selection**, forms at both ends of the range of variation are favored and intermediate forms are selected against (Figure 18.8*b*).

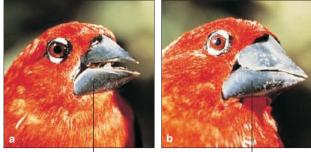
Consider the black-bellied seedcracker (*Pyrenestes* ostrinus) of Cameroon. Females and males of these African finches have large or small bills—but no sizes in between (Figure 18.11). It is like everyone in Texas being four feet or six feet tall, with no one in between.

The pattern holds all through the geographic range. If unrelated to gender or geography, what causes it? If only two bill sizes persist, then disruptive selection may be eliminating birds with intermediate-size bills. Factors that affect feeding performance are the key. Cameroon's swamp forests flood in the wet season; lightning-sparked fires burn in the hot, dry season. Most plants are fire-resistant, grasslike sedges. One species produces hard seeds and the other, soft seeds.

Remember the bills of Galápagos finches (Section 17.3)? Here, also, the ability to crack hard seeds affects survival. All Cameroon seedcrackers prefer soft seeds,



Figure 18.10 Adult sociable weaver (*Philetairus socius*), a native of the African savanna. These birds cooperate in constructing and using large communal nests in a region where trees and other good nesting sites are scarce.



lower bill 12 mm wide

lower bill 15 mm wide

Figure 18.11 Disruptive selection in African finch populations. Selection pressures favor birds with bills that are about 12 *or* 15 millimeters wide. The difference is correlated with competition for scarce food resources during the dry season.

but birds with large bills are better at cracking hard ones. In the dry season, the birds compete fiercely for scarce seeds. A scarcity of both types of seeds during recurring episodes of drought has a disruptive effect on bill size in the seedcracker population. Birds with intermediate sizes are being selected against, and now all bills are either 12 *or* 15 millimeters wide.

In these seedcrackers, bills of a particular size have a genetic basis. In experimental crosses between two birds with the two optimal bill sizes, all offspring had a bill of one size or the other, nothing in between.

With stabilizing selection, intermediate phenotypes are favored and extreme phenotypes at both ends of the range of variation are eliminated.

With disruptive selection, intermediate forms of traits are selected against and extreme forms in the range of variation are favored. NATURAL SELECTION

18.6 Maintaining Variation in a Population

LINKS TO SECTIONS 3.6, 11.1

Natural selection theory helps explain diverse aspects of nature, including male-female differences and the relationship between sickle-cell anemia and malaria.

SEXUAL SELECTION

The individuals of many sexually reproducing species show a distinct male or female phenotype, or sexual dimorphism (dimorphos, having two forms). Often the males are larger and flashier than females. Courtship rituals and male aggression are common.

These adaptations and behaviors seem puzzling. All take energy and time away from an individual's survival activities. Why do they persist if they do not contribute directly to survival? The answer is sexual selection. By this mode of natural selection, winners are the ones that are better at attracting mates and successfully reproducing compared to others of the population. The most adaptive traits help individuals defeat same-sex rivals for mates or are the ones most attractive to the opposite sex.



Figure 18.12 One male bird of paradise in a flashy courtship perhaps, the sexual colorful female. The

the females are drab-colored?)

outcome of sexual selection. This (Paradisaea raggiana) is engaged display. He caught the eye (and, interest) of the smaller, less males of this species compete fiercely for females, which function as selective agents. (Why do you suppose

By choosing mates, a male or female is a selective agent acting on its own species. For example, females of some species shop among a congregation of males, which vary in appearance and courtship behavior. The selected males and the females pass on their alleles to the next generation.

Flashy body parts and behaviors show up among species in which males provide little or no help with raising offspring. The female apparently chooses her partner on the basis of observable signs of health and vigor. Such traits may improve the odds of producing healthy, vigorous offspring (Figure 18.12).

You might be wondering whether we can correlate genes with specific forms of sexual behavior. The sexual deception practiced by an Australian orchid is a case in point. The flowers of *Chiloglottis trapeziformis* attract male wasps by secreting a substance that is identical with a sex pheromone-which female wasps release to attract male wasps. Flowers get pollinated as males attempt to copulate with them.

This orchid is stingy. It gives a male wasp nothing in return, not a single drop of nectar, even though it is the orchid's exclusive pollinator. The female wasps are wingless. They hatch in soil. When males do not lift and carry them to a food source, they starve to death.

When C. trapeziformis puts out blooms, male wasps waste precious time and metabolic energy trying to find females. Evolutionary biologist Florian Schiestl has proposed that selection pressure is afoot for wasps that can produce a new sex pheromone, one that the orchid cannot duplicate.

This interaction exploits male wasps, but Wittko Francke thinks it might put pressure on their brains to evolve. In an orchid patch, the average tiny-brained male wasp copulates blindly with whatever smells right. It will try to copulate even with the head of a pin that has a few micrograms of pheromone sprayed on it. However, a few wasps with a slightly less robotic brain might be able to identify the females by other cues, such as visual ones. Alternatively, both species could face extinction, another pattern in nature.

SICKLE-CELL ANEMIA—LESSER OF TWO EVILS?

With *balancing* selection, two or more alleles of a gene are being maintained at relatively high frequencies in the population. Their persistence is called **balanced** polymorphism (polymorphos, having many forms). The allele frequencies might shift slightly, but often they return to the same values over the long term. We may see this balance when conditions favor heterozygotes. In some way, their nonidentical alleles for a given trait grant them higher fitness compared to homozygotes, which, recall, have identical alleles for the trait.

Consider the environmental pressures that favor an Hb^A/Hb^S pairing in humans. The Hb^S allele codes for a mutant form of hemoglobin, an oxygen-transporting protein in blood. Homozygotes (Hb^S/Hb^S) develop the genetic disorder *sickle-cell anemia* (Section 3.6).

The Hb^S frequency is highest in both tropical and subtropical regions of Asia and Africa. Often, Hb^S/Hb^S homozygotes die in their early teens or early twenties. Yet, in these same regions, heterozygotes (Hb^A/Hb^S) make up nearly a third of the population! Why is this combination maintained at such high frequency?

The balancing act is most pronounced in areas that, historically, have had the highest incidence of *malaria* (Figure 18.13). Mosquitoes transmit the parasitic agent of malaria, *Plasmodium*, to human hosts. The parasite multiplies in the liver and then in red blood cells. The target cells rupture and release new parasites during severe, recurring bouts of infection (Section 22.7).

It turns out that Hb^A/Hb^S heterozygotes are more likely to survive malaria than people who make only normal hemoglobin. Several survival mechanisms are possible. In heterozygotes, the infected cells take on a sickle shape under normal conditions. The abnormal shape marks them as targets for the immune system, which destroys them, along with the parasites inside. In addition, heterozygotes have one functioning Hb^A allele. Although they are not completely healthy, they still produce enough normal hemoglobin to prevent sickle-cell anemia. That is why heterozygotes are more likely to survive long enough to reach reproductive age, compared to Hb^S/Hb^S homozygotes.

In short, the persistence of the "harmful" Hb^S allele may be a matter of relative evils. Malaria has been a selective force for thousands of years in tropical and subtropical areas of Asia, the Middle East, and Africa. Through that time span, natural selection has favored the Hb^A/Hb^S combination in all of the malaria-ridden regions, because heterozygotes show more resistance to the disease. In such environments, the combination has proved to have more survival value than either the Hb^S/Hb^S or the Hb^A/Hb^A combination.

With sexual selection, some version of a gender-related trait gives the individual an advantage in reproductive success. Sexual dimorphism is one outcome of sexual selection.

In a population showing balanced polymorphism, natural selection is maintaining two or more alleles at frequencies greater than 1 percent over the generations.

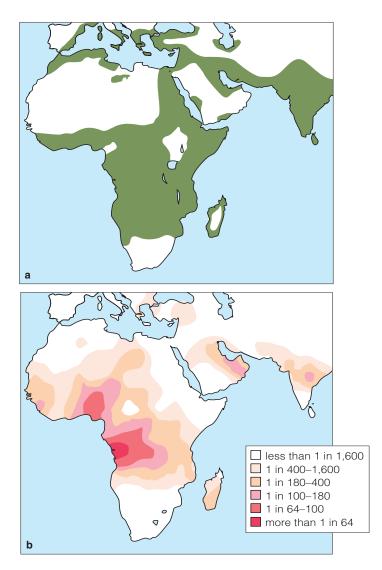




Figure 18.13 (a) Distribution of malaria cases reported in Africa, Asia, and the Middle East in the 1920s, before the start of programs to control mosquitoes, the vector for *Plasmodium*.
(b) Distribution and frequency of people with the sickle-cell trait. Notice the close correlation between the maps. (c) Physician searching for *Plasmodium* larvae in Southeast Asia.

GENETIC DRIFT

18.7 Genetic Drift—The Chance Changes

LINKS TO SECTIONS 11.2, 12.10 Especially in small populations, random changes in allele frequencies can lead to a loss of genetic diversity.

Genetic drift is a random change in allele frequencies over time, brought about by chance alone. Researchers measure it in terms of probability rules. *Probability* is the chance that something will happen relative to the number of times it could happen (Section 11.2). We can measure an event's relative frequency as a fraction on a scale from zero to 1—or 0 to 100 percent of the time. For instance, if 10 million people enter a drawing for a month-long vacation in Hawaii, all expenses paid, each has an equal chance of winning: 1/10,000,000, or an exceedingly improbable 0.00001 percent.

By one probability rule, the expected outcome of some event is less likely to occur if the event happens only rarely. Each time you flip a coin, for example, there is a 50 percent chance it will turn up heads. With 10 flips, odds are high that the proportions of heads and tails will deviate greatly from 50:50. With 1,000 flips, large deviations from 50:50 are less likely.

We can apply the same rule to populations. Because population sizes are not infinite, there will be random changes in allele frequencies. These random changes tend to have minor impact on large populations. They greatly increase the odds that an allele will become more or less prevalent when populations are small.

Steven Rich and his coworkers used small and large populations of the flour beetle (*Tribolium castaneum*) to study genetic drift. They started with beetles that bred true for allele b^+ and other beetles that bred true for mutant allele b. (The superscript plus signifies a wild-type allele.) They hybridized individuals from both groups to get a population of F₁ heterozygotes

(b+b), which they divided into sets of twelve. Different sets consisted of 10, 20, 50, and 100 randomly selected male and female beetles, and the subpopulation sizes were maintained for twenty generations.

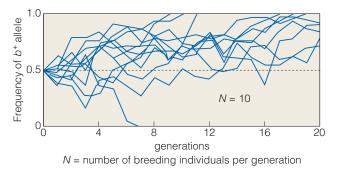
Figure 18.14 shows two of the test results. Drift was greatest in the sets of 10 beetles and least in the sets of 100 beetles. Notice the loss of b^+ from one of the small populations (one graph line ends at 0 in Figure 18.14*a*). Only allele *b* remained. When all of the individuals of a population have become homozygous for one allele only at a locus, we say that **fixation** has occurred.

Thus, random change in allele frequencies leads to the homozygous condition and a loss of genetic diversity over time. This is genetic drift's outcome in all populations; it simply happens faster in small ones (Figure 18.14). Once alleles from the parent population have become fixed, their frequencies will not change again unless mutation or gene flow introduces new alleles.

BOTTLENECKS AND THE FOUNDER EFFECT

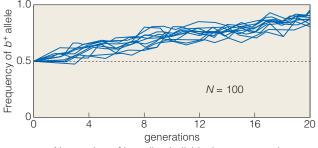
Genetic drift is pronounced when a few individuals rebuild a population or start a new one. This happens after a **bottleneck**, a drastic reduction in population size brought about by severe pressure. Suppose that contagious disease, habitat loss, or hunting nearly wipes out a population. Even if a moderate number of individuals survive a bottleneck, allele frequencies will have been altered at random.

In the 1890s, hunters killed all but twenty of a large population of northern elephant seals. Government restrictions allowed the population to recover to about 130,000 individuals. Each is homozygous for all of the genes analyzed so far.



 The size of twelve populations of beetles was maintained at 10 breeding individuals per generation for twenty generations.

Allele b^+ was lost and b became fixed in one population. Notice that alleles can be fixed or lost even in the absence of selection.



N = number of breeding individuals per generation

b The size of twelve populations was maintained at 100 individuals per generation for twenty generations. Allele *b* did not become fixed. Drift was far less in each generation than it was in the small populations tracked in (**a**).

Figure 18.14 *Animated!* Genetic drift's effect on allele frequencies in small and large populations. The starting frequency of mutant allele b^+ was 0.5.

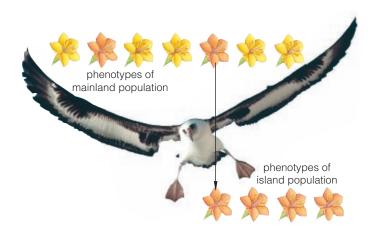


Figure 18.15 Founder effect. This wandering albatross carries seeds, stuck to its feathers, from the mainland to a remote island. By chance, most of the seeds carry an allele for orange flowers that are rare in the original population. Without further gene flow or selection for color, genetic drift will fix the allele on the island.

Unpredictable genetic shifts can occur after a few individuals establish a new population. This form of bottlenecking is a **founder effect**. Genetic diversity might be greatly reduced relative to the original gene pool, as when a lone seed founds a population on a remote island in the middle of the ocean (Figure 18.15).

INBRED POPULATIONS

Genetic drift is less pronounced in inbred populations. **Inbreeding** is nonrandom mating among very close relatives, which share many identical alleles. It leads to the homozygous condition. It also lowers fitness if harmful recessive alleles are increasing in frequency.

Most human societies forbid or discourage incest (inbreeding between parents and children or siblings). Inbreeding among other close relatives is common in geographically or culturally isolated small groups. The Old Order Amish in Pennsylvania are moderately inbred. One outcome is a rather high frequency of a recessive allele that causes *Ellis–van Creveld syndrome*. Affected individuals have extra fingers, toes, or both (Section 12.10). The allele might have been rare when a few founders entered Pennsylvania. Now, about 1 in 8 individuals of the community are heterozygous for the allele, and 1 in 200 are homozygous for it.

Genetic drift is the random change in allele frequencies over the generations, brought about by chance alone. The magnitude of its effect is greatest in small populations, such as one that endures a bottleneck. GENE FLOW

18.8 Gene Flow

Individuals, and their alleles, move into and away from populations. The physical flow of alleles counters changes introduced by other microevolutionary processes.

Individuals of the same species don't always stay put. A population loses alleles when an individual leaves it for good, an event called *emigration*. The population gains alleles when individuals permanently move in, an event called *immigration*. In both cases, **gene flow** —the physical movement of alleles into and out of a population—occurs. This microevolutionary process counters mutation, natural selection, and genetic drift.

Later chapters will give historical examples of how gene flow has kept separated populations genetically similar. For now, simply consider the acorns that blue jays disperse when they gather nuts for the winter. Each fall, jays visit acorn-bearing oak trees repeatedly, then bury acorns in the soil of home territories that may be as much as a mile away (Figure 18.16). Alleles flowing in with the "immigrant acorns" help decrease genetic differences between stands of oak trees.



Figure 18.16 Blue jay, a mover of acorns that helps keep genes flowing between separate oak populations.

Or think of the millions of people from politically explosive, economically bankrupt countries who seek a more stable home. The scale of their emigrations is unprecedented, but the flow of genes is not. Human history is rich with cases of gene flow that minimized many of the genetic differences among geographically separate groups. Remember Genghis Khan? His genes flowed from China to Vienna (Section 12.10). Similarly, the armies of Alexander the Great brought alleles for green eyes from Greece all the way to India.

Gene flow is the physical movement of alleles into and out of a population, through immigration and emigration. It tends to counter the effects of mutation, natural selection, and genetic drift.



18.9 Adaptation to What? A Word of Caution

LINKS TO SECTIONS 1.4, 17.3

Observable traits are not always easy to correlate with conditions in an organism's environment.

"Adaptation" is one of those words that have different meanings in different contexts. An individual plant or animal often can quickly adjust its form, function, and behavior. Junipers in inhospitably windy places grow less tall than junipers of the same species in more sheltered places. This is an example of a *shortterm* adaptation, because it lasts only as long as the individual plant does.

An **evolutionary adaptation** is some aspect of form, function, behavior, or development that improves the odds for surviving and reproducing in a particular environment. This is an *outcome* of microevolution—natural selection especially—an enhancement of the fit between the individual and prevailing conditions.

SALT-TOLERANT TOMATOES

As an example of long-term adaptation, compare how tomato species handle salty water. Tomatoes evolved in Ecuador, Peru, and the Galápagos Islands. The type sold most often in markets, *Lycopersicum esculentum*, has eight close relatives in the wild. If you mix ten grams of table salt with sixty milliliters of water, then pour it into the soil around *L. esculentum*'s roots, the plant will wilt drastically in less than thirty minutes (Figure 18.17*a*). Even when the soil has only 2,500 parts per million of salt, this species grows poorly.

Yet the Galápagos tomato (*L. cheesmanii*) survives and reproduces in seawater-washed soils. We know that its salt tolerance is a heritable adaptation. How? Crosses of a wild species with the commercial species yield a small, edible F_1 hybrid. The hybrid tolerates



Figure 18.17 (a) Severe, rapid wilting of one commercial tomato plant (*Lycopersicum esculentum*) that absorbed salty water. (b) Galápagos tomato plant, *L. cheesmanii*, which stores most absorbed salts in its leaves, not in its fruits.

irrigation water that is two parts fresh and one part salty. It is getting attention in areas where fresh water is scarce and where salts have built up in croplands.

It may take modification of only a few traits to get new salt-tolerant plants. Revving up just one gene for a sodium–hydrogen ion transporter helps the tomato plants use salty water and still bear edible fruits.

NO POLAR BEARS IN THE DESERT

You can safely bet that a polar bear (*Ursus maritimis*) is finely adapted to the icy Arctic, and that its form and function would be a flop in a desert (Figure 18.18). You



Figure 18.18 Which adaptations of a polar bear (*Ursus maritimus*) won't help in a desert? Which ones help an oryx (*Oryx beisa*)? For each animal, make a tentative list of possible structural and functional adaptations to the environment. Later, after you finish reading Unit VI, see how you can expand the list.

CONNECTIONS



Figure 18.19 Adaptation to what? A heritable trait is an adaptation to specific environmental conditions. Hemoglobin of Ilamas, which live at high altitudes, has a high oxygenbinding affinity. However, so does hemoglobin of camels, which live at lower elevations.

might be able to make some educated guesses about why that is so. However, detailed knowledge of its anatomy and physiology might make you view it—or any other animal or plant—with respect. How does a polar bear maintain its internal temperature when it sleeps on ice? How can its muscles function in frigid water? How often must it eat? How does it find food? Conversely, how can an oryx walk about all day in the blistering heat of an African desert? How does it get enough water when there is no water to drink? You will find some answers, or at least ideas about how to look for them, in the next three units of this book.

ADAPTATION TO WHAT?

Bear in mind, it is not always easy to identify a direct relationship between adaptation and the environment. For instance, the prevailing environment may be very different from the one in which a trait evolved.

Consider the llama. It is native to the cloud-piercing peaks of the Andes in western South America (Figure 18.19). The llama lives 4,800 meters (16,000 feet) above sea level. Compared to humans at lower elevations, its lungs have more air sacs and blood vessels. The llama heart has larger chambers, so it pumps larger volumes of blood. Llamas do not have to produce extra blood cells, as people do when they move permanently from lowlands to high elevations. (Extra cells make blood "stickier," so the heart has to pump harder.) But the most publicized adaptation is this: Llama hemoglobin is better than ours at latching on to oxygen. It picks up oxygen in the lungs far more efficiently.

Superficially, at least, the oxygen-binding affinity of llama hemoglobin appears to be an adaptation to thin air at high altitudes. Is it? Apparently not. Llamas are in the same family as dromedary camels. Both share camelid ancestors that evolved in Eocene grasslands and deserts of North America. Later, the ancestors went their separate ways. Forerunners of camels reached Asia's low-elevation grasslands and deserts by a land bridge, which later submerged when the sea level rose. Forerunners of llamas moved down the Isthmus of Panama and on into South America.

Intriguingly, a dromedary camel's hemoglobin also shows a high oxygen-binding capacity. So if the trait arose in a shared ancestor, then how was it adaptive at *low* elevations? We know camels and llamas didn't just *happen* to evolve in the same way. They are close kin, and their most recent ancestors lived in very different environments with different oxygen concentrations.

Who knows why the trait was originally favored? Eocene climates were alternately warm and cool, and hemoglobin's oxygen-binding capacity does go down as temperatures go up. Did it prove adaptive during a long-term shift in climate? Or were its effects neutral at first? What if the allele for efficient hemoglobin was fixed in an ancestral population simply by chance?

Use these "what-ifs" as a reminder to think about observable traits and their presumed connection with a given environment. Identifying the connections takes a great deal of research and experimental tests.

A long-term, heritable adaptation is any aspect of form, function, behavior, or development that contributes to the fit between an individual and its environment.

An adaptive trait improves the odds of surviving and reproducing, or at least it did so under conditions that prevailed when genes for the trait first evolved.

http://biology.brookscole.com/starr11

Summary

Section 18.1 Individuals of a population generally have the same number and kinds of genes for the same traits. Alleles are different molecular forms of a gene. Individuals who inherit different allele combinations vary in details of one or more traits. An allele at any locus may become more or less common relative to other kinds or may be lost.

Mutations are rare in individuals, but they have accumulated in natural populations of all lineages. Mutations are the original source of alleles, the raw material for evolution.

Microevolution refers to changes in allele frequencies of a population brought about by mutation, natural selection, genetic drift, and gene flow (Table 18.1).

Section 18.2 Genetic equilibrium is a state in which a population is not evolving. According to the Hardy–Weinberg equilibrium formula, this occurs only if there is no mutation, the population is infinitely large and isolated from all other populations of the species, there is no natural selection, mating is random, and all individuals survive and produce the same number of offspring. Deviations from this theoretical baseline indicate microevolution is in play.

Biology 🖉 Now

Investigate gene frequencies and genetic equilibrium with the interaction on BiologyNow.

Section 18.3 Natural selection is the outcome of differences in reproduction among individuals of a population that show variations in their shared traits. Three major modes are directional, stabilizing, and disruptive selection. Selection pressures operating on the range of phenotypic variation shift or maintain allele frequencies in the population's gene pool.

Table 18.1 Summary Definitions for Microevolutionary Events

Mutation	A heritable change in DNA; original source of alleles in a population
Natural selection	Outcome of differences in reproduction among individuals of a population that show variation in their shared, heritable traits. Can shift the range of phenotypes in a consistent direction, disrupt it, or stabilize it
Genetic drift	Random changes in a population's allele frequencies through the generations as an outcome of chance alone
Gene flow	Individuals move their alleles into and out of a population by way of immigration and emigration; tends to counter the changes caused by mutation, natural selection, and genetic drift

Section 18.4 Directional selection shifts the range of phenotypic variation in a consistent direction. The individuals at one end of the range of variation are selected against and those at the other end are favored.

Biology 🔊 Now

View the animation of directional selection on BiologyNow. Read the InfoTrac article "AIDS in Africa Has

Potential to Affect Human Evolution,"AIDS Weekly, June 2001.

Section 18.5 Stabilizing selection works against extremes in the range of phenotypic variation, and it favors intermediate forms. Disruptive selection favors forms at both extremes of the range; individuals in the intermediate range are selected against.

Biology 🔊 Now

View the animation of disruptive and stabilizing selection on BiologyNow.

Read the InfoTrac article "Portraits of Evolution: Studies of Coloration in Hawaiian Spiders," Geoffrey S. Oxford, Rosemary G. Gillespie, Bioscience, July 2001.

Section 18.6 Sexual selection, by females or males, leads to forms of traits that favor reproductive success. Persistence in phenotypic differences between males and females (sexual dimorphism) is one outcome.

Selection may result in balanced polymorphism, with nonidentical alleles for a trait being maintained over time at relatively high frequencies.

Biology 🔊 Now

Read the InfoTrac article "High-Risk Defenses," Gregory Cochran, Paul W. Ewald, Natural History, Feb. 1999.

Section 18.7 Genetic drift is a random change in a population's allele frequencies over time due to chance occurrences alone. It tends to lead to the homozygous condition and loss of genetic diversity.

The effect of genetic drift is most pronounced in very small populations, such as ones that have passed through a bottleneck or that arose from a small group of founders. Genetic drift has less effect on inbred populations, which are characterized by nonrandom mating of very close relatives.

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Learn more about genetic drift with the interaction on BiologyNow.

Section 18.8 Gene flow moves alleles into or out of a population by immigration or emigration. The process helps keep populations of the same species genetically alike by countering the effects of mutation, natural selection, and genetic drift.

Section 18.9 Long-term, heritable adaptations are aspects of form, function, behavior, or development that improve the chance of surviving and reproducing, or at least did so under conditions that prevailed when genes for the trait first evolved.

Often it is not easy to correlate an adaptive trait with the particular environmental conditions to which it is assumed to be adapted.

Self-Quiz

Answers in Appendix II

- 1. Individuals don't evolve, _____ do.
- Biologists define evolution as _______.
 a. purposeful change in a lineage
 b. heritable change in a line of descent
 c. acquiring traits during the individual's lifetime
 d. both a and b
- is the original source of new alleles.
 a. Mutation
 b. Natural selection
 c. Genetic drift
 d. Gene flow
 e. All are original sources of new alleles
- 4. Natural selection may occur when there are __________
 a. differences in forms of traits
 b. differences in survival and reproduction among individuals that differ in one or more traits
 c. both a and b
- 5. Directional selection ______.
 a. eliminates common forms of alleles
 b. shifts allele frequencies in a consistent direction
 c. favors intermediate forms of a trait
 - d. works against adaptive traits
- 6. Disruptive selection _____
 - a. eliminates uncommon forms of allelesb. shifts allele frequencies in one direction onlyc. doesn't favor intermediate forms of a traitd. both b and c

7. Sexual selection, especially competition between males for access to fertile females, frequently influences aspects of body form and leads to ______.

a. inbreedingc. sexual dimorphismb. genetic driftd. both b and c

8. The persistence of malaria and sickle-cell anemia

in a population is a case of				
a. bottlenecking	c. natural selection			
b. balanced	d. artificial selection			
polymorphism	e. both b and c			

9. _____ tends to counter changes that occur in the

allele frequencies among	populations of a species.
a. Genetic drift	c. Mutation

b. Gene flow

d. Natural selection

10. Match the evolution concepts.

____ gene flow ____ natural

a. source of new alleles b. changes in a population's allele

selection frequencies due to chance alone

mutation

- c. allele frequencies change owing to
- ____ genetic drift
- d. outcome of differences in survival, reproduction among individuals
- of a population that vary in the details of shared traits

Additional questions are available on Biology ⊗ Now™

Critical Thinking

1. Occasionally, a few of the families in a remote region of Kentucky produce *blue offspring*, a condition caused by an autosomal recessive disorder. Skin of affected individuals appears dark blue. Homozygous individuals do not have



Figure 18.20 Two designer dogs: the Great Dane (*legs, left*) and the chihuahua (*possibly fearful of being stepped on, right*).

the enzyme that maintains hemoglobin in its normal molecular form. Without it, a blue form of hemoglobin accumulates in blood and shows through the skin.

Formulate a hypothesis to explain the recurrence of the blue offspring trait among a cluster of families.

2. Martha is studying a population of tropical birds. The males have brightly colored tail feathers and the females don't. She suspects this difference is maintained by sexual selection. Design an experiment to test her hypothesis.

3. About 50,000 years ago, humans began domesticating wild dogs. By 14,000 years ago, they started to favor new varieties (breeds) by way of artificial selection. Individual dogs having desirable forms of traits were selected from each new litter and, later, encouraged to breed. Those with undesired forms of traits were passed over.

After favoring the pick of the litter for hundreds or thousands of generations, we ended up with sheep-herding border collies, badger-hunting dachshunds, bird-fetching retrievers, and sled-pulling huskies. And at some point we began to delight in the odd, extraordinary dog.

In practically no time at all, evolutionarily speaking, we picked our way through the pool of variant dog alleles and came up with such extreme breeds as Great Danes and chihuahuas (Figure 18.20).

Sometimes the canine designs have exceeded the limits of biological common sense. How long would a tiny, nearly hairless, nearly defenseless, finicky-eating chihuahua last in the wild? Not long. What about English bulldogs, bred for a stubby snout and compressed face? Breeders thought these traits would let the dogs get a better grip on the nose of a bull. (Why they wanted dogs to bite bulls is a story in itself.) So now the roof of the bulldog mouth is ridiculously wide and often flabby, so bulldogs have trouble breathing. Sometimes they get so short of breath they pass out.

Why do you suppose many people easily accept that artificial selection practices can produce startling diversity but will not accept that natural selection might do the same in the wild?