© Mark Moffett/Foto Natura/Minden Pictures

Phenotypic variation. The frog *Dendrobates pumilio* exhibits dramatic color variation in populations that inhabit the Bocas del Toro Islands, Panama.

Study Plan

20 Microevolution: Genetic Changes within Populations

Why It Matters

On November 28, 1942, at the height of American involvement in World War II, a disastrous fire killed more than 400 people in Boston's Cocoanut Grove nightclub. Many more would have died later but for a new experimental drug, penicillin. A product of *Penicillium* mold, penicillin fought the usually fatal infections of *Staphylococcus aureus,* a bacterium that enters the body through damaged skin. Penicillin was the first antibiotic drug based on a naturally occurring substance that kills bacteria.

Until the disaster at the Cocoanut Grove, the production and use of penicillin had been a closely guarded military secret. But after its public debut, the pharmaceutical industry hailed penicillin as a wonder drug, promoting its use for the treatment of the many diseases caused by infectious microorganisms. Penicillin became widely available as an over-the-counter remedy, and Americans dosed themselves with it, hoping to cure all sorts of ills **(Figure 20.1).** But in 1945, Alexander Fleming, the scientist who discovered penicillin, predicted that some bacteria could survive low doses, and that the offspring of those germs would be more resistant to its effects. In 1946—just 4 years after penicillin's use in Boston—14% of the *Staphylococcus* strains

The Advertising Archives, London

Figure 20.1
Selling penicillin. This ad, from a 1944 issue of *Life* magazine, credits penicillin with saving the lives of wounded soldiers.

isolated from patients in a London hospital were resistant. By 1950, more than half the strains were resistant.

Scientists and physicians have discovered numerous antibiotics since the 1940s, and many strains of bacteria have developed resistance to these drugs. In fact, according to the Centers for Disease Control and Prevention, between 30,000 and 40,000 Americans die each year from infection by antibiotic-resistant bacteria.

How do bacteria become resistant to antibiotics? The genomes of bacteria—like those of all other organisms—vary among individuals, and some bacteria have genetic traits that allow them to withstand attack by antibiotics. When we administer antibiotics to an infected patient, we create an environment favoring bacteria that are even slightly resistant to the drug. The surviving bacteria reproduce, and resistant microorganisms—along with the genes that confer antibiotic resistance—become more common in later generations. In other words, bacterial strains adapt to antibiotics through the evolutionary process of selection. Our use of antibiotics is comparable to artificial selection by plant and animal breeders (see Chapter 19), but when we use antibiotics, we inadvertently select for the success of organisms that we are trying to eradicate.

The evolution of antibiotic resistance in bacteria is an example of **microevolution**, which is a heritable change in the genetics of a population. A **population** of organisms includes all the individuals of a single species that live together in the same place and time. Today, when scientists study microevolution, they analyze variation—the differences between individuals—in natural populations and determine how and why these variations are inherited. Darwin recognized the importance of heritable variation within populations; he also realized that natural selection can change the pattern of variation in a population from one generation to the next. Scientists have since learned that microevolutionary change results from several processes, not just natural selection, and that sometimes these processes counteract each other.

In this chapter, we first examine the extensive variation that exists within natural populations. We then take a detailed look at the most important processes that alter genetic variation within populations, causing microevolutionary change. Finally, we consider how microevolution can fine-tune the functioning of populations within their environments.

20.1 Variation in Natural Populations

In some species, individuals vary dramatically in appearance; but in most species, the members of a population look pretty much alike **(Figure 20.2).** Even those that look alike, such as the *Cerion* snails on the right in Figure 20.2, are not identical, however. With a scale and ruler, you could detect differences in their weight as well as in

a. European garden snails

George Bernard/Foto Natura/Photo Researchers, Inc.

b. Bahaman land snails

Timothy A. Pearce, Ph.D./Section of Mollusks/ Carnegie Museum of Natural History. Photograph by Mindy McNaugher

Figure 20.2
Phenotypic variation. **(a)** Shells of the European garden snail *(Cepaea nemoralis)* from a population in Scotland vary considerably in appearance. **(b)** By contrast, shells of *Cerion christophei* from a population in the Bahamas look very similar.

the length and diameter of their shells. With suitable techniques, you could also document variations in their individual biochemistry, physiology, internal anatomy, and behavior. All of these are examples of **phenotypic variation**, differences in appearance or function that are passed from generation to generation.

Evolutionary Biologists Describe and Quantify Phenotypic Variation

Darwin's theory recognized the importance of heritable phenotypic variation, and today, microevolutionary studies often begin by assessing phenotypic variation within populations. Most characters exhibit **quantitative variation:** individuals differ in small, incremental ways. If you weighed everyone in your biology class, for example, you would see that weight varies almost continuously from your lightest to your heaviest classmate. Humans also exhibit quantitative variation in the length of their toes, the number of hairs on their heads, and their height, as discussed in Chapter 12.

We usually display data on quantitative variation in a bar graph or, if the sample is large enough, as a curve **(Figure 20.3).** The width of the curve is proportional to the variability—the amount of variation—among individuals, and the *mean* describes the average value of the character. As you will see shortly, natural selection often changes the mean value of a character or its variability within populations.

Other characters, like those Mendel studied (see Section 12.1), exhibit **qualitative variation:** they exist in two or more discrete states, and intermediate forms are often absent. Snow geese, for example, have *either* blue *or* white feathers **(Figure 20.4).** The existence of discrete variants of a character is called a **polymorphism** (*poly* = many; *morphos* = form); we describe such traits as *polymorphic*. The *Cepaea nemoralis* snail shells in Figure 20.2a are polymorphic in background color, number of stripes, and color of stripes. Biochemical polymorphisms, like the human A, B, AB, and O blood groups (described in Section 12.2), are also common.

We describe phenotypic polymorphisms quantitatively by calculating the percentage or *frequency* of each trait. For example, if you counted 123 blue snow geese and 369 white ones in a population of 492 geese, the frequency of the blue phenotype would be 123/492 or 0.25, and the frequency of the white phenotype would be 369/492 or 0.75.

Phenotypic Variation Can Have Genetic and Environmental Causes

Phenotypic variation within populations may be caused by genetic differences between individuals, by differences in the environmental factors that individuals experience, or by an interaction between genetics and the environment. As a result, genetic and phenotypic variations may not be perfectly correlated. Under some circumstances, organisms with different genotypes exhibit the same phenotype. For example, the black coloration of some rock pocket mice from Arizona is caused by certain mutations in the *Mc1r* gene (see Section 1.2); but black mice from New Mexico do not share those mutations—that is, they have different genotypes—even though they exhibit the same phenotype. On the other hand, organisms with the same genotype sometimes exhibit different phenotypes. For

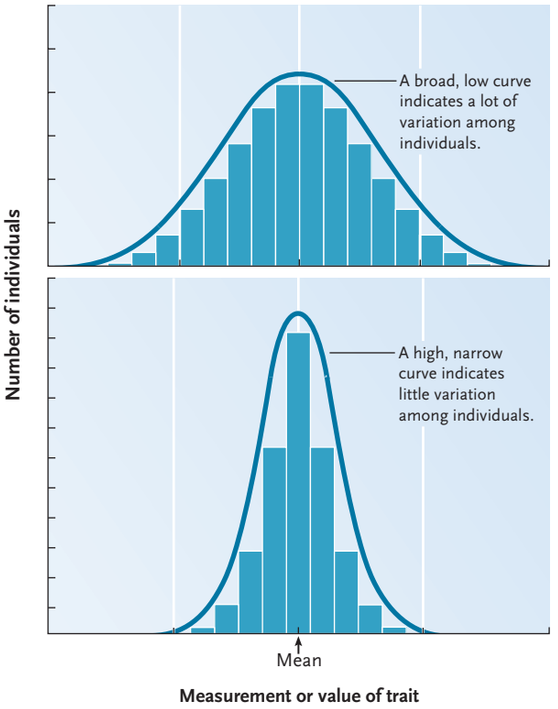


Figure 20.3
Quantitative variation. Many traits vary continuously among members of a population, and a bar graph of the data often approximates a bell-shaped curve. The mean defines the average value of the trait in the population, and the width of the curve is proportional to the variability among individuals.

Figure 20.4
Qualitative variation. Individual snow geese *(Chen caerulescens)* are either blue or white. Although both colors are present in many populations, geese tend to associate with others of the same color.

Figure 20.5
Environmental effects on phenotype. Soil acidity affects the expression of the gene controlling flower color in the common garden plant *Hydrangea macrophylla*. When grown in acid soil, it produces deep blue flowers. In neutral or alkaline soil, its flowers are bright pink.

example, the acidity of soil influences flower color in some plants **(Figure 20.5).**

Knowing whether phenotypic variation is caused by genetic differences, environmental factors, or an interaction of the two is important because *only genetically based variation is subject to evolutionary change*. Moreover, knowing the causes of phenotypic variation has important practical applications. Suppose, for example, that one field of wheat produced more grain than another. If a difference in the availability of nutrients or water caused the difference in yield, a farmer might choose to fertilize or irrigate the less productive field. But if the difference in productivity resulted from genetic differences between plants in the two fields, a farmer might plant only the more productive genotype. Because environmental factors can influence the expression of genes, an organism's phenotype is frequently the product of an interaction between its genotype and its environment. In our hypothetical example, the farmer may maximize yield by fertilizing and irrigating the better genotype of wheat.

How can we determine whether phenotypic variation is caused by environmental factors or by genetic differences? We can test for an environmental cause experimentally by changing one environmental variable and measuring the effects on genetically similar subjects. You can try this yourself by growing some cuttings from an ivy plant in shade and other cuttings from the same plant in full sun. Although they all have the same genotype, the cuttings grown in sun will produce smaller leaves and shorter stems.

Breeding experiments can demonstrate the genetic basis of phenotypic variation. For example, Mendel inferred the genetic basis of qualitative traits, such as flower color in peas, by crossing plants with different phenotypes. Moreover, traits that vary quantitatively will respond to artificial selection only if the variation has some genetic basis. For example, researchers observed that individual house mice *(Mus domesticus)* differ in activity levels, as measured by how much they use an exercise wheel and how fast they run. John G. Swallow, Patrick A. Carter, and Theodore Garland, Jr., then at the University of Wisconsin at Madison, used artificial selection to produce lines of mice that exhibit increased wheel-running behavior, demonstrating that the observed differences in these two aspects of activity level have a genetic basis **(Figure 20.6).**

Breeding experiments are not always practical, however, particularly for organisms with long generation times. Ethical concerns also render these techniques unthinkable for humans. Instead, researchers sometimes study the inheritance of particular traits by analyzing genealogical pedigrees, as discussed in Section 13.2, but this approach often provides poor results for analyses of complex traits.

Several Processes Generate Genetic Variation

Genetic variation, the raw material molded by microevolutionary processes, has two potential sources: the production of new alleles and the rearrangement of existing alleles. Most new alleles probably arise from small scale mutations in DNA (described later in this chapter). The rearrangement of existing alleles into new combinations can result from larger scale changes in chromosome structure or number and from several forms of genetic recombination, including crossing over between homologous chromosomes during meiosis, the independent assortment of nonhomologous chromosomes during meiosis, and random fertilizations between genetically different sperm and eggs.

The shuffling of *existing* alleles into new combinations can produce an extraordinary number of novel genotypes and phenotypes in the next generation. By one estimate, more than 10^{600} combinations of alleles are possible in human gametes, yet there are fewer than 10^{10} humans alive today. So unless you have an identical twin, it is extremely unlikely that another person with your genotype has ever lived or ever will.

Populations Often Contain Substantial Genetic Variation

How much genetic variation actually exists within populations? In the 1960s, evolutionary biologists began to use gel electrophoresis (see Figure 18.7) to identify biochemical polymorphisms in diverse organisms. This technique separates two or more forms of a given protein if they differ significantly in shape, mass, or net electrical charge. The identification of a protein polymorphism allows researchers to infer genetic variation at the locus coding for that protein.

Figure 20.6 Experimental Research

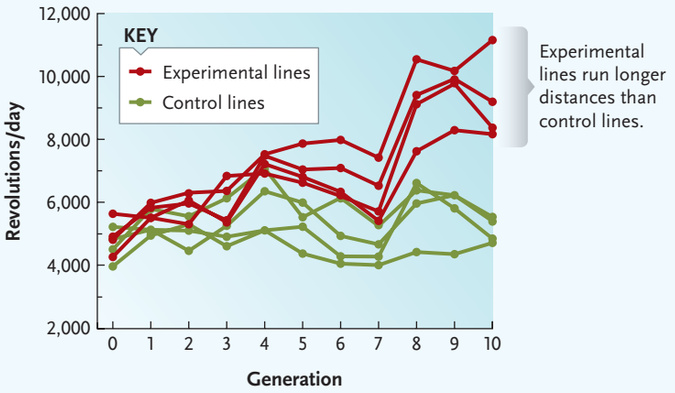
Using Artificial Selection to Demonstrate That Activity Level in Mice Has a Genetic Basis

QUESTION: Do observed differences in activity level among house mice have a genetic basis?

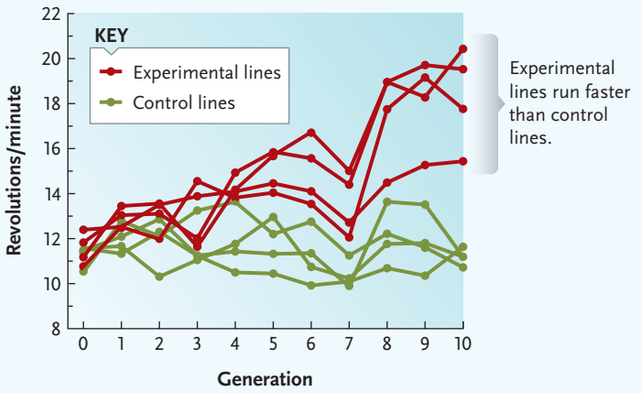
EXPERIMENT: Swallow, Carter, and Garland knew that a phenotypic character responds to artificial selection only if it has a genetic, rather than an environmental, basis. In an experiment with house mice *(Mus domesticus)*, they selected for the phenotypic character of increased wheel-running activity. In four experimental lines, they bred those mice that ran the most. Four other lines, in which breeders were selected at random with respect to activity level, served as controls.

RESULTS: After 10 generations of artificial selection, mice in the experimental lines ran longer distances and ran faster than mice in the control lines. Thus, artificial selection on wheel-running activity in house mice increased **(a)** the distance that mice run per day and **(b)** their average speed. The data illustrate responses of females in four experimental lines and four control lines. Males showed similar responses.

a. Distance run



b. Average speed



CONCLUSION: Because two measures of activity level responded to artificial selection, researchers concluded that variation in this behavioral character has a genetic basis.

Researchers discovered much more genetic variation than anyone had imagined. For example, nearly half the loci surveyed in many populations of plants and invertebrates are polymorphic. Moreover, gel electrophoresis actually underestimates genetic variation because it doesn't detect different amino acid substitutions if the proteins for which they code migrate at the same rate.

Advances in molecular biology now allow scientists to survey genetic variation directly, and researchers have accumulated an astounding knowledge of the structure of DNA and its nucleotide sequences. In general, studies of chromosomal and mitochondrial DNA suggest that every locus exhibits some variability in its nucleotide sequence. The variability is apparent in comparisons of individuals from a single population, populations of one species, and related species. However, some variations detected in the protein-coding regions of DNA may not affect phenotypes because, as explained on page 426, they do not change the amino acid sequences of the proteins for which the genes code.

Study Break

1. If a population of skunks includes some individuals with stripes and others with spots, would you describe the variation as quantitative or qualitative?
2. In the experiment on house mice described in Figure 20.6, how did researchers demonstrate that variations in activity level had a genetic basis?
3. What factors contribute to phenotypic variation in a population?

20.2 Population Genetics

To predict how certain factors may influence genetic variation, population geneticists first describe the genetic structure of a population. They then create hypotheses, which they formalize in mathematical mod-

els, to describe how evolutionary processes may change the genetic structure under specified conditions. Finally, researchers test the predictions of these models to evaluate the ideas about evolution that are embodied within them.

All Populations Have a Genetic Structure

Populations are made up of individuals, each with its own genotype. In diploid organisms, which have pairs of homologous chromosomes, an individual's genotype includes two alleles at every gene locus. The sum of all alleles at all gene loci in all individuals is called the population's **gene pool.**

To describe the structure of a gene pool, scientists first identify the genotypes in a representative sample and calculate **genotype frequencies,** the percentages of individuals possessing each genotype. Knowing that each diploid organism has two alleles (either two copies of the same allele or two different alleles) at each gene locus, a scientist can then calculate **allele frequencies,** the relative abundances of the different alleles. For a locus with two alleles, scientists use the symbol p to identify the frequency of one allele, and q the frequency of the other.

The calculation of genotype and allele frequencies for the two alleles at the gene locus governing flower color in snapdragons (genus *Antirrhinum*) is straightforward **(Table 20.1).** This locus is easy to study because it exhibits incomplete dominance (see Section 12.2). Individuals that are homozygous for the C^R allele (C^RC^R) have red flowers; those homozygous for the C^W allele (C^WC^W) have white flowers; and heterozygotes (C^RC^W) have pink flowers. Genotype frequencies represent how the C^R and C^W alleles are distributed among individuals. In this example, examination of the plants reveals that 45% of individuals have the C^RC^R genotype, 50% have the heterozygous C^RC^W genotype, and the remaining 5% have the C^WC^W genotype. Allele frequencies represent the commonness or rarity of each allele in the gene pool. As calculated in the table, 70% of the alleles in the population are C^R and 30% are C^W. Remember that for a gene locus with two alleles, there are three genotype frequencies, but only two allele frequencies (p and q). The sum of the three genotype frequencies must equal 1; so must the sum of the two allele frequencies.

The Hardy-Weinberg Principle Is a Null Model That Defines How Evolution Does Not Occur

When designing experiments, scientists often use control treatments to evaluate the effect of a particular factor: the control tells us what we would see if the experimental treatment had no effect. As you may recall from the hypothetical example presented in Chapter 1 (see Figure 1.14), to determine whether fertilizer has an effect on plant growth, you must compare the growth of fertilized plants (the experimental treatment) to the growth of plants that received no fertilizer (the control treatment). However, in studies that use observational rather than experimental data, there is often no suitable control. In such cases, investigators develop conceptual models, called **null models,** which predict what they would see if a particular factor had no effect. Null models serve as theoretical reference points against which observations can be evaluated.

Early in the twentieth century, geneticists were puzzled by the persistence of recessive traits because they assumed that natural selection replaced recessive or rare alleles with dominant or common ones. An English mathematician, G. H. Hardy, and a German physician, Wilhelm Weinberg, tackled this problem independently in 1908. Their analysis, now known as the

Table 20.1 **Calculation of Genotype Frequencies and Allele Frequencies for the Snapdragon Flower Color Locus**

Because each diploid individual has two alleles at each gene locus, the entire sample of 1000 individuals has a total of 2000 alleles at the *C* locus.

Flower Color Phenotype	Genotype	Number of Individuals	Genotype Frequency[1]	Total Number of C^R Alleles[2]	Total Number of C^W Alleles[2]
Red	C^RC^R	450	450/1000 = 0.45	2 × 450 = 900	0 × 450 = 0
Pink	C^RC^W	500	500/1000 = 0.50	1 × 500 = 500	1 × 500 = 500
White	C^WC^W	50	50/1000 = 0.05	0 × 50 = 0	2 × 50 = 100
	Total	1000	0.45 + 0.50 + 0.05 = 1.0	1400	600

Calculate allele frequencies using the total of 1400 + 600 = 2000 alleles in the sample:

p = frequency of C^R allele = 1400/2000 = 0.7
q = frequency of C^W allele + 600/2000 = 0.3
$p + q = 0.7 + 0.3 = 1.0$

[1]Genotype frequency = the number of individuals possessing a particular genotype divided by the total number of individuals in the sample.
[2]Total number of C^R or C^W alleles = the number of C^R or C^W alleles present in one individual with a particular genotype multiplied by the number of individuals with that genotype.

Hardy-Weinberg principle, specifies the conditions under which a population of diploid organisms achieves **genetic equilibrium**, the point at which neither allele frequencies nor genotype frequencies change in succeeding generations. Their work also showed that dominant alleles need not replace recessive ones, and that the shuffling of genes in sexual reproduction does not in itself cause the gene pool to change.

The Hardy-Weinberg principle is a mathematical model that describes how genotype frequencies are established in sexually reproducing organisms. According to this model, genetic equilibrium is possible only if *all* of the following conditions are met:

1. No mutations are occurring.
2. The population is closed to migration from other populations.
3. The population is infinite in size.
4. All genotypes in the population survive and reproduce equally well.
5. Individuals in the population mate randomly with respect to genotypes.

If the conditions of the model are met, the allele frequencies of the population will never change, and the genotype frequencies will stop changing after one generation. In short, under these restrictive conditions, microevolution will *not* occur (see *Focus on Research*). The Hardy-Weinberg principle is thus a null model that serves as a reference point for evaluating the circumstances under which evolution *may* occur.

If a population's genotype frequencies do not match the predictions of this model or if its allele frequencies change over time, microevolution may be occurring. Determining which of the model's conditions are not met is a first step in understanding how and why the gene pool is changing. Natural populations never fully meet all five requirements simultaneously, but they often come pretty close.

Study Break

1. What is the difference between the genotype frequencies and the allele frequencies in a population?
2. Why is the Hardy-Weinberg principle considered a null model of evolution?
3. If the conditions of the Hardy-Weinberg principle are met, when will genotype frequencies stop changing?

20.3 The Agents of Microevolution

A population's allele frequencies will change over time if conditions of the Hardy-Weinberg model are violated. The processes that foster microevolutionary change—which include mutation, gene flow, genetic drift, natural selection, and nonrandom mating—are summarized in **Table 20.2.**

Table 20.2 Agents of Microevolutionary Change

Agent	Definition	Effect on Genetic Variation
Mutation	A heritable change in DNA	Introduces new genetic variation into population
Gene flow	Change in allele frequencies as individuals join a population and reproduce	May introduce genetic variation from another population
Genetic drift	Random changes in allele frequencies caused by chance events	Reduces genetic variation, especially in small populations; can eliminate alleles
Natural selection	Differential survivorship or reproduction of individuals with different genotypes	One allele can replace another or allelic variation can be preserved
Nonrandom mating	Choice of mates based on their phenotypes and genotypes	Does not directly affect allele frequencies, but usually prevents genetic equilibrium

Mutations Create New Genetic Variations

A **mutation** is a spontaneous and heritable change in DNA. Mutations are rare events; during any particular breeding season, between one gamete in 100,000 and one in 1 million will include a new mutation at a particular gene locus. New mutations are so infrequent, in fact, that they exert little or no immediate effect on allele frequencies in most populations. But over evolutionary time scales, their numbers are significant—mutations have been accumulating in biological lineages for billions of years. And because it is a mechanism through which entirely new genetic variations arise, *mutation is a major source of heritable variation.*

For most animals, only mutations in the germ line (the cell lineage that produces gametes) are heritable; mutations in other cell lineages have no direct effect on the next generation. In plants, however, mutations may occur in meristem cells, which eventually produce flowers as well as nonreproductive structures (see Chapter 31); in such cases, a mutation may be passed to the next generation and ultimately influence the gene pool.

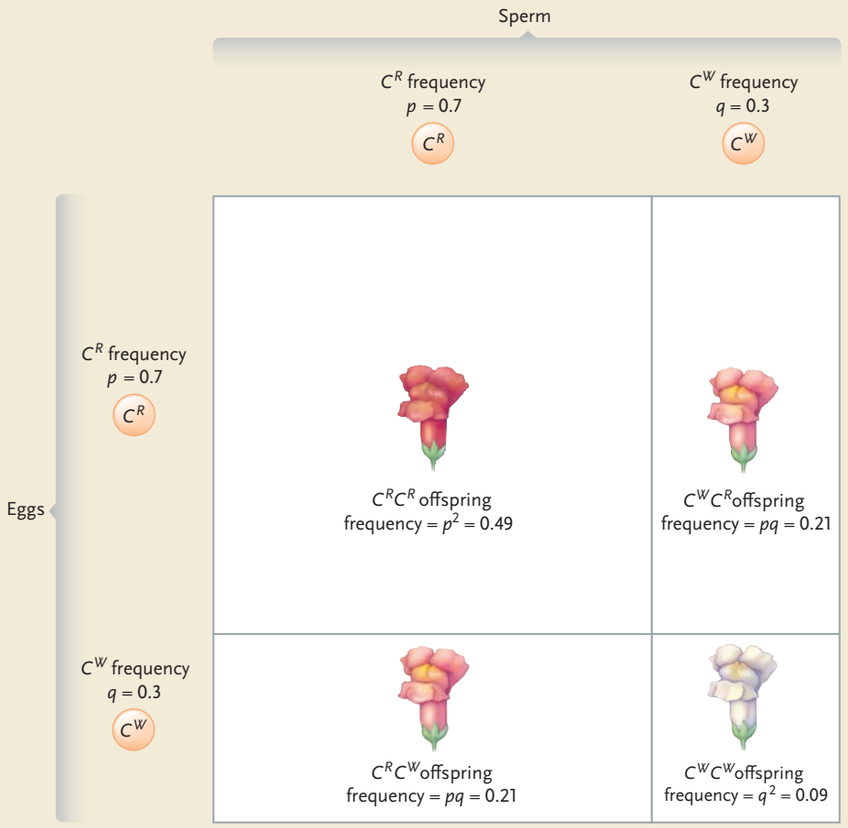
Deleterious mutations alter an individual's structure, function, or behavior in harmful ways. In mammals, for example, a protein called collagen is an essential component of most extracellular structures. Several simple mutations in humans cause forms of Ehlers-Danlos syndrome, a disruption of collagen synthesis that may result in loose skin, weak joints, or sudden death from the rupture of major blood vessels, the colon, or the uterus.

By definition, *lethal mutations* cause the death of organisms carrying them. If a lethal allele is dominant, both homozygous and heterozygous carriers suffer

Focus on Research

Basic Research: Using the Hardy-Weinberg Principle

To see how the Hardy-Weinberg principle can be applied, we will analyze the snapdragon flower color locus, using the hypothetical population of 1000 plants described in Table 20.1. This locus includes two alleles—C^R (with its frequency designated as p) and C^W (with its frequency designated as q)—and three genotypes—homozygous C^RC^R, heterozygous C^RC^W, and homozygous C^WC^W. Table 20.1 lists the number of plants with each genotype: 450 have red flowers (C^RC^R), 500 have pink flowers (C^RC^W), and 50 have white flowers (C^WC^W). It also shows the calculation of both the genotype frequencies (C^RC^R = 0.45, C^RC^W = 0.50, and C^WC^W = 0.05) and the allele frequencies (p = 0.7 and q = 0.3) for the population.



Let's assume for simplicity that each individual produces only two gametes and that both gametes contribute to the production of offspring. This assumption is unrealistic, of course, but it meets the Hardy-Weinberg requirement that all individuals in the population contribute equally to the next generation. In each parent, the two alleles segregate and end up in different gametes:

450 C^RC^R individuals produce → 900 C^R gametes

500 C^RC^W individuals produce → 500 C^R gametes + 500 C^W gametes

50 C^WC^W individuals produce → 100 C^W gametes

You can readily see that 1400 of the 2000 total gametes carry the C^R allele and 600 carry the C^W allele. The frequency of C^R gametes is 1400/2000 or 0.7, which is equal to p; the frequency of C^W gametes is 600/2000 or 0.3, which is equal to q. Thus, the allele frequencies in the gametes are exactly the same as the allele frequencies in the parent generation—it could not be

from its effects; if recessive, it affects only homozygous recessive individuals. A lethal mutation that causes death before the individual reproduces is eliminated from the population.

Neutral mutations are neither harmful nor helpful. Recall from Section 15.1 that in the construction of a polypeptide chain, a particular amino acid can be specified by several different codons. As a result, some DNA sequence changes—especially certain changes at the third nucleotide of the codon—do not alter the amino acid sequence. Not surprisingly, mutations at the third position appear to persist longer in populations than those at the first two positions. Other mutations may change an organism's phenotype without influencing its survival and reproduction. A neutral mutation might even be beneficial later if the environment changes.

Sometimes a change in DNA produces an *advantageous mutation,* which confers some benefit on an individual that carries it. However slight the advantage, natural selection may preserve the new allele and even increase its frequency over time. Once the mutation has been passed to a new generation, other agents of microevolution determine its long-term fate.

Gene Flow Introduces Novel Genetic Variants into Populations

Organisms or their gametes (for example, pollen) sometimes move from one population to another. If the immigrants reproduce, they may introduce novel alleles into the population they have joined. This phenomenon, called **gene flow**, violates the Hardy-Weinberg requirement that populations must be closed to migration.

otherwise because each gamete carries one allele at each locus.

Now assume that these gametes, both sperm and eggs, encounter each other at random. In other words, individuals reproduce without regard to the genotype of a potential mate. We can visualize the process of random mating in the mating table on the left.

We can also describe the consequences of random mating—$(p + q)$ sperm fertilizing $(p + q)$ eggs—with an equation that predicts the genotype frequencies in the offspring generation:

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

If the population is at genetic equilibrium for this locus, p^2 is the predicted frequency of the C^RC^R genotype, $2pq$ the predicted frequency of the C^RC^W genotype, and q^2 the predicted frequency of the C^WC^W genotype. Using the gamete frequencies determined above, we can calculate the predicted genotype frequencies in the next generation:

frequency of C^RC^R =
$$p^2 = (0.7 \times 0.7) = 0.49$$

frequency of C^RC^W =
$$2pq = 2(0.7 \times 0.3) = 0.42$$

frequency of C^WC^W =
$$q^2 = (0.3 \times 0.3) = 0.09$$

Notice that the predicted genotype frequencies in the offspring generation have changed from those in the parent generation: the frequency of heterozygous individuals has decreased, and the frequencies of both types of homozygous individuals have increased. This result occurred because the starting population was *not already* in equilibrium at this gene locus. In other words, the distribution of parent genotypes did not conform to the predicted $p^2 + 2pq + q^2$ distribution.

The 2000 gametes in our hypothetical population produced 1000 offspring. Using the genotype frequencies we just calculated, we can predict how many offspring will carry each genotype:

490 red (C^RC^R)
420 pink (C^RC^W)
90 white (C^WC^W)

In a real study, we would examine the offspring to see how well their numbers match these predictions.

What about the allele frequencies in the offspring? The Hardy-Weinberg principle predicts that they did not change. Let's calculate them and see. Using the method shown in Table 20.1 and the prime symbol (′) to indicate offspring allele frequencies:

$$p' = ([2 \times 490] + 420)/2000 = 1400/2000 = 0.7$$

$$q' = ([2 \times 90] + 420)/2000 = 600/2000 = 0.3$$

You can see from this calculation that the allele frequencies did not change from one generation to the next, even though the alleles were rearranged to produce different proportions of the three genotypes. Thus, the population is now at genetic equilibrium for the flower color locus; neither the genotype frequencies nor the allele frequencies will change in succeeding generations as long as the population meets the conditions specified in the Hardy-Weinberg model.

To verify this, you can calculate the allele frequencies of the gametes for this offspring generation and predict the genotype frequencies and allele frequencies for a third generation. You could continue calculating until you ran out of either paper or patience, but these frequencies will not change.

Researchers use calculations like these to determine whether an actual population is near its predicted genetic equilibrium for one or more gene loci. When they discover that a population is not at equilibrium, they infer that microevolution is occurring and can investigate the factors that might be responsible.

Gene flow is common in some animal species. For example, young male baboons typically move from one local population to another after experiencing aggressive behavior by older males. And many marine invertebrates disperse long distances as larvae carried by ocean currents.

Dispersal agents, such as pollen-carrying wind or seed-carrying animals, are responsible for gene flow in most plant populations. For example, blue jays foster gene flow among populations of oaks by carrying acorns from nut-bearing trees to their winter caches, which may be as much as a mile away **(Figure 20.7).** Transported acorns that go uneaten may germinate and contribute to the gene pool of a neighboring oak population.

Documenting gene flow among populations is not always easy, particularly if it occurs infrequently. Researchers can use phenotypic or genetic markers to

David Neal Parks

W. Carter Johnson

Figure 20.7
Gene flow. Blue jays *(Cyanocitta cristata)* serve as agents of gene flow for oaks (genus *Quercus*) when they carry acorns from one oak population to another. An uneaten acorn may germinate and contribute to the gene pool of the population into which it was carried.

identify immigrants in a population, but they must also demonstrate that immigrants reproduced, thereby contributing to the gene pool of their adopted population. In the San Francisco Bay area, for example, Bay checkerspot butterflies *(Euphydryas editha bayensis)* rarely move from one population to another because they are poor fliers (see Figure 53.16). When adult females do change populations, it is often late in the breeding season, and their offspring have virtually no chance of finding enough food to mature. Thus, many immigrant females do not foster gene flow because they do not contribute to the gene pool of the population they join.

The evolutionary importance of gene flow depends upon the degree of genetic differentiation between populations and the rate of gene flow between them. If two gene pools are very different, a little gene flow may increase genetic variability within the population that receives immigrants, and it will make the two populations more similar. But if populations are already genetically similar, even lots of gene flow will have little effect.

Genetic Drift Reduces Genetic Variability within Populations

Chance events sometimes cause the allele frequencies in a population to change unpredictably. This phenomenon, known as **genetic drift**, has especially dramatic effects on small populations, which clearly violate the Hardy-Weinberg assumption of infinite population size.

A simple analogy clarifies why genetic drift is more pronounced in small populations than in large ones. When individuals reproduce, male and female gametes often pair up randomly, as though the allele in any particular sperm or ovum was determined by a coin toss. Imagine that "heads" specifies the *R* allele and "tails" specifies the *r* allele. If the two alleles are equally common (that is, their frequencies, *p* and *q*, are both equal to 0.5), heads should be as likely an outcome as tails. But if you toss the coin 20 or 30 times to simulate random mating in a small population, you won't often see a 50-50 ratio of heads and tails. Sometimes heads will predominate and sometimes tails will—just by chance. Tossing the coin 500 times to simulate random mating in a somewhat larger population is more likely to produce a 50-50 ratio of heads and tails. And if you tossed the coin 5000 times, you would get even closer to a 50-50 ratio.

Chance deviations from expected results—which cause genetic drift—occur whenever organisms engage in sexual reproduction, simply because their population sizes are not infinitely large. But genetic drift is particularly common in small populations because only a few individuals contribute to the gene pool and because any given allele is present in very few individuals.

Genetic drift generally leads to the loss of alleles and reduced genetic variability. Two general circumstances, population bottlenecks and founder effects, often foster genetic drift.

Population Bottlenecks. On occasion, a stressful factor such as disease, starvation, or drought kills a great many individuals and eliminates some alleles from a population, producing a **population bottleneck.** This cause of genetic drift greatly reduces genetic variation even if the population numbers later rebound.

In the late nineteenth century, for example, hunters nearly wiped out northern elephant seals *(Mirounga angustirostris)* along the Pacific coast of North America **(Figure 20.8).** Since the 1880s, when the species received protected status, the population has increased to more than 30,000, all descended from a group of about 20 survivors. Today the population exhibits no variation in 24 proteins studied by gel electrophoresis. This low level of genetic variation, which is unique among seal species, is consistent with the hypothesis that genetic drift eliminated many alleles when the population experienced the bottleneck.

Founder Effect. When a few individuals colonize a distant locality and start a new population, they carry only a small sample of the parent population's genetic variation. By chance, some alleles may be totally missing from the new population, whereas other alleles that were rare "back home" might occur at relatively high frequencies. This change in the gene pool is called the **founder effect.**

The human medical literature provides some of the best-documented examples of the founder effect. The Old Order Amish, an essentially closed religious community in Lancaster County, Pennsylvania, have an exceptionally high incidence of Ellis–van Creveld syndrome, a genetic disorder caused by a recessive allele. In the homozygous state, the allele produces dwarfism, shortened limbs, and polydactyly (extra fin-

Frans Lanting/Minden Pictures

Figure 20.8
Population bottleneck. Northern elephant seals *(Mirounga angustirostris)* at the Año Nuevo State Reserve in California are descended from a population that was decimated by hunting late in the nineteenth century. In this photo, two large bulls fight to control a harem of females.

Insights from the Molecular Revolution

Genetic Variation Preserved in Humpback Whales

For centuries, hunters slaughtered humpback whales *(Megaptera novaeangliae)* for their meat and oil. By 1966, when an international agreement limited whale hunting, the worldwide population of humpbacks had been reduced to fewer than 5000 individuals. These survivors were distributed among three distinct populations in the North Atlantic, North Pacific, and Southern oceans. Since the hunting agreement was imposed, the populations have recovered to include more than 20,000 individuals.

The derivation of present-day humpback populations from the relatively small number surviving in 1966 is of concern because the population bottleneck may have reduced genetic variability. Such a loss could have adverse effects on the surviving population's reproductive capacity, resistance to disease, and ability to survive unfavorable environmental changes.

How serious was the bottleneck for the surviving humpback whales? A large group of researchers working in Hawaii, the continental United States, Australia, South Africa, Canada, Mexico, and the Dominican Republic set out to answer this question, using molecular techniques to measure the amount of genetic variability in the surviving whale populations.

The researchers chose mitochondrial DNA (mtDNA) for their measurements because it is small, it is easily extracted and identified, and almost all of its variability comes from chance mutations that occur at a steady rate rather than from genetic recombination (see Section 13.5). Except for the few changes produced by mutations since the population bottleneck (which can be estimated from the mutation rate and subtracted from the total), the variability of mtDNA should be the amount remaining from the population that existed before the bottleneck.

Using biopsy darts, the researchers obtained small skin samples from 90 humpback whales distributed among the three oceanic populations. They extracted the mtDNA from the skin samples and amplified it using the polymerase chain reaction (see Figure 18.6). They then isolated a 463-base-pair segment containing the promoters and replication origin for mtDNA, along with spacer sequences. The DNA base sequence was determined for each sample.

The researchers were surprised to find that the mtDNA sequence variation was relatively high in most of their sample, between 76% and 82% of the average variation found in all animal species studied to date. However, a subpopulation of the north Pacific population living near Hawaii showed low genetic variability; in fact, no variability at all was detected in the mtDNA segment of this subpopulation. Why the Hawaiian humpbacks have no variability in the mtDNA segment examined is unclear. One possibility is that this subpopulation originated recently, perhaps during the twentieth century. Information supporting this idea comes from whaling records, which list no sightings or catches of humpbacks in the Hawaiian region during the nineteenth century. Furthermore, the native Hawaiian people have no legends or words describing whales of the humpback type (baleen whales). Perhaps the subpopulation was started by a few whales with the same genetic make-up in the mtDNA region, providing an example of the founder effect.

With the exception of this Hawaiian subpopulation, humpback whales appear to have retained genetic variability comparable to other animals. This retention of variability in the face of near extinction may result from the whales' relatively long generation time. Because they have a potential life span of about 50 years, some individuals that survived the period of commercial hunting are still alive today. The researchers suggest that enough of these long-lived individuals survived to provide a reservoir of variability from the old populations.

These results indicate that the hunting ban came in time to prevent a significant loss of genetic variability in humpback whales. Hopefully, the same is true of other whale species that were hunted nearly to extinction.

gers). Genetic analysis suggests that, although this syndrome affects less than 1% of the Amish in Lancaster County, as many as 13% may be heterozygous carriers of the allele. All of the individuals exhibiting the syndrome are descended from one couple who helped found the community in the mid-1700s.

Conservation Implications. Genetic drift has important implications for conservation biology. By definition, endangered species experience severe population bottlenecks, which result in the loss of genetic variability. Moreover, the small number of individuals available for captive breeding programs may not fully represent a species' genetic diversity. Without such variation, no matter how large a population may become in the future, it will be less resistant to diseases or less able to cope with environmental change.

For example, scientists believe that an environmental catastrophe produced a population bottleneck in the African cheetah *(Acinonyx jubatus)* 10,000 years ago. Cheetahs today are remarkably uniform in genetic make-up. Their populations are highly susceptible to diseases; they also have a high proportion of sperm cell abnormalities and a reduced reproductive capacity. Thus, limited genetic variation, as well as small numbers, threatens populations of endangered species. *Insights from the Molecular Revolution* describes techniques used to determine whether hunting has had the same effect on humpback whales.

Natural Selection Shapes Genetic Variability by Favoring Some Traits over Others

The Hardy-Weinberg model requires all genotypes in a population to survive and reproduce equally well. But as you know from Section 19.2, heritable traits enable some individuals to survive better and reproduce more than others. **Natural selection** is the process by which such traits become more common in subsequent generations. Thus, natural selection violates a requirement of the Hardy-Weinberg equilibrium.

Although natural selection can change allele frequencies, *it is the phenotype of an individual organism, rather than any particular allele, that is successful or not.* When individuals survive and reproduce, their alleles—both favorable and unfavorable—are passed to the next generation. Of course, an organism with harmful or lethal dominant alleles will probably die before reproducing, and all the alleles it carries will share that unhappy fate, even those that are advantageous.

To evaluate reproductive success, evolutionary biologists consider **relative fitness,** the number of surviving offspring that an individual produces compared with the number left by others in the population. Thus, a particular allele will increase in frequency in the next generation if individuals carrying that allele leave *more*

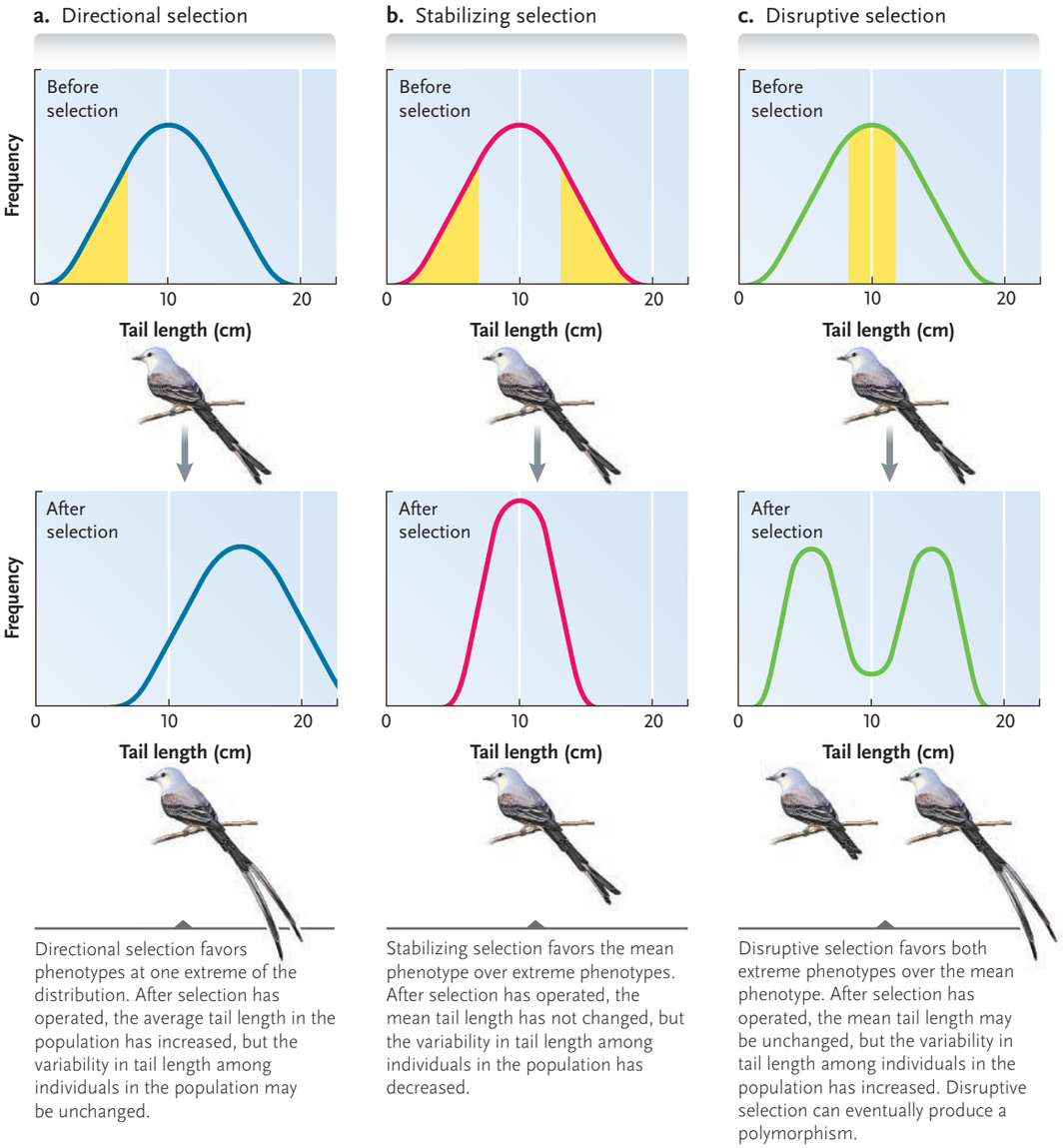


Figure 20.9

Three modes of natural selection. This hypothetical example uses tail length of birds as the quantitative trait subject to selection. The yellow shading in the top graphs indicates phenotypes that natural selection does *not* favor. Notice that the area under each curve is constant because each curve presents the frequencies of all phenotypes in the population. When stabilizing selection **(b)** reduces variability in the trait, the curve becomes higher and narrower.

offspring than individuals carrying other alleles. Differences in the *relative* success of individuals are the essence of natural selection.

Natural selection tests fitness differences at nearly every stage of the life cycle. One plant may be fitter than others in the population because its seeds survive colder conditions, because the arrangement of its leaves captures sunlight more efficiently, or because its flowers are more attractive to pollinators. However, natural selection exerts little or no effect on traits that appear during an individual's postreproductive life. For example, Huntington disease, a dominant-allele disorder that first strikes humans after the age of 40, is not subject to strong selection. Carriers of the disease-causing allele reproduce before the onset of the condition, passing it to the next generation.

Biologists measure the effects of natural selection on phenotypic variation by recording changes in the mean and variability of characters over time (see Figure 20.3). Three modes of natural selection have been identified: directional selection, stabilizing selection, and disruptive selection **(Figure 20.9).**

Directional Selection. Traits undergo **directional selection** when individuals near one end of the phenotypic spectrum have the highest relative fitness. Directional selection shifts a trait away from the existing mean and toward the favored extreme (see Figure 20.9a). After selection, the trait's mean value is higher or lower than before.

Directional selection is extremely common. For example, predatory fish promote directional selection for larger body size in guppies when they selectively feed on the smallest individuals in a guppy population (see *Focus on Research* in Chapter 49). And most cases of artificial selection, including the experiment on the activity levels of house mice, are directional, aimed at increasing or decreasing specific phenotypic traits. Humans routinely use directional selection to produce domestic animals and crops with desired characteristics, such as the small size of chihuahuas and the intense "bite" of chili peppers.

Stabilizing Selection. Traits undergo **stabilizing selection** when individuals expressing intermediate phenotypes have the highest relative fitness (see Figure 20.9b). By eliminating phenotypic extremes, stabilizing selection reduces genetic and phenotypic variation and increases the frequency of intermediate phenotypes. Stabilizing selection is probably the most common mode of natural selection, affecting many familiar traits. For example, very small and very large human newborns are less likely to survive than those born at an intermediate weight **(Figure 20.10).**

Warren G. Abrahamson and Arthur E. Weis of Bucknell University have shown that opposing forces of directional selection can sometimes produce an overall pattern of stabilizing selection **(Figure 20.11).** The gallmaking fly *(Eurosta solidaginis)* is a small insect that feeds on the tall goldenrod plant *(Solidago altissima)*. When a fly larva hatches from its egg, it bores into a goldenrod stem, and the plant responds by producing a spherical growth deformity called a gall. The larva feeds on plant tissues inside the gall. Galls vary dramatically in size; genetic experiments indicate that gall size is a heritable trait of the fly, although plant genotype also has an effect.

Fly larvae inside galls are subjected to two opposing patterns of directional selection. On one hand, a tiny wasp *(Eurytoma gigantea)* parasitizes gallmaking flies by laying eggs in fly larvae inside their galls. After hatching, the young wasps feed on the fly larvae, killing them in the process. However, adult wasps are

Figure 20.10 Observational Research

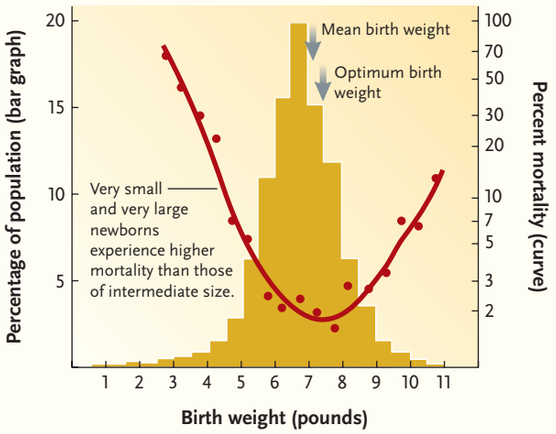
Evidence for Stabilizing Selection in Humans

HYPOTHESIS: Human birth weight has been adjusted by natural selection.

NULL HYPOTHESIS: Natural selection has not affected human birth weight.

METHOD: Two noted human geneticists, Luigi Cavalli-Sforza and Sir Walter Bodmer of Stanford University, collected data on the variability in human birth weight, a character exhibiting quantitative variation, and on the mortality rates of babies born at different weights. The researchers then searched for a relationship between birth weight and mortality rate by plotting both data sets on the same graph. A lack of correlation between birth weight and mortality rate would support the null hypothesis.

RESULTS: When plotted together on the same graph, the bar graph (birth weight) and the curve (mortality rate) illustrate that the mean birth weight is very close to the optimum birth weight (the weight at which mortality is lowest). The two data sets also show that few babies are born at the very low and very high weights associated with high mortality.



CONCLUSION: The shapes and positions of the birth weight bar graph and the mortality rate curve suggest that stabilizing selection has adjusted human birth weight to an average of 7 to 8 pounds.

Figure 20.11 Observational Research

How Opposing Forces of Directional Selection Produce Stabilizing Selection

HYPOTHESIS: The size of galls made by larvae of the gallmaking fly *(Eurosta solidaginis)* is governed by conflicting selection pressures established by parasitic wasps and predatory birds.

PREDICTION: Gallmaking flies that produce galls of intermediate size will be more likely to survive than those that make either small galls or large galls.

METHOD: Abrahamson and his colleagues surveyed galls made by the larvae of the gallmaking fly in Pennsylvania. They measured the diameters of the galls they encountered, and, for those galls in which the larvae had died, they determined whether they had been killed by **(a)** a parasitic wasp *(Eurytoma gigantea)* or **(b)** a predatory bird, such as the downy woodpecker *(Dendrocopus pubescens)*.

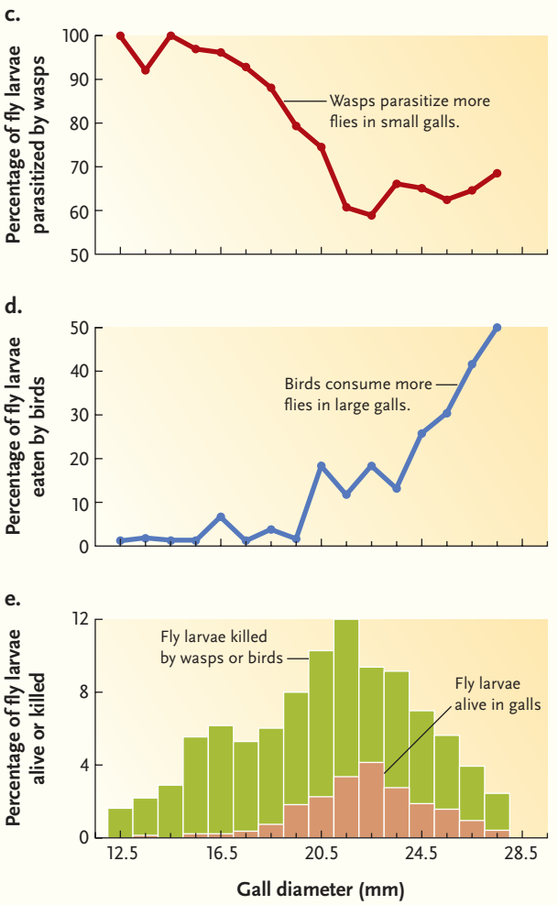
a. *Eurytoma gigantea*, a parasitic wasp

Forrest W. Buchanan/Visuals Unlimited

b. *Dendrocopus pubescens*, a predatory bird

Gregory K. Scott/Photo Researchers, Inc.

RESULTS: Tiny wasps are more likely to parasitize gallmaking fly larvae inside small galls **(c),** fostering directional selection in favor of large galls. By contrast, birds usually feed on fly larvae inside large galls **(d),** fostering directional selection in favor of small galls. These opposing patterns of directional selection create stabilizing selection for the size of galls that the fly larvae make **(e).**



CONCLUSION: Because wasps preferentially parasitize fly larvae in small galls and birds preferentially eat fly larvae in large galls, the opposing forces of directional selection establish an overall pattern of stabilizing selection in favor of medium-sized galls.

so small that they cannot easily penetrate the thick walls of a large gall; they generally lay eggs in fly larvae occupying small galls. Thus, wasps establish directional selection favoring flies that produce large galls, which are less likely to be parasitized. On the other hand, several bird species open galls to feed on mature fly larvae; these predators preferentially open large galls, fostering directional selection in favor of small galls.

In about one-third of the populations surveyed in central Pennsylvania, wasps and birds attacked galls with equal frequency, and flies producing galls of intermediate size had the highest survival rate. The smallest and largest galls—as well as the genetic pre-

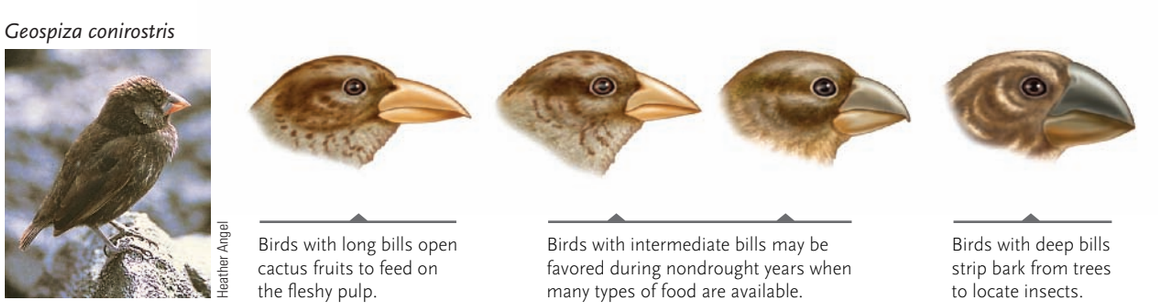


Figure 20.12 **Disruptive selection.** Cactus finches *(Geospiza conirostris)* on Genovesa exhibit extreme variability in the size and shape of their bills.

disposition to make very small or very large galls—were eliminated from the population.

Disruptive Selection. Traits undergo **disruptive selection** when extreme phenotypes have higher relative fitness than intermediate phenotypes (see Figure 20.9c). Thus, alleles producing extreme phenotypes become more common, promoting polymorphism. Under natural conditions, disruptive selection is much less common than directional selection and stabilizing selection.

Peter Grant of Princeton University, the world's expert on the ecology and evolution of the Galápagos finches, has analyzed a likely case of disruptive selection on the size and shape of the bill in a population of cactus finches *(Geospiza conirostris)* on the island of Genovesa. During normal weather cycles the finches feed on ripe cactus fruits, seeds, and exposed insects. During drought years, when food is scarce, they also search for insects by stripping bark from the branches of bushes and trees.

During the long drought of 1977, about 70% of the cactus finches on Genovesa died; the survivors exhibited unusually high variability in their bills **(Figure 20.12).** Grant suggested that this morphological variability allowed birds to specialize on particular foods. Birds that stripped bark from branches to look for insects had particularly deep bills, and birds that opened cactus fruits to feed on the fleshy interior had especially long bills. Thus, birds with extreme bill phenotypes appeared to feed efficiently on specific resources, establishing disruptive selection on the size and shape of their bills. The selection may be particularly strong when drought limits the variety and overall availability of food. However, intermediate bill morphologies may be favored during nondrought years when insects and small seeds are abundant.

Sexual Selection Often Exaggerates Showy Structures in Males

Darwin hypothesized that a special process, which he called **sexual selection**, has fostered the evolution of showy structures—such as brightly colored feathers, long tails, or impressive antlers—as well as elaborate courtship behavior in the males of many animal species. Sexual selection encompasses two related processes. As the result of *intersexual selection* (that is, selection based on the interactions between males and females), males produce these otherwise useless structures simply because females find them irresistibly attractive. Under *intrasexual selection* (that is, selection based on the interactions between members of the same sex), males use their large body size, antlers, or tusks to intimidate, injure, or kill rival males. In many species, sexual selection is the most probable cause of **sexual dimorphism**, differences in the size or appearance of males and females.

Like directional selection, sexual selection pushes phenotypes toward one extreme. But the products of sexual selection are sometimes bizarre—such as the ridiculously long tail feathers of male African widowbirds. How could evolutionary processes favor the production of such costly structures? Malte Andersson of the University of Gothenburg, Sweden, conducted a field experiment to determine whether the long tail feathers were the product of either intersexual selection or intrasexual selection **(Figure 20.13).** Male widowbirds compete vigorously for favored patches of habitat in which they court females. After surveying the behavior of birds under natural conditions, Andersson lengthened the tails of some males, shortened those of others, and left some males essentially unaltered to serve as controls. His results suggest that females are more strongly attracted to males with long tails than to males with short tails, but that tail length had no effect on a male's ability to compete with other males for space in the habitat. Thus, the long tail of the African widowbird is a product of intersexual selection, not intrasexual selection. Behavioral aspects of sexual selection are described further in Chapter 55.

Nonrandom Mating Can Influence Genotype Frequencies

The Hardy-Weinberg model requires individuals to select mates randomly with respect to their genotypes. This requirement is, in fact, often met; humans, for example, generally marry one another in total ignorance of their genotypes for digestive enzymes or blood types.

Nevertheless, many organisms mate nonrandomly, selecting a mate with a particular phenotype

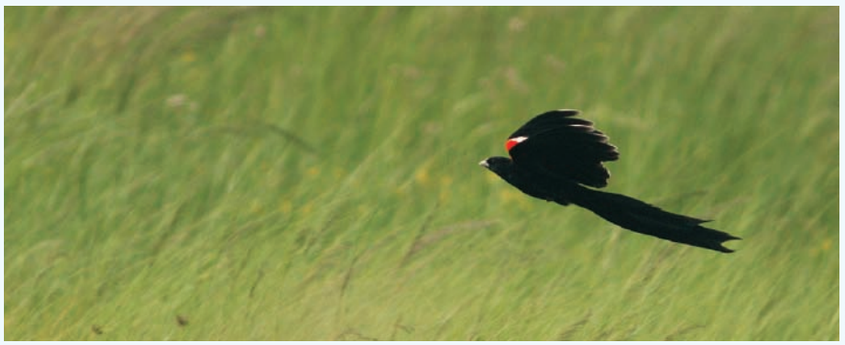
Figure 20.13 Experimental Research

Sexual Selection in Action

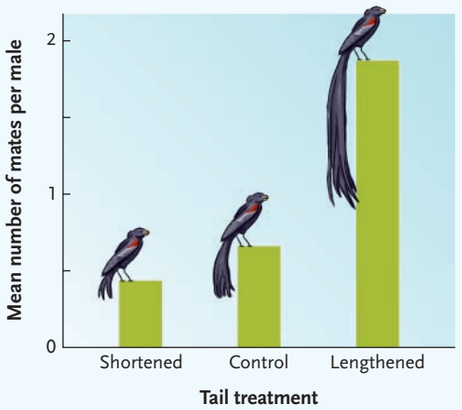
QUESTION: Is the long tail of the male long-tailed widowbird *(Euplectes progne)* the product of intrasexual selection, intersexual selection, or both?

EXPERIMENT: Andersson counted the number of females that associated with individual male widowbirds in the grasslands of Kenya. He then shortened the tails of some individuals by cutting the feathers, lengthened the tails of others by gluing feather extensions to their tails, and left a third group essentially unaltered as a control. One month later, he again counted the number of females associating with each male and compared the results from the three groups.

RESULTS: Males with experimentally lengthened tails attracted more than twice as many mates as males in the control group, and males with experimentally shortened tails attracted fewer. Andersson observed no differences in the ability of altered males and control group males to maintain their display areas.



© 2008 Josef Hlasak



CONCLUSION: Female widowbirds clearly prefer males with experimentally lengthened tails to those with normal tails or experimentally shortened tails. Tail length had no obvious effect on the interactions between males. Thus, the long tail of male widowbirds is the product of intersexual selection.

and underlying genotype. Snow geese, for example, usually select mates of their own color, and a tall woman is more likely to marry a tall man than a short man. If no one phenotype is preferred by all potential mates, nonrandom mating does not establish selection for one phenotype over another. But because individuals with similar genetically based phenotypes mate with each other, the next generation will contain fewer heterozygous offspring than the Hardy-Weinberg model predicts.

Inbreeding is a special form of nonrandom mating in which individuals that are genetically related mate with each other. Self-fertilization in plants (see Chapter 34) and a few animals (see Chapter 47) is an extreme example of inbreeding because offspring are produced from the gametes of a single parent. However, other organisms that live in small, relatively closed populations often mate with related individuals. Because relatives often carry the same alleles, inbreeding generally increases the frequency of homozygous genotypes and decreases the frequency of heterozygotes. Thus, recessive phenotypes are often expressed. For example, the high incidence of Ellis–van Creveld syndrome among the Old Order Amish population, mentioned earlier, is caused by inbreeding. Although the founder effect originally established the disease-causing allele in this population, inbreeding increases the likelihood that it will be expressed. Most human societies discourage matings between genetically close relatives, thereby reducing inbreeding and the production of recessive homozygotes.

Study Break

1. Which agents of microevolution tend to increase genetic variation within populations, and which ones tend to decrease it?
2. Which mode of natural selection increases the representation of the average phenotype in a population?
3. In what way is sexual selection like directional selection?

20.4 Maintaining Genetic and Phenotypic Variation

Evolutionary biologists continue to discover extraordinary amounts of genetic and phenotypic variation in most natural populations. How can so much variation persist in the face of stabilizing selection and genetic drift?

Diploidy Can Hide Recessive Alleles from the Action of Natural Selection

The diploid condition reduces the effectiveness of natural selection in eliminating harmful recessive alleles from a population. Although such alleles are disadvantageous in the homozygous state, they may have little or no effect on heterozygotes. Thus, recessive alleles can be protected from natural selection by the phenotypic expression of the dominant allele.

In most cases, the masking of recessive alleles in heterozygotes makes it almost impossible to eliminate them completely through selective breeding. Experimentally, we can prevent homozygous recessive organisms from mating. But, as the frequency of a recessive allele decreases, an increasing proportion of its remaining copies is "hidden" in heterozygotes **(Table 20.3).** Thus, the diploid state preserves recessive alleles at low frequencies, at least in large populations. In small populations, a combination of natural selection and genetic drift can eliminate harmful recessive alleles.

Table 20.3 **Masking of Recessive Alleles in Diploid Organisms**

When a recessive allele is common in a population (top), most copies of the allele are present in homozygotes. But when the allele is rare (bottom), most copies of it exist in heterozygotes. Thus, rare alleles that are completely recessive are protected from the action of natural selection because they are masked by dominant alleles in heterozygous individuals.

Frequency of Allele *a*	Genotype Frequencies*			% of Allele *a* Copies in	
	AA	*Aa*	*aa*	*Aa*	*aa*
0.99	0.0001	0.0198	0.9801	1	99
0.90	0.0100	0.1800	0.8100	10	90
0.75	0.0625	0.3750	0.5625	25	75
0.50	0.2500	0.5000	0.2500	50	50
0.25	0.5625	0.3750	0.0625	75	25
0.10	0.8100	0.1800	0.0100	90	10
0.01	0.9801	0.0198	0.0001	99	1

*Population is assumed to be in genetic equilibrium.

Natural Selection Can Maintain Balanced Polymorphisms

A **balanced polymorphism** is one in which two or more phenotypes are maintained in fairly stable proportions over many generations. Natural selection preserves balanced polymorphisms when heterozygotes have higher relative fitness, when different alleles are favored in different environments, and when the rarity of a phenotype provides an advantage.

Heterozygote Advantage. A balanced polymorphism can be maintained by **heterozygote advantage,** when heterozygotes for a particular locus have higher relative fitness than either homozygote. The best-documented example of heterozygote advantage is the maintenance of the *HbS* (sickle) allele, which codes for a defective form of hemoglobin in humans. As you learned in Chapter 12, hemoglobin is an oxygen-transporting molecule in red blood cells. The hemoglobin produced by the *HbS* allele differs from normal hemoglobin (coded by the *HbA* allele) by just one amino acid. In *HbS*/*HbS* homozygotes, the faulty hemoglobin forms long fibrous chains under low oxygen conditions, causing red blood cells to assume a sickle shape (as shown in Figure 12.1). Homozygous *HbS*/*HbS* individuals often die of sickle-cell disease before reproducing, yet in tropical and subtropical Africa, *HbS*/*HbA* heterozygotes make up nearly 25% of many populations.

Why is the harmful allele maintained at such high frequency? It turns out that sickle-cell disease is most common in regions where malarial parasites infect red blood cells in humans **(Figure 20.14).** When heterozygous *HbA*/*HbS* individuals contract malaria, their infected red blood cells assume the same sickle shape as those of homozygous *HbS*/*HbS* individuals. The sickled cells lose potassium, killing the parasites, which limits their spread within the infected individual. Heterozygous individuals often survive malaria because the parasites do not multiply quickly inside them; their immune systems can effectively fight the infection; and they retain a large population of uninfected red blood cells. Homozygous *HbA*/*HbA* individuals are also subject to malarial infection, but because their infected cells do not sickle, the parasites multiply rapidly, causing a severe infection with a high mortality rate.

Therefore, *HbA*/*HbS* heterozygotes have greater resistance to malaria and are more likely to survive severe infections in areas where malaria is prevalent. Natural selection preserves the *HbS* allele in these populations because heterozygotes in malaria-prone areas have higher relative fitness than homozygotes for the normal *HbA* allele.

Selection in Varying Environments. Genetic variability can also be maintained within a population when different alleles are favored in different places or at different times. For example, the shells of European garden

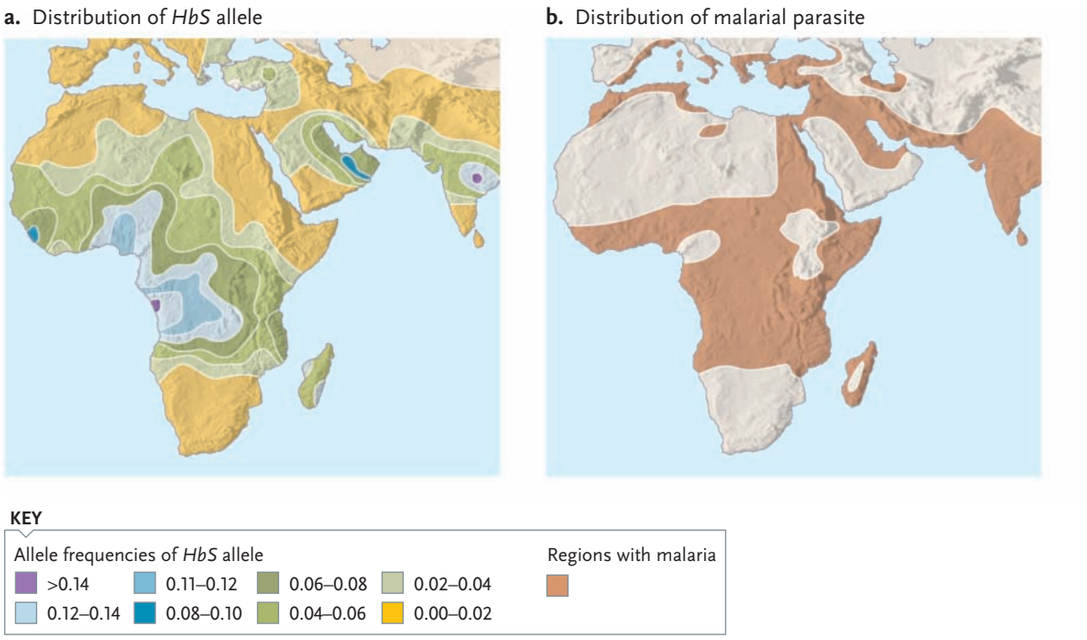


Figure 20.14
Heterozygote advantage. The distribution of the *HbS* allele **(a)**, which causes sickle-cell disease in homozygotes, roughly matches the distribution of the malarial parasite *Plasmodium falciparum* **(b)** in southern Europe, Africa, the Middle East, and India. Gene flow among human populations has carried the *HbS* allele to some malaria-free regions.

snails range in color from nearly white to pink, yellow, or brown, and may be patterned by one to five stripes of varying color (see Figure 20.2a). This polymorphism, which is relatively stable through time, is controlled by several gene loci. The variability in color and in striping pattern can be partially explained by selection for camouflage in different habitats.

Predation by song thrushes *(Turdus ericetorum)* is a major agent of selection on the color and pattern of these snails in England. When a thrush finds a snail, it smacks it against a rock to break the shell. The bird eats the snail, but leaves the shell near its "anvil." Researchers used the broken shells near an anvil to compare the phenotypes of captured snails to a random sample of the entire snail population. Their analyses indicated that thrushes are visual predators, usually capturing snails that are easy to find. Thus, well-camouflaged snails survive, and the alleles that specify their phenotypes increase in frequency.

The success of camouflage varies with habitat, however; local subpopulations of the snail, which occupy different habitats, often differ markedly in shell color and pattern. The predators eliminate the most conspicuous individuals in each habitat; thus, natural selection differs from place to place **(Figure 20.15).** In woods where the ground is covered with dead leaves, snails with unstriped pink or brown shells predominate. In hedges and fields, where the vegetation includes thin stems and grass, snails with striped yellow shells are the most common. In populations that span several habitats, selection preserves different alleles in different places, thus maintaining variability in the population as a whole.

Frequency-Dependent Selection. Sometimes genetic variability is maintained in a population simply because rare phenotypes—whatever they happen to be—have higher relative fitness than more common phenotypes. The rare phenotype will increase in frequency until it becomes so common that it loses its advantage. Such phenomena are examples of **frequency-dependent selection** because the selective advantage enjoyed by a particular phenotype depends on its frequency in the population.

Predator-prey interactions can establish frequency-dependent selection because predators often focus their attention on the most common types of prey (see Chapter 50). For example, the aquatic insects called water boatmen occur in three different shades of brown. When all three shades are available at moderate frequencies, fish preferentially feed on the darkest individuals, which are the least camouflaged. But if any one phenotype is very common, fish will learn to focus their attention on that phenotype (see Chapter 54), consuming it in disproportionately large numbers **(Figure 20.16).**

Some Genetic Variations May Be Selectively Neutral

Many biologists believe that some genetic variations are neither preserved nor eliminated by natural selection. According to the **neutral variation hypothesis**, some of the genetic variation at loci coding for enzymes and other soluble proteins is **selectively neutral.** Even if various alleles code for slightly different amino acid sequences in proteins, the different forms of the proteins may function equally well. In those cases, natural selection would not favor some alleles over others.

Biologists who support the neutral variation hypothesis do not question the role of natural selection in producing complex anatomical structures or useful biochemical traits. They also recognize that selection reduces the frequency of harmful alleles. But they argue that we should not simply assume that every genetic variant that persists in a population has been preserved by natural selection. In practice, it is often very difficult to test the natural variation hypothesis because the fitness effects of different alleles are often subtle and vary with small changes in the environment.

The neutral variation hypothesis helps to explain why we see different levels of genetic variation in different populations. It proposes that genetic variation is directly proportional to a population's size and the length of time over which variations have accumulated. Small populations experience fewer mutations than large populations simply because they include fewer replicating genomes. Small populations also lose rare alleles more readily through genetic drift. Thus, small populations should exhibit less genetic variation than large ones, and a population, like the northern elephant seals, that has experienced a recent population bottleneck should exhibit an exceptionally low level of genetic variation. These predictions of the neutral variation hypothesis are generally supported by empirical data.

Study Break

1. How does the diploid condition protect harmful recessive alleles from natural selection?
2. What is a balanced polymorphism?
3. Why is the allele that causes sickle-cell disease very rare in human populations that are native to northern Europe?

20.5 Adaptation and Evolutionary Constraints

Although natural selection preserves alleles that confer high relative fitness on the individuals that carry them, researchers are cautious about interpreting the benefits that particular traits may provide.

Figure 20.15 Observational Research

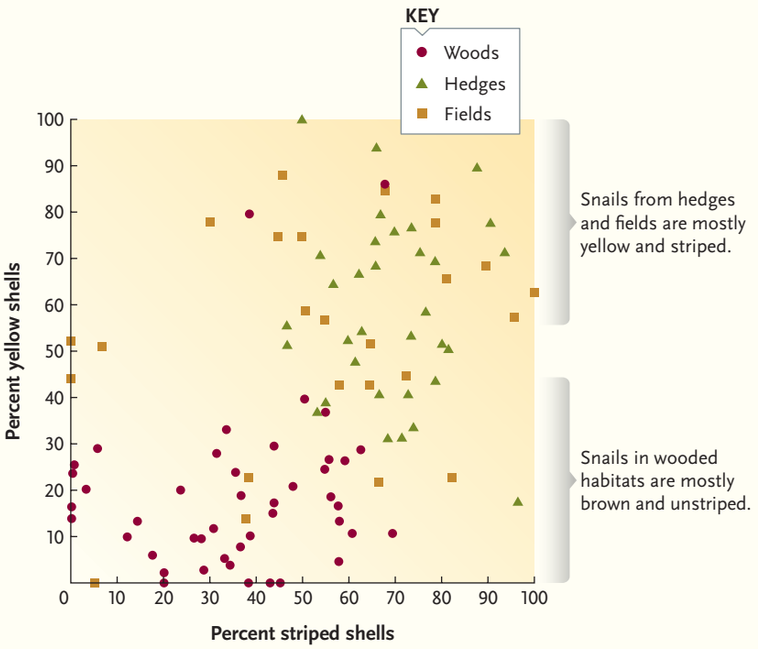
Habitat Variation in Color and Striping Patterns of European Garden Snails

HYPOTHESIS: Genetically based variations in the shell color and striping patterns of the European garden snail *(Cepaea nemoralis)* differ substantially from one type of vegetation to another because birds and other visual predators establish strong selection for camouflage in local populations.

PREDICTION: Snails with plain, dark-colored shells will be most abundant in woodland habitats, but snails with striped, light-colored shells will be most abundant in hedges and fields.

METHOD: Two British researchers, A. J. Cain and P. M. Shepard, surveyed the distribution of color and striping patterns of snails in many local populations. They plotted the data on a graph showing the percentage of snails with yellow shells versus the percentage of snails with striped shells, noting the vegetation type where each local population lived.

RESULTS: The shell color and striping patterns of snails living in a particular vegetation type tend to be clustered on the graph, reflecting phenotypic differences that enable the snails to be camouflaged in different habitats. Thus, the alleles that control these characters vary from one local population to another.



CONCLUSION: Variations in the color and striping patterns on the shells of European garden snails allow most snails to be camouflaged in whatever habitat they occupy. Because these traits are genetically based, the frequencies of the alleles that control them also differ among snails living in different vegetation types. Natural selection therefore favors different alleles in different local populations, maintaining genetic variability in populations that span several vegetation types.

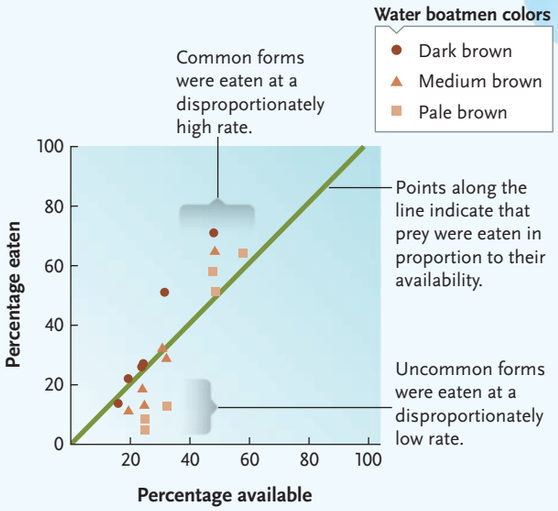
Figure 20.16 Experimental Research

Demonstration of Frequency-Dependent Selection

QUESTION: How does the frequency of a prey type influence the likelihood that it will be captured by predators?

EXPERIMENT: Water boatmen *(Sigara distincta)* occur in three color forms, which vary in the effectiveness of their camouflage. Researchers offered different proportions of the three color forms to predatory fishes in the laboratory and recorded how many of each form were eaten.

RESULTS: When all three phenotypes were available, predatory fishes consumed a disproportionately large number of the most common form, thereby reducing its frequency in the population.



CONCLUSION: Predators tend to feed disproportionately on whatever form of their prey is most abundant, thereby reducing its frequency in the prey population.

Scientists Construct Hypotheses about the Evolution of Adaptive Traits

An **adaptive trait** is any product of natural selection that increases the relative fitness of an organism in its environment. **Adaptation** is the accumulation of adaptive traits over time, and this book describes many examples. The change in the oxygen-binding capacity of hemoglobin in response to carbon dioxide concentration, the water-retaining structures and special photosynthetic pathways of desert plants, and the warning coloration of poisonous animals can all be interpreted as adaptive traits.

In fact, we can concoct an adaptive explanation for almost any characteristic we observe in nature. But such explanations are just fanciful stories unless they are framed as testable hypotheses about the relative fitness of different phenotypes and genotypes. Unfortunately, evolutionary biologists cannot always conduct straightforward experiments because they sometimes study traits that do not vary much within a population or species. In such cases, they may compare variations of a trait in closely related species living in different environments. For example, one can test how the traits of desert plants are adaptive by comparing them to traits in related species from moister habitats.

When biologists try to unravel how and why a particular characteristic evolved, they must also remember that a trait they observe today may have had a different function in the past. For example, the structure of the shoulder joint in birds allows them to move their wings first upward and backward and then downward and forward during flapping flight. But analyses of the fossil record reveal that this adaptation, which is essential for flight, did not originate in birds: some predatory nonflying dinosaurs, including the ancestors of birds, had a similarly constructed shoulder joint. Researchers hypothesize that these fast-running predators may have struck at prey with a flapping motion similar to that used by modern birds. Thus, the structure of the shoulder may have first evolved as an adaptation for capturing prey, and only later proved useful for flapping flight. This hypothesis—however plausible it may be—cannot be tested by direct experimentation because the nonflying ancestors of bird have been extinct for millions of years. Instead, evolutionary biologists must use anatomical studies of birds and their ancestors as well as theoretical models about the mechanics of movement to challenge and refine the hypothesis.

Finally, although evolution has produced all the characteristics of organisms, not all are necessarily adaptive. Some traits may be the products of chance events and genetic drift. Others are produced by alleles that were selected for unrelated reasons (see Section 12.2). And still other characteristics result from the action of basic physical laws. For example, the seeds of many plants fall to the ground when they mature, reflecting the inevitable effect of gravity.

Several Factors Constrain Adaptive Evolution

When we analyze the structure and function of an organism, we often marvel at how well adapted it is to its environment and mode of life. However, the adaptive traits of most organisms are compromises produced by competing selection pressures. Sea turtles, for example, must lay their eggs on beaches because their embryos cannot acquire oxygen under water. Although flippers allow females to crawl to nesting sites on beaches, they are not ideally suited for terrestrial locomotion. Their structure reflects their primary function in underwater locomotion.

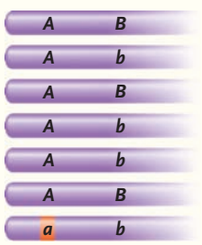
Moreover, no organism can be perfectly adapted to its environment because environments change over

Unanswered Questions

What are the evolutionary forces affecting molecular variation within populations?

This question may sound like a simple restatement of the entire chapter you have just read, but it is one of the *fundamental* questions in population genetics today—and we have only begun to scratch its surface. The Hardy-Weinberg principle provides a useful null hypothesis, but since we know that evolution happens routinely, that null hypothesis is very frequently rejected. Recent studies have attempted to address this question using theoretical models, extensive DNA sequence data, and detailed measures of recombination rate.

Recombination generates new variation, and, most importantly, it causes the evolutionary forces acting on some genes to become independent of forces acting on other genes. Let's imagine that genes *A* and *B* are on the same chromosome, as shown in this depiction of chromosomes sampled from different individuals within a population:



Gene *B* has two alleles (*B* and *b*), but they have no phenotypic effect, and natural selection does not act on them. Suppose that a new advantageous allele at gene *A* (designated *a*) arises in one chromosome. If there is no recombination between genes *A* and *B,* then as allele *a* spreads in the population by selection, so too will allele *b,* even though there was no selection directly favoring the *b* allele. This effect of selection on nearby genes is called a *selective sweep.* By contrast, if genes *A* and *B* frequently recombine, then allele *a* may not remain associated with allele *b*. Under frequent recombination, the spread of allele *a* may have little or no effect on gene *B:* sometimes *a* will be associated with *b,* but at other times *a* will be associated with *B*.

In the 1990s, evolutionary geneticists were greatly excited by several studies that identified a strong and positive relationship between the recombination rate between particular genes and the amount of genetic variation within those genes. In other words, genes that experienced a lot of recombination also exhibited a great deal of variability. This relationship is consistent with the hypothesis that natural selection often occurs throughout the genome—new advantageous alleles arise frequently, and the impact of their "sweeps" is proportional to their recombination rates. This relationship between recombination and genetic variation was first documented in *Drosophila* (fruit flies) by Chip Aquadro and his team at Cornell University, but it has since been demonstrated in humans and various plants. Hence, this pattern appears to be very general.

However, our initial interpretation may be too simplistic. Brian Charlesworth, then at the University of Chicago, suggested that the observed pattern may result from the frequent appearance of detrimental mutations that eliminate variation in regions of low recombination—called *background selection*—rather than from sweeps associated with the spread of advantageous alleles. Given that detrimental mutations arise far more frequently than advantageous ones, background selection surely explains some of this general pattern, and perhaps much of it.

An alternative hypothesis that may explain the relationship between recombination rate and genetic variation suggests that recombination rate and the level of genetic variation may be mechanistically connected. A direct connection may operate if recombination itself induces mutations, resulting in higher mutation rates in regions of high recombination. Alternatively, the connection may be indirect: recombination rate is known to be related to the base composition in specific regions of the genome, and base composition is known to influence mutation rates. In 2006, Chris Spencer and his colleagues at Oxford University examined the impact of recombination rates on patterns of nucleotide variation at a very fine scale across the human genome. They found that recombination rates had very local effects on variation, an observation that is consistent with the alternative hypothesis of a mechanistic connection between recombination and mutation rate; their results are not consistent with explanations involving natural selection.

Although biologists first thought that the observed relationship between recombination rate and genetic variation had solved questions about the evolutionary forces that affect molecular variation, this observation has become a puzzle in and of itself. Many of us continue to address this question, now using whole-genome sequences and theoretical and empirical tools for estimating recombination rates. We know that the "final answer" will be that all of the processes described above contribute to this relationship, but knowing their specific contributions will help us understand how, how much, and what kinds of natural selection shape variation within genomes.

Mohamed Noor is an associate professor of biology at Duke University. His research interests include speciation and evolutionary genetics, and recombination. To learn more about his research go to http://www.biology.duke.edu/noorlab/Noorlab.html.

Dr. Noor was a PhD student with Dr. Jerry Coyne, who contributed the Unanswered Questions for Chapter 21.

time. When selection occurs in a population, it preserves alleles that are successful under the prevailing environmental conditions. Thus, each generation is adapted to the environmental conditions under which its parents lived. If the environment changes from one generation to the next, adaptation will always lag behind.

Another constraint on the evolution of adaptive traits is historical. Natural selection is not an engineer that designs new organisms from scratch. Instead, it acts on new mutations and existing genetic variation. Because new mutations are fairly rare, natural selection works primarily with alleles that have been pres-

ent for many generations. Thus, adaptive changes in the morphology of an organism are almost inevitably based on small modifications of existing structures. The bipedal (two-footed) posture of humans, for example, evolved from the quadrupedal (four-footed) posture of our ancestors. Natural selection did not produce an entirely new skeletal design to accompany this radical behavioral shift. Instead, existing characteristics of the spinal column and the musculature of the legs and back were modified, albeit imperfectly, for an upright stance.

The agents of evolution cause microevolutionary changes in the gene pools of populations. In the next chapter, we examine how microevolution in different populations can cause their gene pools to diverge. The extent of genetic divergence is sometimes sufficient to cause the populations to evolve into different species.

Study Break

1. How can a biologist test whether a trait is adaptive?
2. Why are most organisms adapted to the environments in which their parents lived?

Review

Go to ThomsonNOW™ at www.thomsonedu.com/login to access quizzing, animations, exercises, articles, and personalized homework help.

20.1 Variation in Natural Populations

- Phenotypic traits exhibit either quantitative or qualitative variation within populations of all organisms (Figures 20.2 and 20.3).
- Genetic variation, environmental factors, or an interaction between the two cause phenotypic variation within populations. Only genetically based phenotypic variation is heritable and subject to evolutionary change.
- Genetic variation arises within populations largely through mutation and genetic recombination. Artificial selection experiments and analyses of protein and DNA sequences reveal that most populations include significant genetic variation (Figure 20.6).

20.2 Population Genetics

- All the alleles in a population comprise its gene pool, which can be described in terms of allele frequencies and genotype frequencies.
- The Hardy-Weinberg principle of genetic equilibrium is a null model that describes the conditions under which microevolution will not occur: mutations do not occur; populations are closed to migration; populations are infinitely large; natural selection does not operate; and individuals select mates at random. Microevolution, a change in allele frequencies through time, occurs in populations when the restrictive requirements of the model are not met.

Animation: How to find out if a population is evolving

20.3 The Agents of Microevolution

- Several processes cause microevolution in populations. Mutation introduces completely new genetic variation. Gene flow carries novel genetic variation into a population through the arrival and reproduction of immigrants. Genetic drift causes random changes in allele frequencies, especially in small populations. Natural selection occurs when the genotypes of some individuals enable them to survive and reproduce more than others. Nonrandom mating within a population can cause its genotype frequencies to depart from the predictions of the Hardy-Weinberg equilibrium.
- Natural selection alters phenotypic variation in one of three ways (Figure 20.9). Directional selection increases or decreases the mean value of a trait, shifting it toward a phenotypic extreme. Stabilizing selection increases the frequency of the mean phenotype and reduces variability in the trait (Figure 20.10). Disruptive selection increases the frequencies of extreme phenotypes and decreases the frequency of intermediate phenotypes (Figure 20.12).
- Sexual selection promotes the evolution of exaggerated structures and behaviors (Figure 20.13).
- Although nonrandom mating does not change allele frequencies, it can affect genotype frequencies, producing more homozygotes and fewer heterozygotes than the Hardy-Weinberg model predicts.

Animation: Directional selection

Animation: Change in moth population

Animation: Stabilizing selection

Animation: Disruptive selection

Animation: Disruptive selection among African finches

Animation: Simulation of genetic drift

20.4 Maintaining Genetic and Phenotypic Variation

- Diploidy can maintain genetic variation in a population if alleles coding for recessive traits are not expressed in heterozygotes and are thus hidden from natural selection.
- Polymorphisms are maintained in populations when heterozygotes have higher relative fitness than both homozygotes (Figure 20.14), when natural selection occurs in variable environments (Figure 20.15), or when the relative fitness of a phenotype varies with its frequency in the population (Figure 20.16).
- Some biologists believe that many genetic variations are selectively neutral, conferring neither advantages nor disadvantages on the individuals that carry them. The neutral variation hypothesis explains why large populations and those that have not experienced a recent population bottleneck exhibit the highest levels of genetic variation.

Animation: Distribution of sickle-cell trait

Animation: Life cycle of *Plasmodium*

20.5 Adaptation and Evolutionary Constraints

- Adaptive traits increase the relative fitness of individuals carrying them. Adaptive explanations of traits must be framed as testable hypotheses.
- Natural selection cannot result in perfectly adapted organisms because most adaptive traits represent compromises among conflicting needs; because most environments are constantly changing; and because natural selection can affect only existing genetic variation.

Animation: Adaptation to what?

Questions

Self-Test Questions

1. Which of the following represents an example of qualitative phenotypic variation?
   a. the lengths of people's toes
   b. the body sizes of pigeons
   c. human ABO blood groups
   d. the birth weights of humans
   e. the number of leaves on oak trees
2. A population of mice is at Hardy-Weinberg equilibrium at a gene locus that controls fur color. The locus has two alleles, *M* and *m*. A genetic analysis of one population reveals that 60% of its gametes carry the *M* allele. What percentage of mice contains both the *M* and *m* alleles?
   a. 60%
   b. 48%
   c. 40%
   d. 36%
   e. 16%
3. If the genotype frequencies in a population are 0.60 *AA*, 0.20 *Aa*, and 0.20 *aa*, and if the requirements of the Hardy-Weinberg principle apply, the genotype frequencies in the offspring generation will be:
   a. 0.60 *AA*, 0.20 *Aa*, 0.20 *aa*.
   b. 0.36 *AA*, 0.60 *Aa*, 0.04 *aa*.
   c. 0.49 *AA*, 0.42 *Aa*, 0.09 *aa*.
   d. 0.70 *AA*, 0.00 *Aa*, 0.30 *aa*.
   e. 0.64 *AA*, 0.32 *Aa*, 0.04 *aa*.
4. The reason spontaneous mutations do not have an immediate effect on allele frequencies in a large population is that:
   a. mutations are random events, and mutations may be either beneficial or harmful.
   b. mutations usually occur in males and have little effect on eggs.
   c. many mutations exert their effects after an organism has stopped reproducing.
   d. mutations are so rare that mutated alleles are greatly outnumbered by nonmutated alleles.
   e. most mutations do not change the amino acid sequence of a protein.
5. The phenomenon in which chance events cause unpredictable changes in allele frequencies is called:
   a. gene flow.
   b. genetic drift.
   c. inbreeding.
   d. balanced polymorphism.
   e. stabilizing selection.
6. An Eastern European immigrant carrying the allele for Tay Sachs disease settled in a small village on the St. Lawrence River. Many generations later, the frequency of the allele in that village is statistically higher than it is in the immigrant's homeland. The high frequency of the allele in the village probably provides an example of:
   a. natural selection.
   b. the concept of relative fitness.
   c. the Hardy-Weinberg genetic equilibrium.
   d. phenotypic variation.
   e. the founder effect.
7. If a storm kills many small sparrows in a population, but only a few medium-sized and large ones, which type of selection is probably operating?
   a. directional selection
   b. stabilizing selection
   c. disruptive selection
   d. intersexual selection
   e. intrasexual selection
8. Which of the following phenomena explains why the allele for sickle-cell hemoglobin is common in some tropical and subtropical areas where the malaria parasite is prevalent?
   a. balanced polymorphism
   b. heterozygote advantage
   c. sexual dimorphism
   d. neutral selection
   e. stabilizing selection
9. The neutral variation hypothesis proposes that:
   a. complex structures in most organisms have not been fostered by natural selection.
   b. most mutations have a strongly harmful effect.
   c. some mutations are not affected by natural selection.
   d. natural selection cannot counteract the action of gene flow.
   e. large populations are subject to stronger natural selection than small populations.
10. Phenotypic characteristics that increase the fitness of individuals are called:
    a. mutations.
    b. founder effects.
    c. heterozygote advantages.
    d. adaptive traits.
    e. polymorphisms.

Questions for Discussion

1. Most large commercial farms routinely administer antibiotics to farm animals to prevent the rapid spread of diseases through a flock or herd. Explain why you think that this practice is either wise or unwise.
2. Many human diseases are caused by recessive alleles that are not expressed in heterozygotes. Explain why it is almost impossible to eliminate such genetic traits from human populations.
3. Using two types of beans to represent two alleles at the same gene locus, design an exercise to illustrate how population size affects genetic drift.
4. In what ways are the effects of sexual selection, disruptive selection, and nonrandom mating different? How are they similar?

Experimental Analysis

Design an experiment to test the hypothesis that the differences in size among adult guppies are determined by the amount of food they eat rather than by genetic factors.

Evolution Link

Captive breeding programs for endangered species often have access to a limited supply of animals for a breeding stock. As a result, their offspring are at risk of being highly inbred. Why and how might zoological gardens and conservation organizations avoid or minimize inbreeding?

How Would You Vote?

The symptoms of Huntington disease and some other genetically based diseases in humans appear only after the carriers of the disease-causing allele have already reproduced. As a result, they pass the alleles to their offspring and the disease persists in the population. Do you think that all people should be screened for disease-causing alleles and that carriers of such alleles should be discouraged or even prevented from having children? Go to www.thomsonedu.com/login to investigate both sides of the issue and then vote.