20
Genes within Populations

Concept Outline

20.1 Genes vary in natural populations.

Gene Variation Is the Raw Material of Evolution. Selection acts on the genetic variation present in populations, favoring variants that increase the likelihood of survival and reproduction.

Gene Variation in Nature. Natural populations contain considerable amounts of variation, present at the DNA level and expressed in proteins.

20.2 Why do allele frequencies change in populations?

The Hardy–Weinberg Principle. The proportion of homozygotes and heterozygotes in a population is not altered by meiosis or sexual reproduction.

Five Agents of Evolutionary Change. The frequency of alleles in a population can be changed by evolutionary forces like gene flow and selection.

Identifying the Evolutionary Forces Maintaining Polymorphism. A number of processes can influence allele frequencies in natural populations, but it is difficult to ascertain their relative importance.

Heterozygote Advantage.—In some cases, heterozygotes are superior to either type of homozygote. The gene for sickle cell anemia is one particularly well-understood example.

20.3 Selection can act on traits affected by many genes.

Forms of Selection. Selection can act on traits like height or weight to stabilize or change the level at which the trait is expressed.

Limits to What Selection Can Accomplish. Selection cannot act on traits with little or no genetic variation.

FIGURE 20.1
Genetic variation. The range of genetic material in a population is expressed in a variety of ways—including color.

No other human being is exactly like you (unless you have an identical twin). Often the particular characteristics of an individual have an important bearing on its survival, on its chances to reproduce, and on the success of its offspring. Evolution is driven by such consequences. Genetic variation that influences these characteristics provides the raw material for natural selection, and natural populations contain a wealth of such variation. In plants (figure 20.1), insects, and vertebrates, practically every gene exhibits some level of variation. In this chapter, we will explore genetic variation in natural populations and consider the evolutionary forces that cause allele frequencies in natural populations to change. These deceptively simple matters lie at the core of evolutionary biology.

20.1 Genes vary in natural populations.

Gene Variation Is the Raw Material of Evolution

Evolution Is Descent with Modification

The word "*evolution*" is widely used in the natural and social sciences. It refers to how an entity—be it a social system, a gas, or a planet—changes through time. Although development of the modern concept of evolution in biology can be traced to Darwin's *On the Origin of Species*, the first five editions of this book never actually used the term! Rather, Darwin used the phrase "descent with modification." Although many more complicated definitions have been proposed, Darwin's phrase probably best captures the essence of biological evolution: all species arise from other, pre-existing species. However, through time, they accumulate differences such that ancestral and descendant species are not identical.

Natural Selection Is an Important Mechanism of Evolutionary Change

Darwin was not the first to propose a theory of evolution. Rather, he followed a long line of earlier philosophers and naturalists who deduced that the many kinds of organisms around us were produced by a process of evolution. Unlike his predecessors, however, Darwin proposed **natural selection** as the mechanism of evolution. Natural selection produces evolutionary change when in a population some individuals, which possess certain inherited characteristics, produce more surviving offspring than individuals lacking these characteristics. As a result, the population will gradually come to include more and more individuals with the advantageous characteristics. In this way, the population evolves and becomes better adapted to its local circumstances.

Natural selection was by no means the only evolutionary mechanism proposed. A rival theory, championed by the prominent biologist Jean-Baptiste Lamarck, was that evolution occurred by **the inheritance of acquired characteristics.** According to Lamarck, individuals passed on to offspring body and behavior changes acquired during their lives. Thus, Lamarck proposed that ancestral giraffes with short necks tended to stretch their necks to feed on tree leaves, and this extension of the neck was passed on to subsequent generations, leading to the long-necked giraffe (figure 20.2*a*). In Darwin's theory, by contrast, the variation is not created by experience, but is the result of preexisting genetic differences among individuals (figure 20.2*b*).

Although the efficacy of natural selection is now widely accepted, it is not the only process that can lead to changes in the genetic makeup of populations. Allele frequencies can also change as the result of repeated mutations from one allele to another and from migrants bringing alleles into a population. In addition, when populations are small, the frequencies of alleles can change randomly as the result of chance events. Evolutionary biologists debate the relative strengths of these processes. Although no one denies that natural selection is a powerful force leading to adaptive change, the importance of other processes is less certain.

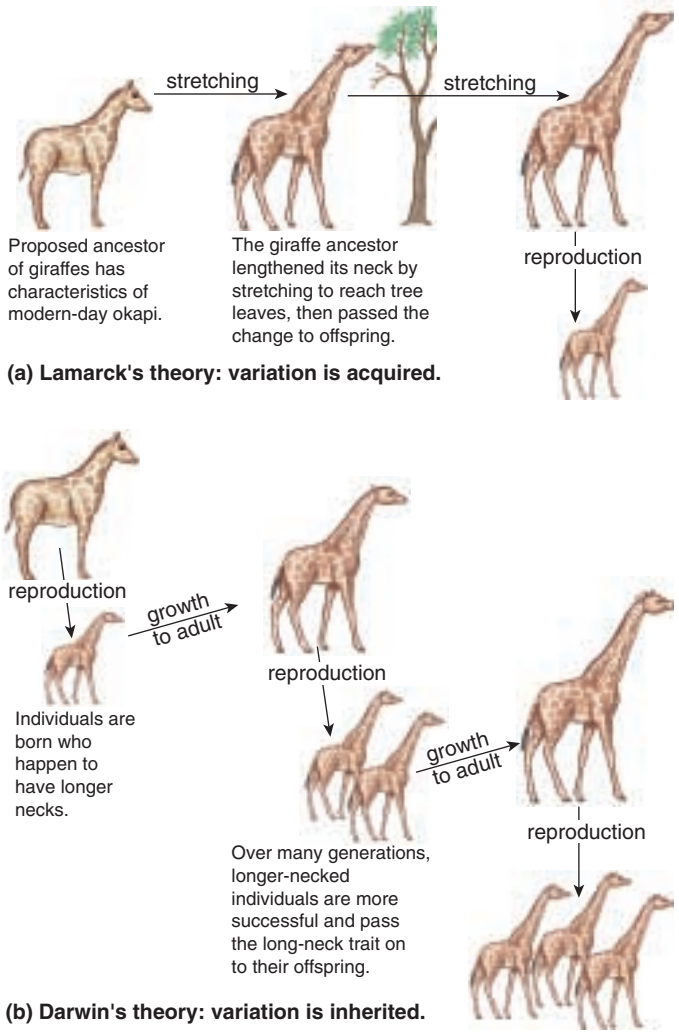

FIGURE 20.2
How did giraffes evolve a long neck?

Darwin proposed that natural selection on variants within populations leads to the evolution of different species.

Gene Variation in Nature

Evolution within a species may result from any process that causes a change in the genetic composition of a population. In considering this theory of population genetics, it is best to start by looking at the genetic variation present among individuals within a species. This is the raw material available for the selective process.

Measuring Levels of Genetic Variation

As we saw in chapter 13, a natural population can contain a great deal of genetic variation. This is true not only of humans, but of all organisms. How much variation usually occurs? Biologists have looked at many different genes in an effort to answer this question:

1. **Blood groups.** Chemical analysis has revealed the existence of more than 30 blood group genes in humans, in addition to the ABO locus. At least a third of these genes are routinely found in several alternative allelic forms in human populations. In addition to these, there are more than 45 variable genes encoding other proteins in human blood cells and plasma which are not considered blood groups. Thus, there are more than 75 genetically variable genes in this one system alone.
2. **Enzymes.** Alternative alleles of genes specifying particular enzymes are easy to distinguish by measuring how fast the alternative proteins migrate in an electric field (a process called **electrophoresis**). A great deal of variation exists at enzyme-specifying loci. About 5% of the enzyme loci of a typical human are heterozygous: if you picked an individual at random, and in turn selected one of the enzyme-encoding genes of that individual at random, the chances are 1 in 20 (5%) that the gene you selected would be heterozygous in that individual.

Considering the entire human genome, it is fair to say that almost all people are different from one another. This is also true of other organisms, except for those that reproduce asexually. In nature, genetic variation is the rule.

Enzyme Polymorphism

Many loci in a given population have more than one allele at frequencies significantly greater than would occur from mutation alone. Researchers refer to a locus with more variation than can be explained by mutation as **polymorphic** (*poly*, "many," *morphic*, "forms") (figure 20.3). The extent of such variation within natural populations was not even suspected a few decades ago, until modern techniques such as gel electrophoresis made it possible to examine enzymes and other proteins directly. We now know that most populations of insects and plants are polymorphic (that is, have more than one allele occurring at a frequency greater than 5%) at more than half of their enzyme-encoding loci, although vertebrates are somewhat less polymorphic. **Heterozygosity** (that is, the probability that a randomly selected gene will be heterozygous for a randomly selected individual) is about 15% in *Drosophila* and other invertebrates, between 5% and 8% in vertebrates, and around 8% in outcrossing plants. These high levels of genetic variability provide ample supplies of raw material for evolution.

FIGURE 20.3
Polymorphic variation. These Australian snails, all of the species *Bankivia fasciata*, exhibit considerable variation in pattern and color. Individual variations are heritable and passed on to offspring.

DNA Sequence Polymorphism

With the advent of gene technology, it has become possible to assess genetic variation even more directly by sequencing the DNA itself. In a pioneering study in 1989, Martin Kreitman sequenced ADH genes isolated from 11 individuals of the fruit fly *Drosophila melanogaster*. He found 43 variable sites, only one of which had been detected by protein electrophoresis! In the following decade, numerous other studies of variation at the DNA level have confirmed these findings: abundant variation exists in both the coding regions of genes and in their nontranslated introns—considerably more variation than we can detect examining enzymes with electrophoresis.

Natural populations contain considerable amounts of genetic variation—more than can be accounted for by mutation alone.

20.2 Why do allele frequencies change in populations?

Population genetics is the study of the properties of genes in populations. Genetic variation within natural populations was a puzzle to Darwin and his contemporaries. The way in which meiosis produces genetic segregation among the progeny of a hybrid had not yet been discovered. Selection, scientists then thought, should always favor an optimal form, and so tend to eliminate variation. Moreover, the theory of **blending inheritance**—in which offspring were expected to be phenotypically intermediate relative to their parents—was widely accepted. If blending inheritance were correct, then the effect of any new genetic variant would quickly be diluted to the point of disappearance in subsequent generations.

The Hardy–Weinberg Principle

Following the rediscovery of Mendel's research, two people in 1908 independently solved the puzzle of why genetic variation persists—G. H. Hardy, an English mathematician, and G. Weinberg, a German physician. They pointed out that the original proportions of the genotypes in a population will remain constant from generation to generation, as long as the following assumptions are met:

1. The population size is very large.
2. Random mating is occurring.
3. No mutation takes place.
4. No genes are input from other sources (no immigration takes place).
5. No selection occurs.

Dominant alleles do not, in fact, replace recessive ones. Because their proportions do not change, the genotypes are said to be in **Hardy–Weinberg equilibrium.**

In algebraic terms, the Hardy–Weinberg principle is written as an equation. Consider a population of 100 cats, with 84 black and 16 white cats. In statistics, **frequency** is defined as the proportion of individuals falling within a certain category in relation to the total number of individuals under consideration. In this case, the respective frequencies would be 0.84 (or 84%) and 0.16 (or 16%). Based on these phenotypic frequencies, can we deduce the underlying frequency of genotypes? If we assume that the white cats are homozygous recessive for an allele we designate *b*, and the black cats are therefore either homozygous dominant *BB* or heterozygous *Bb*, we can calculate the **allele frequencies** of the two alleles in the population from the proportion of black and white individuals. Let the letter *p* designate the frequency of one allele and the letter *q* the frequency of the alternative allele. Because there are only two alleles, *p* plus *q* must always equal 1.

The Hardy-Weinberg equation can now be expressed in the form of what is known as a binomial expansion:

$$(p+q)^2 = p^2 + 2pq + q^2$$

p^2	$2pq$	q^2
(Individuals homozygous for allele *B*)	(Individuals heterozygous with alleles *B* + *b*)	(Individuals homozygous for allele *b*)

If $q^2 = 0.16$ (the frequency of white cats), then $q = 0.4$. Therefore, *p*, the frequency of allele *B*, would be 0.6 (1.0 – 0.4 = 0.6). We can now easily calculate the **genotype frequencies:** there are $p^2 = (0.6)^2 \times 100$ (the number of cats in the total population), or 36 homozygous dominant *BB* individuals. The heterozygous individuals have the *Bb* genotype, and there would be $2pq$, or $(2 \times 0.6 \times 0.4) \times 100$, or 48 heterozygous *Bb* individuals.

Phenotypes			
Genotypes	*BB*	*Bb*	*bb*
Frequency of genotype in population	0.36	0.48	0.16
Frequency of gametes	0.36 + 0.24 = 0.6*B*	0.24 + 0.16 = 0.4*b*	

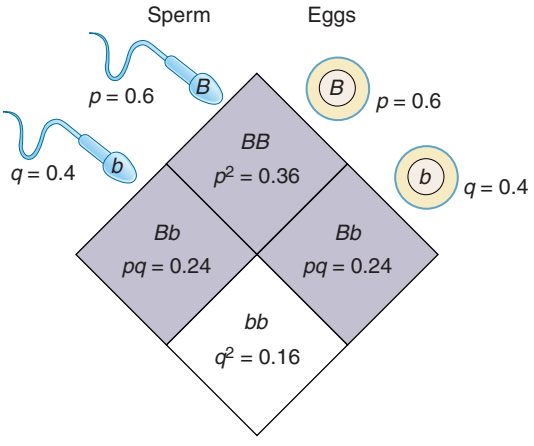

FIGURE 20.4
The Hardy–Weinberg equilibrium. In the absence of factors that alter them, the frequencies of gametes, genotypes, and phenotypes remain constant generation after generation.

Using the Hardy–Weinberg Equation

The Hardy–Weinberg equation is a simple extension of the Punnett square described in chapter 13, with two alleles assigned frequencies p and q. Figure 20.4 allows you to trace genetic reassortment during sexual reproduction and see how it affects the frequencies of the *B* and *b* alleles during the next generation. In constructing this diagram, we have assumed that the union of sperm and egg in these cats is random, so that all combinations of *b* and *B* alleles occur. For this reason, the alleles are mixed randomly and represented in the next generation in proportion to their original representation. Each individual egg or sperm in each generation has a 0.6 chance of receiving a *B* allele ($p = 0.6$) and a 0.4 chance of receiving a *b* allele ($q = 0.4$).

In the next generation, therefore, the chance of combining two *B* alleles is p^2, or 0.36 (that is, 0.6×0.6), and approximately 36% of the individuals in the population will continue to have the *BB* genotype. The frequency of *bb* individuals is q^2 (0.4×0.4) and so will continue to be about 16%, and the frequency of *Bb* individuals will be $2pq$ ($2 \times 0.6 \times 0.4$), or approximately 48%. Phenotypically, if the population size remains at 100 cats, we will still see approximately 84 black individuals (with either *BB* or *Bb* genotypes) and 16 white individuals (with the *bb* genotype) in the population. Allele, genotype, and phenotype frequencies have remained unchanged from one generation to the next.

This simple relationship has proved extraordinarily useful in assessing actual situations. Consider the recessive allele responsible for the serious human disease cystic fibrosis. This allele is present in North Americans of Caucasian descent at a frequency q of about 22 per 1000 individuals, or 0.022. What proportion of North American Caucasians, therefore, is expected to express this trait? The frequency of double recessive individuals (q^2) is expected to be 0.022×0.022, or 1 in every 2000 individuals. What proportion is expected to be heterozygous carriers? If the frequency of the recessive allele q is 0.022, then the frequency of the dominant allele p must be $1 - 0.022$, or 0.978. The frequency of heterozygous individuals ($2pq$) is thus expected to be $2 \times 0.978 \times 0.022$, or 43 in every 1000 individuals.

How valid are these calculated predictions? For many genes, they prove to be very accurate. As we will see, for some genes the calculated predictions do *not* match the actual values. The reasons they do not tell us a great deal about evolution.

Why Do Allele Frequencies Change?

According to the Hardy–Weinberg principle, both the allele and genotype frequencies in a large, random-mating population will remain constant from generation to generation if no mutation, no gene flow, and no selection occur. The stipulations tacked onto the end of the statement are important. In fact, they are the key to the importance of the Hardy–Weinberg principle, because individual allele frequencies often change in natural populations, with some alleles becoming more common and others decreasing in frequency. The Hardy–Weinberg principle establishes a convenient baseline against which to measure such changes. By looking at how various factors alter the proportions of homozygotes and heterozygotes, we can identify the forces affecting particular situations we observe.

Table 20.1 Agents of Evolutionary Change

Factor	Description
Mutation	The ultimate source of variation. Individual mutations occur so rarely that mutation alone does not change allele frequency much.
Gene flow	A very potent agent of change. Populations exchange members.
Nonrandom mating	Inbreeding is the most common form. It does not alter allele frequency but decreases the proportion of heterozygotes.
Genetic drift	Statistical accidents. Usually occurs only in very small populations.
Selection	The only form that produces *adaptive* evolutionary changes.

Many factors can alter allele frequencies. Only five, however, alter the proportions of homozygotes and heterozygotes enough to produce significant deviations from the proportions predicted by the Hardy–Weinberg principle: mutation, gene flow (including both immigration into and emigration out of a given population), nonrandom mating, genetic drift (random change in allele frequencies, which is more likely in small populations), and selection (table 20.1). Of these, only selection produces adaptive evolutionary change because only in selection does the result depend on the nature of the environment. The other factors operate relatively independently of the environment, so the changes they produce are not shaped by environmental demands.

The Hardy–Weinberg principle states that in a large population mating at random and in the absence of other forces that would change the proportions of the different alleles at a given locus, the process of sexual reproduction (meiosis and fertilization) alone will not change these proportions.

Five Agents of Evolutionary Change

1. Mutation

Mutation from one allele to another can obviously change the proportions of particular alleles in a population. Mutation rates are generally so low that they have little effect on the Hardy–Weinberg proportions of common alleles. A single gene may mutate about 1 to 10 times per 100,000 cell divisions (although *some* genes mutate much more frequently than that). Because most environments are constantly changing, it is rare for a population to be stable enough to accumulate changes in allele frequency produced by a process this slow. Nonetheless, mutation is the ultimate source of genetic variation and thus makes evolution possible. It is important to remember, however, that the likelihood of a particular mutation occurring is not affected by natural selection; that is, mutations do not occur more frequently in situations in which they would be favored by natural selection.

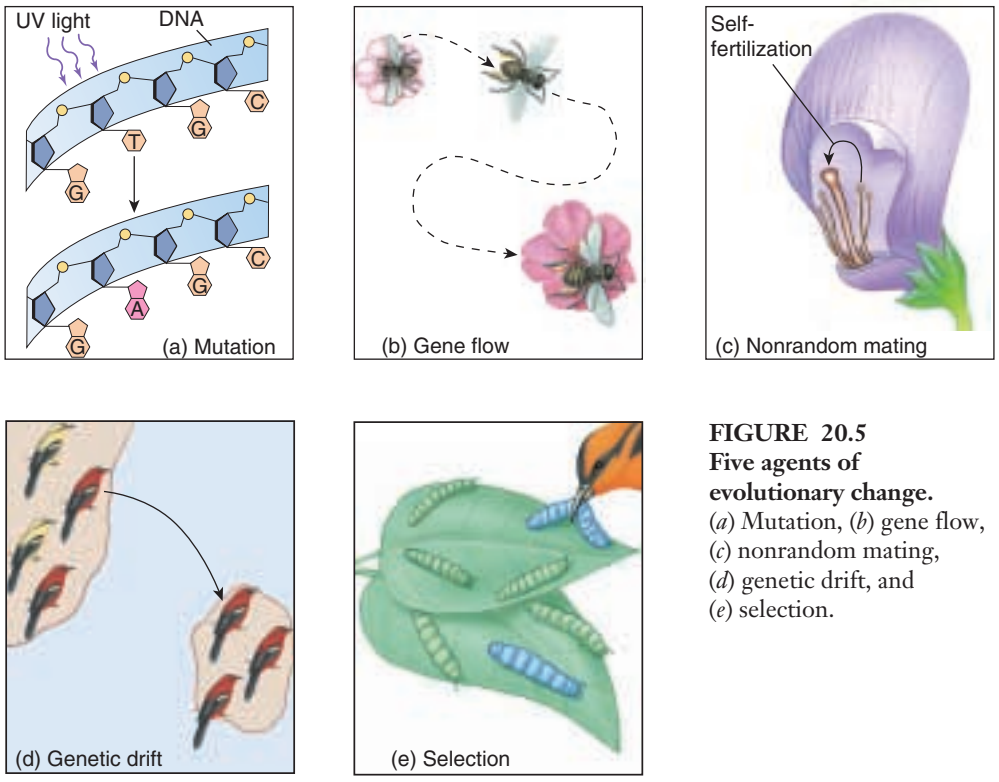

FIGURE 20.5
Five agents of evolutionary change.
(*a*) Mutation, (*b*) gene flow, (*c*) nonrandom mating, (*d*) genetic drift, and (*e*) selection.

2. Gene Flow

Gene flow is the movement of alleles from one population to another. It can be a powerful agent of change because members of two different populations may exchange genetic material. Sometimes gene flow is obvious, as when an animal moves from one place to another. If the characteristics of the newly arrived animal differ from those of the animals already there, and if the newcomer is adapted well enough to the new area to survive and mate successfully, the genetic composition of the receiving population may be altered. Other important kinds of gene flow are not as obvious. These subtler movements include the drifting of gametes or immature stages of plants or marine animals from one place to another (figure 20.5). Male gametes of flowering plants are often carried great distances by insects and other animals that visit their flowers. Seeds may also blow in the wind or be carried by animals or other agents to new populations far from their place of origin. In addition, gene flow may also result from the mating of individuals belonging to adjacent populations.

However it occurs, gene flow can alter the genetic characteristics of populations and prevent them from maintaining Hardy–Weinberg equilibrium. In addition, even low levels of gene flow tend to homogenize allele frequencies among populations and thus keep the populations from diverging genetically. In some situations, gene flow can counter the effect of natural selection by bringing an allele into a population at a rate greater than that at which the allele is removed by selection.

3. Nonrandom Mating

Individuals with certain genotypes sometimes mate with one another more commonly than would be expected on a random basis, a phenomenon known as nonrandom mating. **Inbreeding** (mating with relatives) is a type of nonrandom mating that causes the frequencies of particular genotypes to differ greatly from those predicted by the Hardy–Weinberg principle. Inbreeding does not change the frequency of the alleles, but rather increases the proportion of homozygous individuals because relatives are likely be genetically similar and thus produce offspring with two copies of the same allele. This is why populations of self-fertilizing plants consist primarily of homozygous individuals, whereas **outcrossing** plants, which interbreed with individuals different from themselves, have a higher proportion of heterozygous individuals.

By increasing homozygosity in a population, inbreeding increases the expression of recessive alleles. It is for this reason that marriage between close relatives is discouraged and to some degree outlawed—it increases the possibility of producing children homozygous for an allele associated with one or more of the recessive genetic disorders discussed in chapter 13.

4. Genetic Drift

In small populations, frequencies of particular alleles may change drastically by chance alone. Such changes in allele frequencies occur randomly, as if the frequencies were drifting, and are thus known as **genetic drift.** For this reason, a population must be large to be in Hardy–Weinberg equilibrium. If the gametes of only a few individuals form the next generation, the alleles they carry may by chance not be representative of the parent population from which they were drawn, as illustrated in figure 20.6, where a small number of individuals are removed from a bottle containing many. By chance, most of the individuals removed are blue, so the new population has a much higher population of blue individuals than the parent one had.

A set of small populations that are isolated from one another may come to differ strongly as a result of genetic drift even if the forces of natural selection do not differ between the populations. Indeed, because of genetic drift, harmful alleles may increase in frequency in small populations, despite selective disadvantage, and favorable alleles may be lost even though selectively advantageous. It is interesting to realize that humans have lived in small groups for much of the course of their evolution; consequently, genetic drift may have been a particularly important factor in the evolution of our species.

Even large populations may feel the effect of genetic drift. Large populations may have been much smaller in the past, and genetic drift may have greatly altered allele frequencies at that time. Imagine a population containing only two alleles of a gene, *B* and *b*, in equal frequency (that is, $p = q = 0.5$). In a large Hardy–Weinberg population, the genotype frequencies are expected to be 0.25 *BB*, 0.50 *Bb*, and 0.25 *bb*. If only a small sample produces the next generation, large deviations in these genotype frequencies can occur by chance. Imagine, for example, that four individuals form the next generation, and that by chance they are two *Bb* heterozygotes and two *BB* homozygotes—the allele frequencies in the next generation are $p = 0.75$ and $q = 0.25$! If you were to replicate this experiment 1000 times, each time randomly drawing four individuals from the parental population, one of the two alleles would be missing entirely from about 8 of the 1000 populations. This leads to an important conclusion: genetic drift leads to the loss of alleles in isolated populations. Two related causes of decreases in a population's size are founder effects and bottlenecks.

Founder Effects. Sometimes one or a few individuals disperse and become the founders of a new, isolated population at some distance from their place of origin. These pioneers are not likely to have all the alleles present in the source population. Thus, some alleles may be lost from the new population and others may change drastically in frequency. In some cases, previously rare alleles in the source population may be a significant fraction of the new population's genetic endowment. This phenomenon is called the founder effect. Founder effects are not rare in nature.

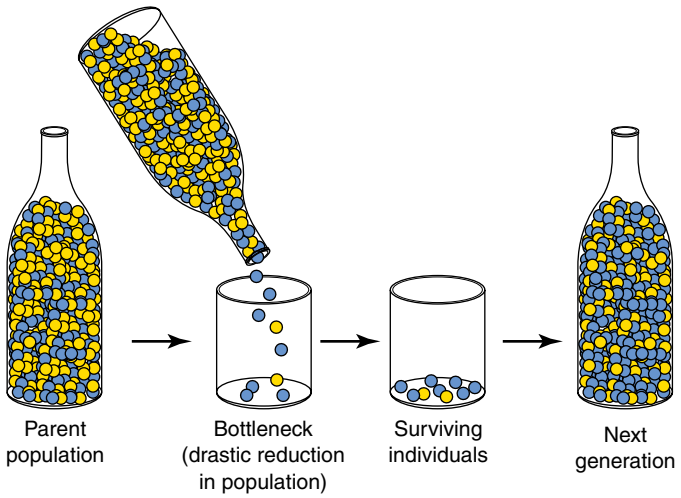

FIGURE 20.6
Genetic drift: The bottleneck effect. The parent population contains roughly equal numbers of blue and yellow individuals. By chance, the few remaining individuals that comprise the next generation are mostly blue. The bottleneck occurs because so few individuals form the next generation, as might happen after an epidemic or catastrophic storm.

Many self-pollinating plants start new populations from a single seed.

Founder effects have been particularly important in the evolution of organisms on distant oceanic islands, such as the Hawaiian Islands and the Galápagos Islands visited by Darwin. Most of the organisms in such areas probably derive from one or a few initial "founders." In a similar way, isolated human populations are often dominated by genetic features characteristic of their particular founders.

The Bottleneck Effect. Even if organisms do not move from place to place, occasionally their populations may be drastically reduced in size. This may result from flooding, drought, epidemic disease, and other natural forces, or from progressive changes in the environment. The few surviving individuals may constitute a random genetic sample of the original population (unless some individuals survive specifically because of their genetic makeup). The resultant alterations and loss of genetic variability has been termed the **bottleneck effect.**

Some living species appear to be severely depleted genetically and have probably suffered from a bottleneck effect in the past. For example, the northern elephant seal, which breeds on the western coast of North America and nearby islands, was nearly hunted to extinction in the nineteenth century and was reduced to a single population containing perhaps no more than 20 individuals on the island of Guadalupe off the coast of Baja, California. As a result of this bottleneck, even though the seal populations have rebounded and now number in the tens of thousands, this species has lost almost all of its genetic variation.

5. Selection

As Darwin pointed out, some individuals leave behind more progeny than others, and the rate at which they do so is affected by phenotype and behavior. We describe the results of this process as **selection** and speak of both **artificial selection** and **natural selection.** In artificial selection, the breeder selects for the desired characteristics. In natural selection, environmental conditions determine which individuals in a population produce the most offspring. For natural selection to occur and result in evolutionary change, three conditions must be met:

1. **Variation must exist among individuals in a population.** Natural selection works by favoring individuals with some traits over individuals with alternative traits. If no variation exists, natural selection cannot operate.
2. **Variation among individuals results in differences in number of offspring surviving in the next generation.** This is the essence of natural selection. Because of their phenotype or behavior, some individuals are more successful than others in producing offspring and thus passing their genes on to the next generation.
3. **Variation must be genetically inherited.** For natural selection to result in evolutionary change, the selected differences must have a genetic basis. However, not all variation has a genetic basis—even genetically identical individuals may be phenotypically quite distinctive if they grow up in different environments. Such environmental effects are common in nature. In many turtles, for example, individuals that hatch from eggs laid in moist soil are heavier, with longer and wider shells, than individuals from nests in drier areas. As a result of these environmental effects, variation within a population does not always indicate the existence of underlying genetic variation. When phenotypically different individuals do not differ genetically, then differences in the number of their offspring will not alter the genetic composition of the population in the next generation and, thus, no evolutionary change will have occurred.

It is important to remember that natural selection and evolution are not the same—the two concepts often are incorrectly equated. Natural selection is a process, whereas evolution is the historical record of change through time. Evolution is an outcome, not a process. Natural selection (the process) can lead to evolution (the outcome), but natural selection is only one of several processes that can produce evolutionary change. Moreover, natural selection can occur without producing evolutionary change; only if variation is genetically based will natural selection lead to evolution.

Selection to Avoid Predators. Many of the most dramatic documented instances of adaptation involve genetic changes which decrease the probability of capture by a predator. The caterpillar larvae of the common sulphur butterfly *Colias eurytheme* usually exhibit a dull Kelly green color, providing excellent camouflage on the alfalfa plants on which they feed. An alternative bright blue color morph is kept at very low frequency because this color renders the larvae highly visible on the food plant, making it easier for bird predators to see them. In a similar fashion, the way the shell markings in the land snail *Cepaea nemoralis* match its background habitat reflects the same pattern of avoiding predation by camouflage.

One of the most dramatic examples of background matching involves ancient lava flows in the middle of deserts in the American southwest. In these areas, the black rock formations produced when the lava cooled contrasts starkly to the surrounding bright glare of the desert sand. Populations of many species of animals—including lizards, rodents, and a variety of insects—occurring on these rocks are dark in color, whereas sand-dwelling populations in surrounding areas are much lighter (figure 20.7). Predation is the likely cause selecting for these differences in color. Laboratory studies have confirmed that predatory birds are adept at picking out individuals occurring on backgrounds to which they are not adapted.

FIGURE 20.7
Pocket mice from the Tularosa Basin of New Mexico whose color matches their background. (*a*) The rock pocket mouse lives on lava, (*b*) while the Apache pocket mouse lives on white sand.

Selection to Match Climatic Conditions. Many studies of selection have focused on genes encoding enzymes because in such cases the investigator can directly assess the consequences to the organism of changes in the frequency of alternative enzyme alleles. Often investigators find that enzyme allele frequencies vary latitudinally, with one allele more common in northern populations but progressively less common at more southern locations. A superb example is seen in studies of a fish, the mummichog, *Fundulus heteroclitus*, which ranges along the eastern coast of North America. In this fish, allele frequencies of the gene that produces the enzyme lactase dehydrogenase, which catalyzes the conversion of pyruvate to lactate, vary geographically (figure 20.8). Biochemical studies show that the enzymes formed by these alleles function differently at different temperatures, thus explaining their geographic distributions. For example, the form of the enzyme that is more frequent in the north is a better catalyst at low temperatures than the enzyme from the south. Moreover, functional studies indicate that at low temperatures, individuals with the northern allele swim faster, and presumably survive better, than individuals with the alternative allele.

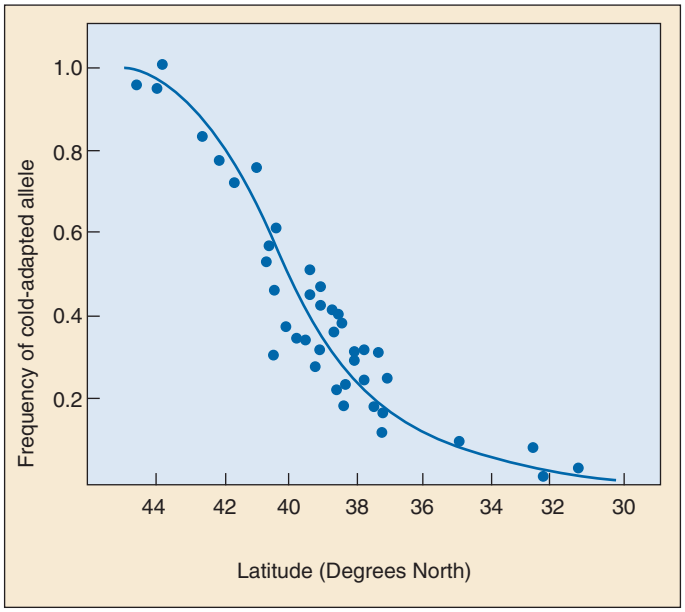

FIGURE 20.8
Selection to match climatic conditions. Frequency of the cold-adapted allele for lactase dehydrogenase in the mummichog (*Fundulus heteroclitus*) decreases at lower latitudes, which are warmer.

Selection for Pesticide Resistance. A particularly clear example of selection in action in natural populations is provided by studies of pesticide resistance in insects. The widespread use of insecticides has led to the rapid evolution of resistance in more than 400 pest species. For example, the resistance allele at the *pen* gene decreases the uptake of insecticide, whereas alleles at the *kdr* and *dld-r* genes decrease the number of target sites, thus decreasing the binding ability of the insecticide (figure 20.9). Other alleles enhance the ability of the insects' enzymes to identify and detoxify insecticide molecules.

Single genes are also responsible for resistance in other organisms. The pigweed, *Amaranthus hybridus*, is one of about 28 agricultural weeds that have evolved resistance to the herbicide Triazine. Triazine inhibits photosynthesis by binding to a protein in the chloroplast membrane. Single amino acid substitutions in the gene encoding the protein diminish the ability of Triazine to decrease the plant's photosynthetic capabilities. Similarly, Norway rats are normally susceptible to the pesticide Warfarin, which diminishes the clotting ability of the rat's blood and leads to fatal hemorrhaging. However, a resistance allele at a single gene alters a metabolic pathway and renders Warfarin ineffective.

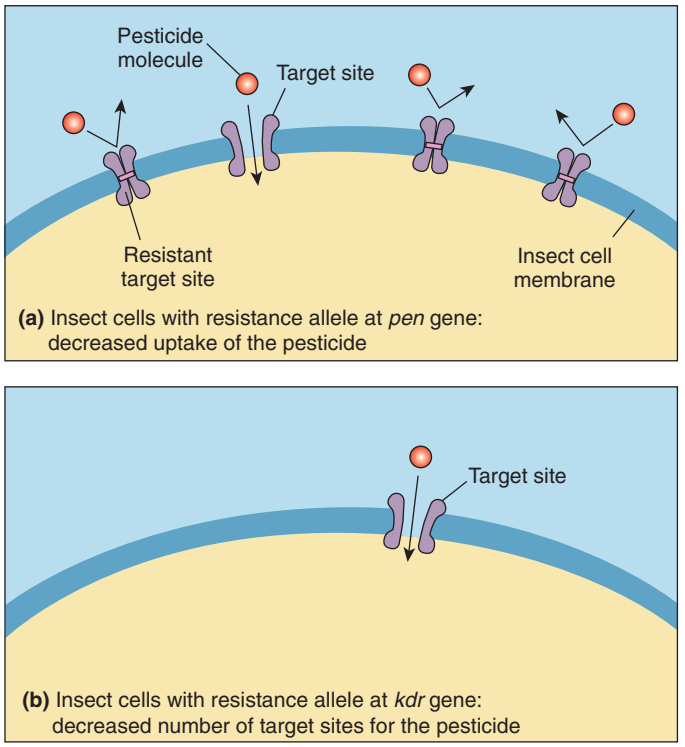

FIGURE 20.9
Selection for pesticide resistance. Resistance alleles at genes like *pen* and *kdr* allow insects to be more resistant to pesticides. Insects that possess these resistance alleles have become more common through selection.

Five factors can bring about a deviation from the proportions of homozygotes and heterozygotes predicted by the Hardy-Weinberg principle. Only selection regularly produces adaptive evolutionary change, but the genetic constitution of individual populations, and thus the course of evolution, can also be affected by mutation, gene flow, nonrandom mating, and genetic drift.

Identifying the Evolutionary Forces Maintaining Polymorphism

The Adaptive Selection Theory

As evidence began to accumulate in the 1970s that natural populations exhibit a great deal of genetic polymorphism (that is, many alleles of a gene exist in the population), the question arose: What evolutionary force is maintaining the polymorphism? As we have seen, there are in principle five processes that act on allele frequencies: mutation, migration, nonrandom mating, genetic drift, and selection. Because migration and nonrandom mating are not major influences in most natural populations, attention focused on the other three forces.

The first suggestion, advanced by R. C. Lewontin (one of the discovers of enzyme polymorphism) and many others, was that selection was the force acting to maintain the polymorphism. Natural environments are often quite heterogeneous, so selection might reasonably be expected to pull gene frequencies in different directions within different microhabitats, generating a condition in which many alleles persist. This proposal is called the **adaptive selection theory.**

The Neutral Theory

A second possibility, championed by the great Japanese geneticist Moto Kimura, was that a balance between mutation and genetic drift is responsible for maintaining polymorphism. Kimura used elegant mathematics to demonstrate that, even in the absence of selection, natural populations could be expected to contain considerable polymorphism if mutation rates (generating the variation) were high enough and population sizes (promoting genetic drift) were small enough. In this proposal, selection is not acting, differences between alleles being "neutral to selection." The proposal is thus called the **neutral theory.**

Kimura's theory, while complex, can be stated simply:

$$\bar{H} = 1/(4N_e\mu + 1)$$

$\bar{H}$, the mean heterozygosity, is the likelihood that a randomly selected member of the population will be heterozygous at a randomly selected locus. In a population without selection, this value is influenced by two variables, the effective population size (N_e) and the mutation rate (μ).

The peculiar difficulty of the neutral theory is that the level of polymorphism, as measured by $\bar{H}$, is determined by the product of a very large number, N_e, and a very small number, μ, both very difficult to measure with precision. As a result, the theory can account for almost any value of $\bar{H}$, making it very difficult to prove or disprove. As you might expect, a great deal of controversy has resulted.

Testing the Neutral Theory

Choosing between the adaptive selection theory and the neutral theory is not simple, for they both appear to account for much of the data on gene polymorphism in natural populations. A few well-characterized instances where selection acts on enzyme alleles do not settle the more general issue. An attempt to test the neutral theory by examining large-scale patterns of polymorphism sheds light on the difficulty of choosing between the two theories:

Population size: According to the neutral theory, polymorphism as measured by $\bar{H}$ should be proportional to the effective population size N_e, assuming the mutation rate among neutral alleles μ is constant. Thus, $\bar{H}$ should be much greater for insects than humans, as there are far more individuals in an insect population than in a human one. When DNA sequence variation is examined, the fruit fly *Drosophila melanogaster* indeed exhibits sixfold higher levels of variation, as the theory predicts; but when enzyme polymorphisms are examined, levels of variation in fruit flies and humans are similar. If the level of DNA variation correctly mirrors the predictions of the neutral theory, then something (selection?) is increasing variation at the enzyme level in humans. These sorts of patterns argue for rejection of the neutral theory.

The nearly neutral model: One way to rescue the neutral theory from these sorts of difficulties is to retreat from the assumption of strict neutrality, modifying the theory to assume that many of the variants are slightly deleterious rather than strictly neutral to selection. With this adjustment, it is possible to explain many of the population-size-dependent large-scale patterns. However, little evidence exists that the wealth of enzyme polymorphism in natural populations is in fact slightly deleterious.

As increasing amounts of DNA sequence data become available, a detailed picture of variation at the DNA level is emerging. It seems clear that most nucleotide substitutions that change amino acids are disadvantageous and are eliminated by selection. But what about the many protein alleles that are seen in natural populations? Are they nearly neutral or advantageous? No simple answer is yet available, although the question is being actively investigated. Levels of polymorphism at enzyme-encoding genes may depend on both the action of selection on the gene (the adaptive selection theory) and on the population dynamics of the species (the nearly neutral theory), with the relative contribution varying from one gene to the next.

Adaptive selection clearly maintains some enzyme polymorphisms in natural populations. Genetic drift seems to play a major role in producing the variation we see at the DNA level. For most enzyme-level polymorphism, investigators cannot yet choose between the selection theory and the nearly neutral theory.

Interactions among Evolutionary Forces

When alleles are not selectively neutral, levels of variation retained in a population may be determined by the relative strength of different evolutionary processes. In theory, for example, if allele *B* mutates to allele *b* at a high enough rate, allele *b* could be maintained in the population even if natural selection strongly favored allele *B*. In nature, however, mutation rates are rarely high enough to counter the effects of natural selection.

The effect of natural selection also may be countered by genetic drift. Both processes may act to remove variation from a population. However, whereas selection is a deterministic process that operates to increase the representation of alleles that enhance survival and reproductive success, drift is a random process. Thus, in some cases, drift may lead to a decrease in the frequency of an allele that is favored by selection. In some extreme cases, drift may even lead to the loss of a favored allele from a population. Remember, however, that the magnitude of drift is negatively related to population size; consequently, natural selection is expected to overwhelm drift except when populations are very small.

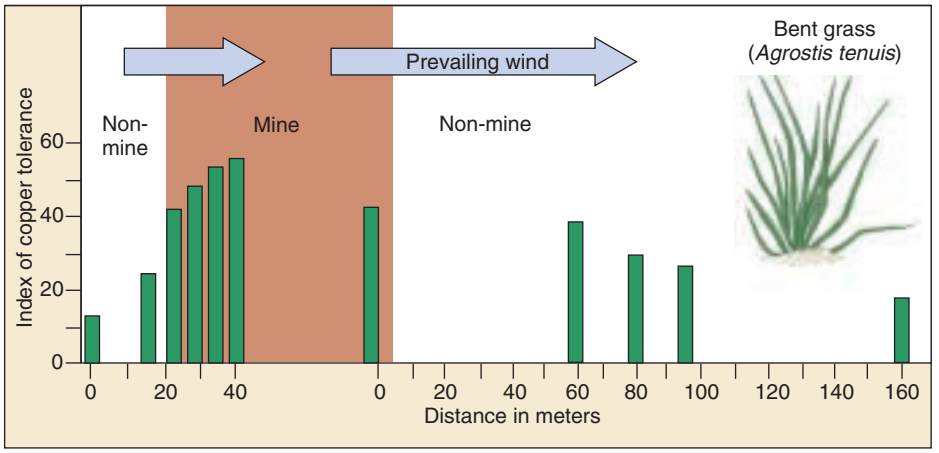

FIGURE 20.10
Degree of copper tolerance in grass plants on and near ancient mine sites. Prevailing winds blow pollen containing nontolerant alleles onto the mine site and tolerant alleles beyond the site's borders.

Gene Flow versus Natural Selection

Gene flow can be either a constructive or a constraining force. On one hand, gene flow can increase the adaptedness of a species by spreading a beneficial mutation that arises in one population to other populations within a species. On the other hand, gene flow can act to impede adaptation within a population by continually importing inferior alleles from other populations. Consider two populations of a species that live in different environments. In this situation, natural selection might favor different alleles—*B* and *b*—in the different populations. In the absence of gene flow and other evolutionary processes, the frequency of *B* would be expected to reach 100% in one population and 0% in the other. However, if gene flow were going on between the two populations, then the less favored allele would continually be reintroduced into each population. As a result, the frequency of the two alleles in each population would reflect a balance between the rate at which gene flow brings the inferior allele into a population versus the rate at which natural selection removes it.

A classic example of gene flow opposing natural selection occurs on abandoned mine sites in Great Britain. Although mining activities ceased hundreds of years ago, the concentration of metal ions in the soil is still much greater than in surrounding areas. Heavy metal concentrations are generally toxic to plants, but alleles at certain genes confer resistance. The ability to tolerate heavy metals comes at a price, however; individuals with the resistance allele exhibit lower growth rates on non-polluted soil. Consequently, we would expect the resistance allele to occur with a frequency of 100% on mine sites and 0% elsewhere. Heavy metal tolerance has been studied particularly intensively in the slender bent grass, *Agrostis tenuis*, in which researchers have found that the resistance allele occurs at intermediate levels in many areas (figure 20.10). The explanation relates to the reproductive system of this grass in which pollen, the male gamete (that is, the floral equivalent of sperm), is dispersed by the wind. As a result, pollen—and the alleles it carries—can be blown for great distances, leading to levels of gene flow between mine sites and unpolluted areas high enough to counteract the effects of natural selection.

In general, the extent to which gene flow can hinder the effects of natural selection should depend on the relative strengths of the two processes. In species in which gene flow is generally strong, such as birds and wind-pollinated plants, the frequency of the less favored allele may be relatively high, whereas in more sedentary species which exhibit low levels of gene flow, such as salamanders, the favored allele should occur at a frequency near 100%.

Evolutionary processes may act to either remove or maintain genetic variation within a population. Allele frequency sometimes may reflect a balance between opposed processes, such as gene flow and natural selection. In such cases, observed frequencies will depend on the relative strength of the processes.

Heterozygote Advantage

In the previous pages, natural selection has been discussed as a process that removes variation from a population by favoring one allele over others at a genetic locus. However, if heterozygotes are favored over homozygotes, then natural selection actually will tend to maintain variation in the population. The reason is simple. Instead of tending to remove less successful alleles from a population, such **heterozygote advantage** will favor individuals with copies of both alleles, and thus will work to maintain both alleles in the population. Some evolutionary biologists believe that heterozygote advantage is pervasive and can explain the high levels of polymorphism observed in natural populations. Others, however, believe that it is relatively rare.

Sickle Cell Anemia

The best documented example of heterozygote advantage is sickle cell anemia, a hereditary disease affecting hemoglobin in humans. Individuals with sickle cell anemia exhibit symptoms of severe anemia and contain abnormal red blood cells which are irregular in shape, with a great number of long and sickle-shaped cells. The disease is particularly common among African Americans. In chapter 13, we noted that this disorder, which affects roughly 3 African Americans out of every 1000, is associated with a particular recessive allele. Using the Hardy–Weinberg equation, you can calculate the frequency of the sickle cell allele in the African-American population; this frequency is the square root of 0.003, or approximately 0.054. In contrast, the frequency of the allele among white Americans is only about 0.001.

Sickle cell anemia is often fatal. Until therapies were developed to more effectively treat its symptoms, almost all affected individuals died as children. Even today, 31% of patients in the United States die by the age of 15. The disease occurs because of a single amino acid change, repeated in the two beta chains of the hemoglobin molecule. In this change, a valine replaces the usual glutamic acid at a location on the surface of the protein near the oxygen-binding site. Unlike glutamic acid, valine is nonpolar (hydrophobic). Its presence on the surface of the molecule creates a "sticky" patch that attempts to escape from the polar water environment by binding to another similar patch. As long as oxygen is bound to the hemoglobin molecule there is no problem, because the hemoglobin atoms shield the critical area of the surface. When oxygen levels fall, such as after exercise or when an individual is stressed, oxygen is not so readily bound to hemoglobin and the exposed sticky patch binds to similar patches on other hemoglobin molecules, eventually producing long, fibrous clumps (figure 20.11). The result is a deformed, "sickle-shaped" red blood cell.

Individuals who are heterozygous or homozygous for the valine-specifying allele (designated allele *S*) are said to possess the sickle cell trait. Heterozygotes produce some sickle-shaped red blood cells, but only 2% of the number seen in homozygous individuals. The reason is that in heterozygotes, one-half of the molecules do not contain valine at the critical location. Consequently, when a molecule produced by the non-sickle cell allele is added to the chain, there is no further "sticky" patch available to add additional molecules and chain elongation stops. Hence, most chains in heterozygotes are too short to produce sickling of the cell.

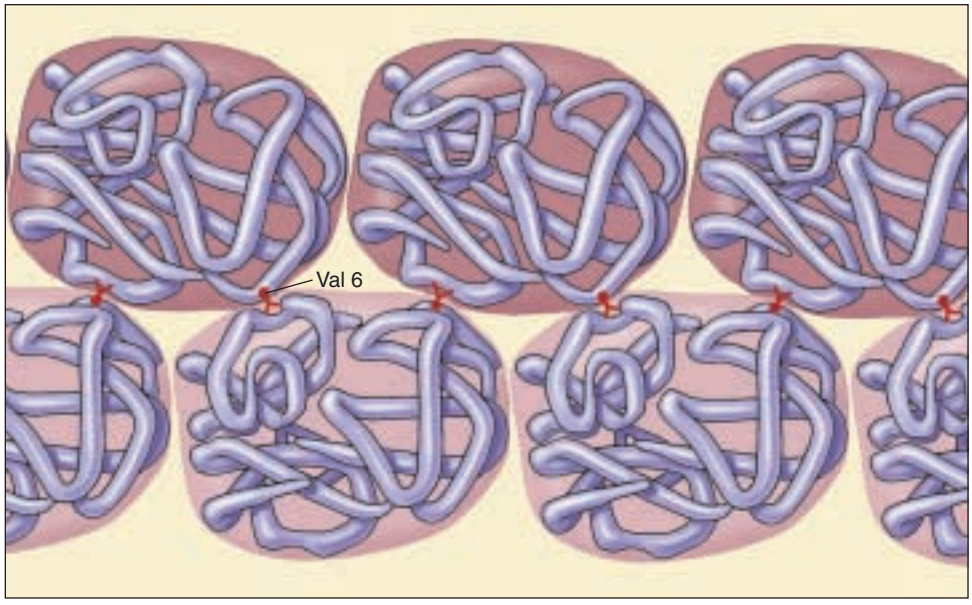

FIGURE 20.11
Why the sickle cell mutation causes hemoglobin to clump. The sickle cell mutation changes the sixth amino acid in the hemoglobin β chain (position B6) from glutamic acid (very polar) to valine (nonpolar). The unhappy result is that the nonpolar valine at position B6, protruding from a corner of the hemoglobin molecule, fits into a nonpolar pocket on the opposite side of another hemoglobin molecule, causing the two molecules to clump together. As each molecule has both a B6 valine and an opposite nonpolar pocket, long chains form. When polar glutamic acid (the normal allele) occurs at position B6, it is not attracted to the nonpolar pocket, and no clumping occurs.

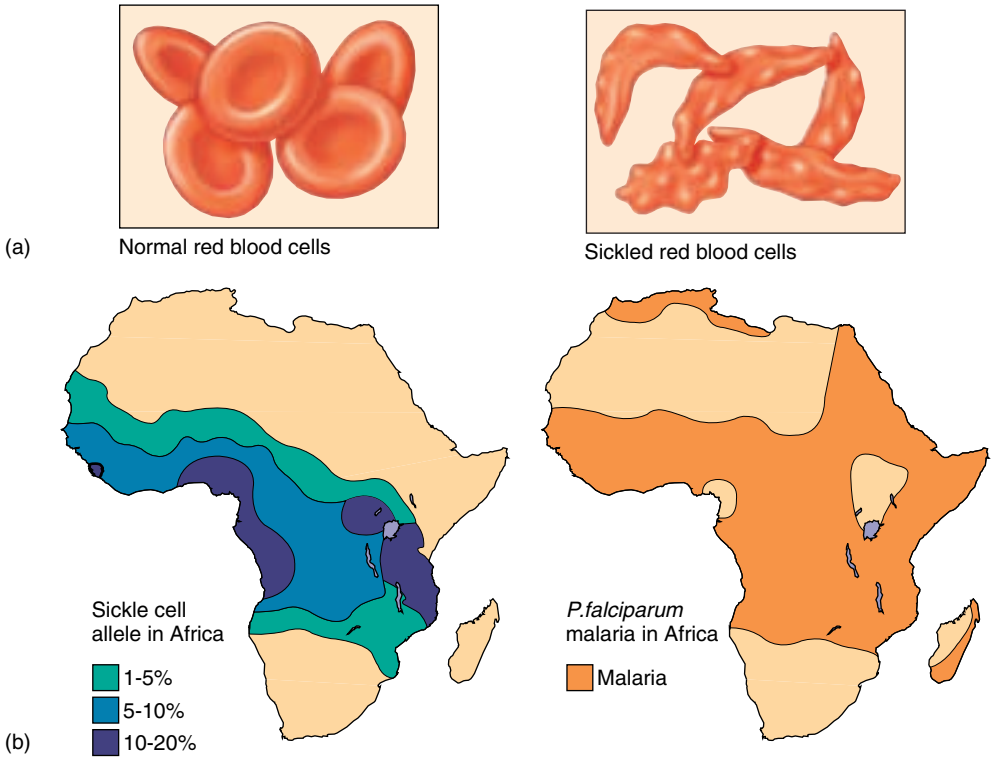

FIGURE 20.12
Frequency of sickle cell allele and distribution of *Plasmodium falciparum* malaria. (*a*)The red blood cells of people homozygous for the sickle cell allele collapse into sickled shapes when the oxygen level in the blood is low. (*b*) The distribution of the sickle cell allele in Africa coincides closely with that of *P. falciparum* malaria.

Malaria and Heterozygote Advantage

The average incidence of the *S* allele in the Central African population is about 0.12, far higher than that found among African Americans. From the Hardy–Weinberg principle, you can calculate that 1 in 5 Central African individuals are heterozygous at the *S* allele, and 1 in 100 develops the fatal form of the disorder. People who are homozygous for the sickle cell allele almost never reproduce because they usually die before they reach reproductive age. Why is the *S* allele not eliminated from the Central African population by selection, rather than being maintained at such high levels? People who are heterozygous for the sickle cell allele are much less susceptible to malaria—one of the leading causes of illness and death in Central Africa, especially among young children—in the areas where the allele is common. The reason is that when the parasite that causes malaria, *Plasmodium falciparum*, enters a red blood cell, it causes extremely low oxygen tension in the cell, which leads to cell sickling even in heterozygotes. Such cells are quickly filtered out of the bloodstream by the spleen, thus eliminating the parasite (the spleen's filtering effect is what leads to anemia in homozygotes as large numbers of red blood cells are removed).

Consequently, even though most homozygous recessive individuals die before they have children, the sickle cell allele is maintained at high levels in these populations (it is selected for) because of its association with resistance to malaria in heterozygotes and also, for reasons not yet fully understood, with increased fertility in female heterozygotes.

For people living in areas where malaria is common, having the sickle cell allele in the heterozygous condition has adaptive value (figure 20.12). Among African Americans, however, many of whose ancestors have lived for some 15 generations in a country where malaria has been relatively rare and is now essentially absent, the environment does not place a premium on resistance to malaria. Consequently, no adaptive value counterbalances the ill effects of the disease; in this nonmalarial environment, selection is acting to eliminate the *S* allele. Only 1 in 375 African Americans develop sickle cell anemia, far less than in Central Africa.

The hemoglobin allele *S*, responsible for sickle cell anemia in homozygotes, is maintained by heterozygote advantage in Central Africa, where heterozygotes for the *S* allele are resistant to malaria.

20.3 Selection can act on traits affected by many genes.

Forms of Selection

In nature many traits, perhaps most, are affected by more than one gene. The interactions between genes are typically complex, as you saw in chapter 13. For example, alleles of many different genes play a role in determining human height (see figure 13.18). In such cases, selection operates on all the genes, influencing most strongly those that make the greatest contribution to the phenotype. How selection changes the population depends on which genotypes are favored.

Disruptive Selection

In some situations, selection acts to eliminate rather than to favor intermediate types. A clear example is the different beak sizes of the African fire-bellied seedcracker finch *Pyronestes ostrinus*. Populations of these birds contain individuals with large and small beaks, but very few individuals with intermediate-sized beaks. As their name implies, these birds feed on seeds, and the available seeds fall into two size categories: large and small. Only large-beaked birds can open the tough shells of large seeds, whereas birds with the smallest beaks are most adept at handling small seeds. Birds with intermediate-sized beaks are at a disadvantage with both seed types: unable to open large seeds and too clumsy to efficiently process small seeds. Consequently, selection acts to eliminate the intermediate phenotypes, in effect partitioning the population into two phenotypically distinct groups. This form of selection is called **disruptive selection** (figure 20.13*a*).

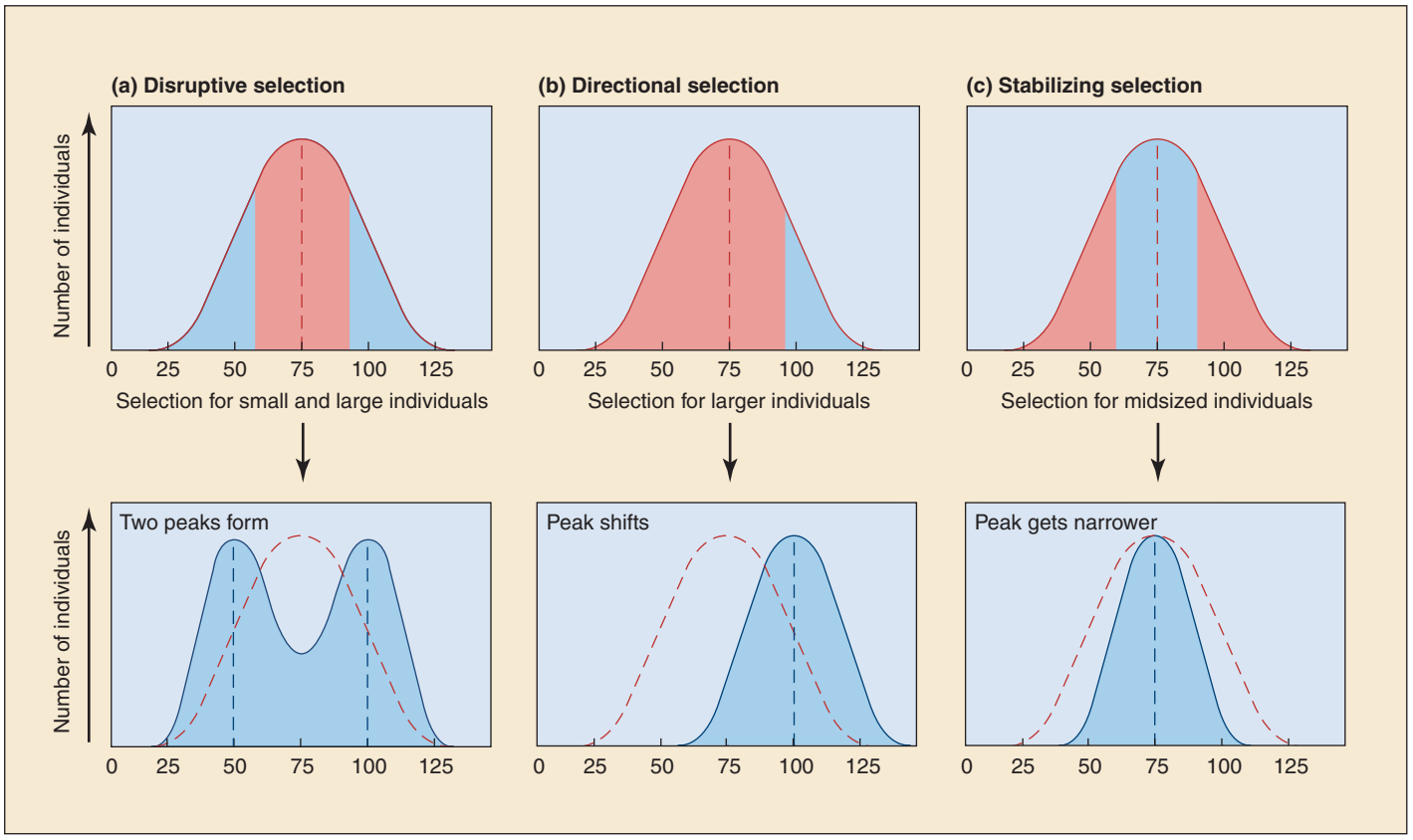

FIGURE 20.13
Three kinds of natural selection. The top panels show the populations before selection has occurred, with the forms that will be selected against shaded red and the forms that will be favored shaded blue. The bottom panels indicate what the populations will look like after selection has occurred. (*a*) In *disruptive selection*, individuals in the middle of the range of phenotypes of a certain trait are selected against (*red*), and the extreme forms of the trait are favored (*blue*). (*b*) In *directional selection*, individuals concentrated toward one extreme of the array of phenotypes are favored. (*c*) In *stabilizing selection*, individuals with midrange phenotypes are favored, with selection acting against both ends of the range of phenotypes.

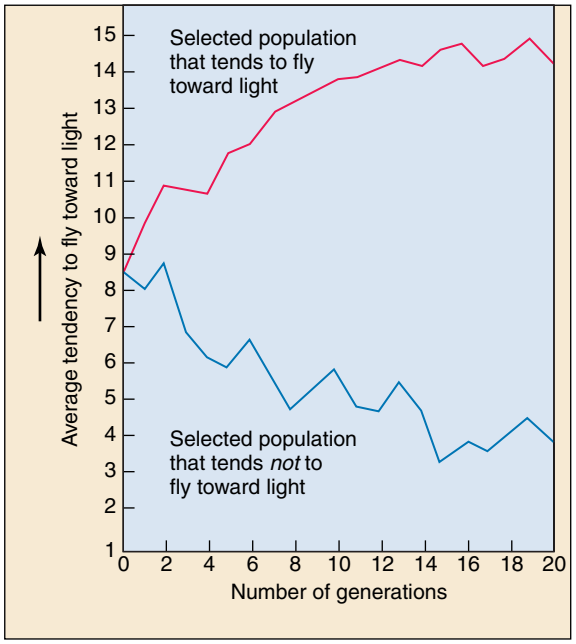

FIGURE 20.14
Directional selection for phototropism in *Drosophila*. In generation after generation, individuals of the fly *Drosophila* were selectively bred to obtain two populations. When flies with a strong tendency to fly toward light (positive phototropism) were used as parents for the next generation, their offspring had a greater tendency to fly toward light (top curve). When flies that tended *not* to fly toward light were used as parents for the next generation, their offspring had an even greater tendency not to fly toward light (bottom curve).

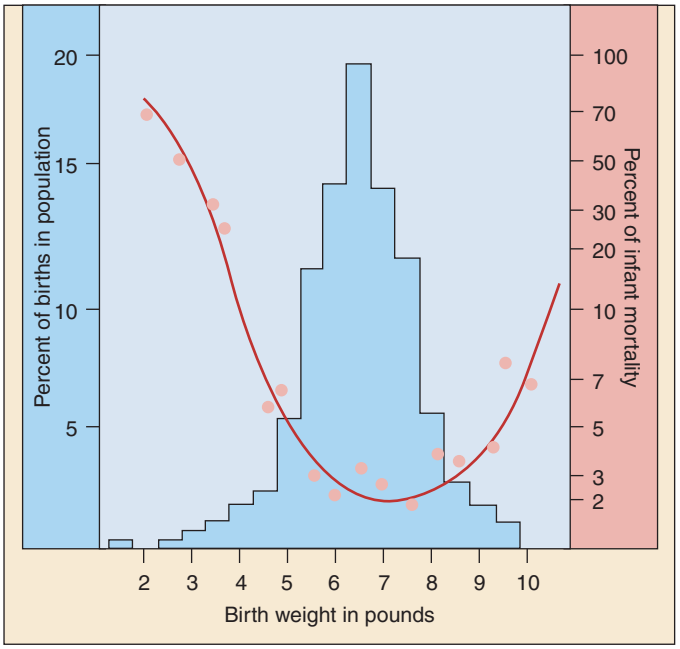

FIGURE 20.15
Stabilizing selection for birth weight in human beings. The death rate among babies (red curve; right *y*-axis) is lowest at an intermediate birth weight; both smaller and larger babies have a greater tendency to die than those around the optimum weight (blue area; left *y*-axis) of between 7 and 8 pounds.

Directional Selection

When selection acts to eliminate one extreme from an array of phenotypes (figure 20.13*b*), the genes promoting this extreme become less frequent in the population. Thus, in the *Drosophila* population illustrated in figure 20.14, the elimination of flies that move toward light causes the population to contain fewer individuals with alleles promoting such behavior. If you were to pick an individual at random from the new fly population, there is a smaller chance it would spontaneously move toward light than if you had selected a fly from the old population. Selection has changed the population in the direction of lower light attraction. This form of selection is called **directional selection.**

Stabilizing Selection

When selection acts to eliminate *both* extremes from an array of phenotypes (figure 20.13*c*), the result is to increase the frequency of the already common intermediate type. In effect, selection is operating to prevent change away from this middle range of values. Selection does not change the most common phenotype of the population, but rather makes it even more common by eliminating extremes. Many examples are known. In humans, infants with intermediate weight at birth have the highest survival rate (figure 20.15). In ducks and chickens, eggs of intermediate weight have the highest hatching success. This form of selection is called **stabilizing selection.**

Components of Fitness

Natural selection occurs when individuals with one phenotype leave more surviving offspring in the next generation than individuals with an alternative phenotype. Evolutionary biologists quantify reproductive success as **fitness,** the number of surviving offspring left in the next generation. Although selection is often characterized as "survival of the fittest," differences in survival are only one component of fitness. Even if no differences in survival occur, selection may operate if some individuals are more successful than others in attracting mates. In many territorial animal species, large males mate with many females and small mates rarely get to mate. In addition, the number of offspring produced per mating is also important. Large female frogs and fish lay more eggs than smaller females and thus may leave more offspring in the next generation.

Selection on traits affected by many genes can favor both extremes of the trait, or intermediate values, or only one extreme.

Limits to What Selection Can Accomplish

Although selection is perhaps the most powerful of the five principal agents of genetic change, there are limits to what it can accomplish. These limits arise because alternative alleles may interact in different ways with other genes and because alleles often affect multiple aspects of the phenotype (the phenomena of epistasis and pleiotropy discussed in chapter 13). These interactions tend to set limits on how much a phenotype can be altered. For example, selecting for large clutch size in barnyard chickens eventually leads to eggs with thinner shells that break more easily. For this reason, we do not have gigantic cattle that yield twice as much meat as our leading strains, chickens that lay twice as many eggs as the best layers do now, or corn with an ear at the base of every leaf, instead of just at the base of a few leaves.

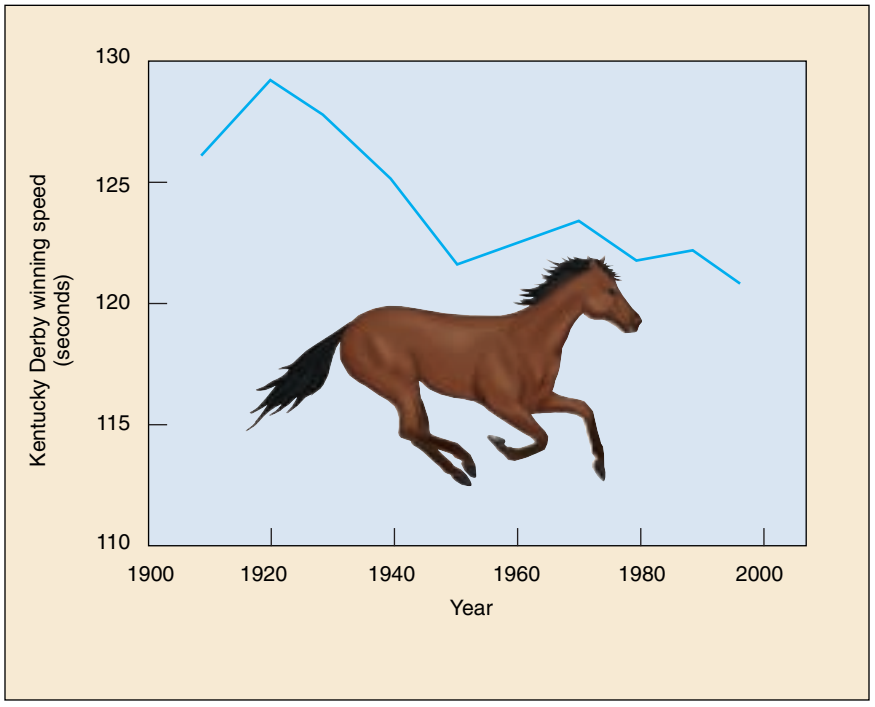

FIGURE 20.16
Selection for increased speed in racehorses is no longer effective. Kentucky Derby winning speeds have not improved significantly since 1950.

Evolution Requires Genetic Variation

Over 80% of the gene pool of the thoroughbred horses racing today goes back to 31 known ancestors from the late eighteenth century. Despite intense directional selection on thoroughbreds, their performance times have not improved for the last 50 years (figure 20.16). Years of intense selection presumably have removed variation from the population at a rate greater than it could be replenished by mutation such that now no genetic variation remains and evolutionary change is not possible.

In some cases, phenotypic variation for a trait may never have had a genetic basis. The compound eyes of insects are made up of hundreds of visual units, termed ommatidia. In some individuals, the left eye contains more ommatidia than the right eye. However, despite intense selection in the laboratory, scientists have never been able to produce a line of fruit flies that consistently have more ommatidia in the left eye than in the right. The reason is that separate genes do not exist for the left and right eyes. Rather, the same genes affect both eyes, and differences in the number of ommatidia result from differences that occur as the eyes are formed in the development process (figure 20.17). Thus, despite the existence of phenotypic variation, no genetic variation is available for selection to favor.

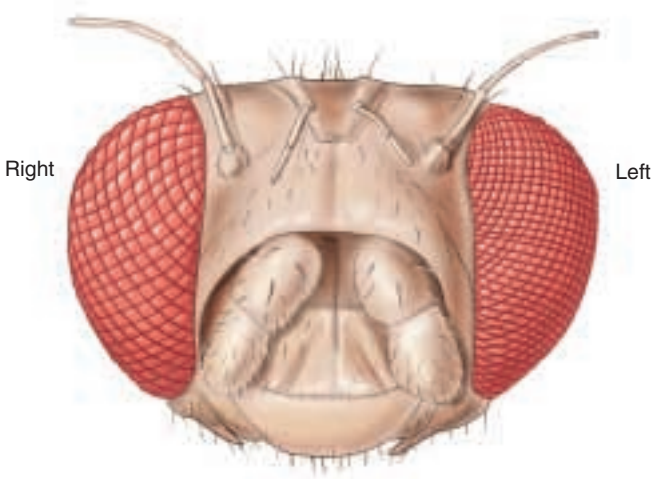

FIGURE 20.17
Phenotypic variation in insect ommatidia. In some individuals, the number of ommatidia in the left eye is greater than the number in the right eye. However, this difference is not genetically based; developmental processes cause the difference.

Selection against Rare Alleles

A second factor limits what selection can accomplish: selection acts only on phenotypes. For this reason, selection does not operate efficiently on rare recessive alleles, simply because there is no way to select against them unless they come together as homozygotes. For example, when a recessive allele *a* is present at a frequency *q* equal to 0.2, only four out of a hundred individuals (q^2) will be double recessive and display the phenotype associated with this allele (figure 20.18). For lower allele frequencies, the effect is even more dramatic: if the frequency in the population of the recessive allele $q = 0.01$, the frequency of recessive homozygotes in that population will be only 1 in 10,000.

The fact that selection acts on phenotypes rather than genotypes means that selection against undesirable genetic traits in humans or domesticated animals is difficult unless the heterozygotes can also be detected. For example, if a particular recessive allele *r* ($q = 0.01$) was considered undesirable, and none of the homozygotes for this allele were allowed to breed, it would take 1000 generations, or about 25,000 years in humans, to lower the allele frequency by half to 0.005. At this point, after 25,000 years of work, the frequency of homozygotes would still be 1 in 40,000, or 25% of what it was initially.

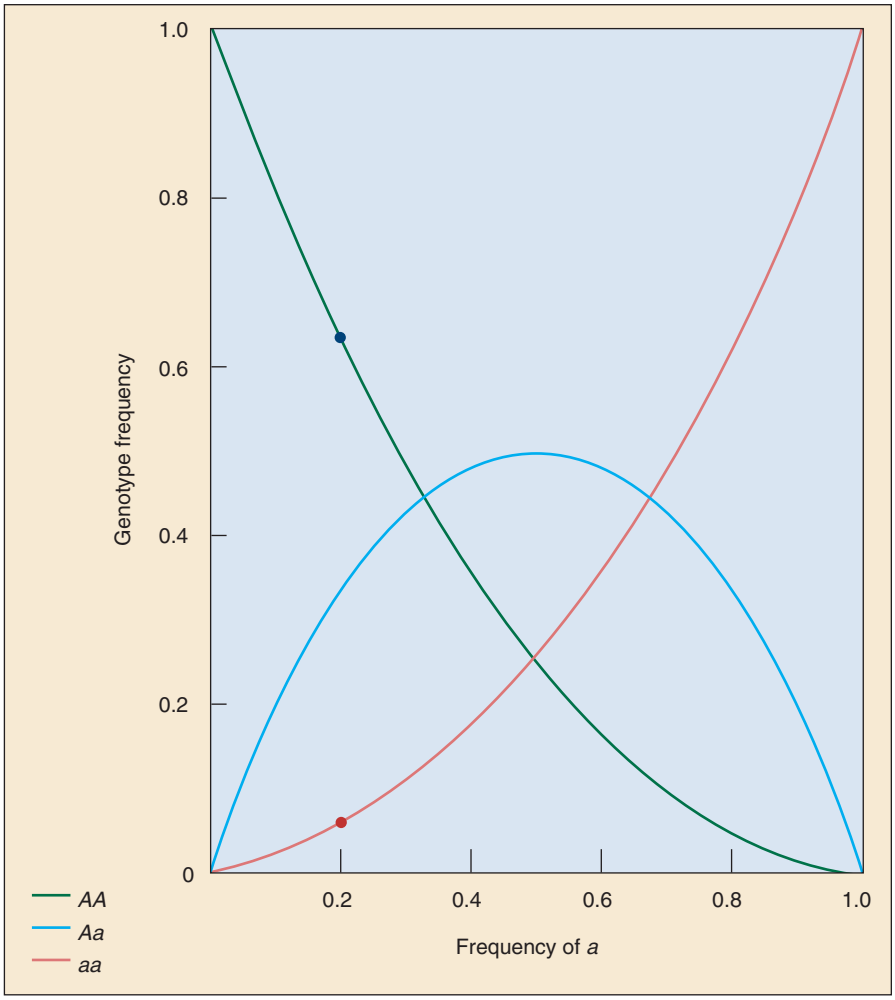

FIGURE 20.18
The relationship between allele frequency and genotype frequency. If allele *a* is present at a frequency of 0.2, the double recessive genotype *aa* is only present at a frequency of 0.04. In other words, only 4 in 100 individuals will have a homozygous recessive genotype, while 64 in 100 will have a homozygous dominant genotype.

Selection in Laboratory Environments

One way to assess the action of selection is to carry out artificial selection in the laboratory. Strains that are genetically identical except for the gene subject to selection can be crossed so that the possibility of linkage disequilibrium does not confound the analysis. Populations of bacteria provide a particularly powerful tool for studying selection in the laboratory because bacteria have a short generation time (less than an hour) and can be grown in huge numbers in growth vats called chemostats. In pioneering studies, Dan Hartl and coworkers backcrossed bacteria with different alleles of the enzyme 6-PGD into a homogeneous genetic background, and then compared the growth of the different strains when they were fed only gluconate, the enzyme's substrate. Hartl found that all of the alleles grew at the same rate! The different alleles were thus selectively neutral in a normal genetic background. However, when Hartl disabled an alternative biochemical pathway for the metabolism of gluconate, so that only 6-PGD mediated the utilization of this sole source of carbon, he obtained very different results: several alleles were markedly superior to others. Selection was clearly able to operate on these alleles, but only under certain conditions.

The ability of selection to produce evolutionary change is hindered by a variety of factors, including multiple effects of single genes, gene interactions, and lack of genetic variation. Moreover, selection can only eliminate rare recessive alleles very slowly.

Chapter 20

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Summary | Questions | Media Resources

20.1 Genes vary in natural populations.

- Evolution is best defined as "descent with modification."
- Darwin's primary insight was to propose that evolutionary change resulted from the operation of natural selection.
- By the 1860s, natural selection was widely accepted as the correct explanation for the process of evolution. The field of evolution did not progress much further, however, until the 1920s because of the lack of a suitable explanation of how hereditary traits are transmitted.
- Invertebrates and outcrossing plants are often heterozygous at about 12 to 15% of their loci; the corresponding value for vertebrates is about 4 to 8%.

1. What is the difference between natural selection and evolution?

2. What is adaptation? How does it fit into Darwin's concept of evolution?

3. What is genetic polymorphism? What has polymorphism to do with evolution?

- Scientists on Science: from Butterflies to Global Preservation
- Student Research: Cotton Boll Weevil

20.2 Why do allele frequencies change in populations?

- Studies of how allele frequencies shift within populations allow investigators to study evolution in action.
- Meiosis does not alter allele frequencies within populations. Unless selection or some other force acts on the genes, the frequencies of their alleles remain unchanged from one generation to the next.
- A variety of processes can lead to evolutionary change within a population, including genetic drift, inbreeding, gene flow, and natural selection.
- For evolution to occur by natural selection, three conditions must be met: 1. variation must exist in the population; 2. the variation must have a genetic basis; and 3. variation must be related to the number of offspring left in the next generation.
- Natural selection can usually overpower the effects of genetic drift, except in very small populations.
- Natural selection can overwhelm the effects of gene flow in some cases, but not in others.

4. Given that allele A is present in a large random-mating population at a frequency of 54 per 100 individuals, what is the proportion of individuals in that population expected to be heterozygous for the allele? homozygous dominant? homozygous recessive?

5. Why does the founder effect have such a profound influence on a population's genetic makeup? How does the bottleneck effect differ from the founder effect?

6. What effect does inbreeding have on allele frequency? Why is marriage between close relatives discouraged?

- Hardy Weinberg Equilibrium

- Activity: Natural Selection
- Activity: Allele Frequencies
- Activity: Genetic Drift
- Types of Selection
- Evolutionary Variation
- Other Processes of Evolution
- Adaptation

20.3 Selection can act on traits affected by many genes.

- Directional selection acts to eliminate one extreme from an array of phenotypes; stabilizing selection acts to eliminate *both* extremes; and disruptive selection acts to eliminate rather than to favor the intermediate type.
- Natural selection is not all powerful; genetic variation is required for natural selection to produce evolutionary change.

7. Define *selection.* How does it alter allele frequencies? What are the three types of selection? Give an example of each.

8. Why are there limitations to the success of selection?

- Book Review: *The Evolution of Jane* by Schine