SAMPLE CHAPTER 3
Principles of Biological Evolution

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Chapter 3
Principles of Biological Evolution

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CHAPTER SUMMARY

Photo above: Blue-footed boobies from the Galapagos Islands
Because humans belong to the animal kingdom, they can be studied in the same way as any species of animal. Humans are biologically unique in their combination of bipedal locomotion and large brains relative to body size; they are also unique in their ability to speak, in making and appreciating art, and in actively teaching their offspring all manner of things from survival skills to mother-in-law jokes. But as Robert Foley (1987) suggests, humans are not the unique species—they are “just another unique species.” Because all forms of life today use a variant of the same DNA code, scientists are convinced that the differences between humans and viruses result from different amounts and sequences of DNA. Therefore, anthropologists can study humans using the same principles that scientists use in studying other animals. But because evolutionary biology is a historical science, “narrative hypotheses” must substitute for experiments to describe and explain evolutionary happenings and processes.

Studying humans biologically has inherent problems that studying most other animal species does not have. First, human generations are very long, averaging around 20 years, not 14 days as with fruit flies or 65 days as with guinea pigs. Some species, such as elephants, outdo humans in generation length, but these are rare. A second problem results from the fact that experimenters cannot control human matings the way they can control fruit fly, mouse, or guinea pig matings. Finally, the number of offspring produced by individual human couples is very small compared with the number produced by, say, rabbits or fish. It is therefore not surprising that Gregor Mendel established the principles of heredity using common peas, not humans, and that later scientists used guinea pigs and fruit flies to work out the details of modern genetics. The similarity in the DNA code from insects to humans allows scientists to move fairly freely among various animal species using the same biological principles, knowing that a principle discovered in one species is likely transferable to others.

Anthropologists study humans in groups rather than as individuals, and biological anthropologists are interested not only in how and why the human lineage evolved as it did from the past to the present, but also in the nature of modern human populations. In the descriptions and analyses of humans in subsequent chapters, it is the group that is the focus of inquiry, not the individual; and although this chapter focuses on the principles of evolution, these principles are valid for modern humans as well.

Before we look at the long “story of evolution” from the beginning of life to modern species, you need to understand the basic principles of evolution as they apply to any and all organisms and at any and all times. Because this book focuses on the human species, it will seem as though first life evolved directly to modern humans, but nothing could be further from the truth. In actuality, first life led to the millions of varied species of plants and animals that have ever lived, those that became extinct and those that survived to evolve to modern species. Evolution as a science (and as a paradigm for, or way to look at, life on Earth) has had a chronological history offering its own lessons to be learned. This chapter therefore begins with a look at what evolution is and then considers what early scientific thinkers believed about what we now call evolution. Those early ideas were not about change at all, but rather about fixation of particular kinds of plants and animals. Slowly, however, the geological events of Earth’s past began to be discovered, and scientists came to realize that there was enough time for evolutionary happenings to have occurred. The mid-nineteenth century saw two pioneers—Gregor Mendel and Charles Darwin—begin the modern science of evolution, followed by a synthesis of many evolutionary hypotheses in the mid-twentieth century. The last half of the chapter focuses on how modern biology views evolution today, particularly in terms of the causes of evolution in the past and the present.

What Is Evolution?

Biological evolution is both simple and complex. Charles Darwin defined evolution in 1859 as “descent with modification,” with the word descent referring to time, and modification referring to change. A hundred and fifty years later, a good short definition...
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of evolution is still “biological change through time.” Fleshed out a bit, biological evolution is the process by which ancestral populations accumulate morphological and genetic changes to become more modern populations. Given this definition, biological evolution is fact, not hypothesis or theory, because change can often be observed.

For example, controlled experiments with fruit flies show the fact of evolution. If a fruit fly population is left alone in a cage with banana mush but no human manipulation, experimenters can compare two generations of fruit flies morphologically (in terms of outer appearance), inferring their genetics, for signs of change. Geneticists traditionally use fruit flies because some of their chromosomes are large and easily observed, and generation length is a mere two weeks. Variations in such observable traits as wing size (long, medium, short, or just nubbins) or eye color (red or white) can be counted for the first generation and recounted when the second generation takes its place, and simple proportions compared. The proportions will probably not be exactly the same. If the populations are numerically large, mutations or selection may alter the counts slightly; a single mutation, for example, can change wing size to nubbins in the mutated individual. If the populations are numerically small, chance will probably alter the counts. In any case, when a new population has changed biologically through time, it has, by definition, evolved. This change can be observed and measured. After five or six generations of fruit fly evolution, again with no human manipulation beyond feeding and removing dead flies, patterns of evolution can be established and causes hypothesized for the accumulated change. We will explore evolutionary causes later in this chapter.

Other lines of support, too, point to evolution as fact. First, the fossil record shows many different kinds of support for evolution. Scientists trace entire lineages by documenting succeedingly older and older fossils, demonstrating that change is occurring over time. Second, many cases of natural selection can be documented in nature. Such cases show how differential success in reproduction in a population results in differences in morphology and genetics in subsequent generations. Third, on the molecular level, humans are said to share 98.7 percent of both their structural genes and DNA with chimpanzees and bonobos. This argues strongly for a common ancestor of those three modern species in the not-too-distant past (Marks 2000a, 2000b). On the other hand, both humans and apes share a lot of their DNA with bananas as well (Marks 2000a)! The fact that all life is composed of the same amino acids points directly to an original common ancestor for living things that are as different today as bananas, chickens, and humans.

One line of evidence might be argued away, but the many different kinds of evidence argue that evolution is fact, and no successful alternative explanation for the fossil record and the molecular similarities has ever been found.

Early Ideas about Evolution

Scholars have known for centuries that biological change occurs, but the scope and reasons for change were not known or appreciated until the nineteenth century. Unlike most classical philosophers, Anaximander, a sixth-century BC Greek, was one of the first classical thinkers to use a kind of evolutionary reasoning; he proposed that humans evolved from fish (Harris 1981). Two classical Greek philosophical tenets, however, were instrumental in keeping the idea of changing life forms from being accepted until the seventeenth century. Plato, like most scholars of his time, believed in essentialism, the notion that ideal types had been created in perfect form and had neither the ability nor the need to change. Aristotle proposed that these ideal types were arranged in a hierarchy of perfection, the “Great Chain of Being.” As long as people regarded species as perfect and fixed, there could be no concept of change or evolution.

Two other obstacles stood in the way of acceptance of the idea of evolution. First, as long as the Catholic Church insisted on a literal reading of the Bible that claimed that God created individual species, there could be no acknowledgment of change. For centuries the Church exerted an enormous influence over scientists and scientific thinking,
with death as the punishment for contradicting its dogma. Second, for centuries there was a widespread belief among the scientific, clerical, and lay world that the Earth was not very old. Until a long existence for the planet was acknowledged, the concept of evolution could not take root.

As long as these obstacles remained, biological science consisted only of collections, descriptions, and classifications. During the seventeenth century, however, scientists began to seek explanations about the world by looking at the world itself instead of relying on philosophy or religion. Geologists began to ask “How old is the Earth?” instead of assuming the literal accuracy of Biblical stories, and they began to reconstruct the history of the Earth and its living creatures by studying the fossil record in its geological context. In time, people realized that both geological and biological records demonstrated the passage of enormous amounts of time, far beyond the mere 5,658 years (6,004 in the original) that Bishop Ussher proposed in 1654. Just as time constraining was the belief that the Flood had occurred in 2,349 BC, giving only a bit more than 4,000 years for the entire world of animals (including humans) to repopulate the globe from Mount Ararat in present-day Turkey.

Eighteenth-Century Ideas

The Age of Exploration and discoveries of fossils and of stratified rock layers that ordered the fossils paved the way for new ways of thinking about life. As early eighteenth-century traders, missionaries, and explorers traveled around the world, they encountered peoples whose history seemed longer than the Bible would allow and plants and animals that were often very different from those at home. And if people looked different from one another on different continents, yet all came from the same ultimate source, Adam and Eve (which most people believed quite literally), then the existence of change was obvious. It was but one step for many scholars to consider all life as susceptible to change. Animal fossils, excavated in contexts that gave confidence in their great age, called into question the Great Chain of Being and its fixity. Fossils that were almost, but not quite, like modern populations also raised questions about fixity, particularly when they were of familiar animals, such as horses. And finding fossils of entire extinct animal lineages such as dinosaurs must have evoked wonder at first, and later the idea of extinction itself, which is the antithesis of fixity.

The Swedish botanist Carl von Linne (1707–1778), writing under the Latinized name of Linnaeus, standardized the rather haphazard early taxonomy (classification) into a scientifically based system. He classified each species, or group, as a member of a genus (plural genera) and added two more hierarchical levels, classes and orders. Each more inclusive level was based on more generalized traits. Two thousand years of classical thinking and knowledge, combined with considerable fieldwork by Linnaeus himself, formed the basis of modern taxonomy (Eckhardt 2000). Linnaeus’s classification system has its inconsistencies, and scientists have learned a great deal more about taxonomic principles since the eighteenth century, but his system is so embedded in scientific communication that we continue to use it, warts and all. Although Linnaeus was a firm believer in the fixity of all species when he began his work, in the last edition of his famous Scala Naturae (1758), he began to weaken in his belief that all species were fixed and unchangeable. Linnaeus believed that fossils and the Bible could coexist.

Jean Baptiste Lamarck (1744–1829) is best remembered for his theory of acquired characteristics. Along with most scientists during the late eighteenth century, he believed that life changed, progressively, from simple to complex and from imperfect to perfect, all based on the needs and will of the organism. Although Lamarck never used giraffes as an example (Shanklin 1994), modern models use the giraffe’s neck to explain these Lamarckian principles. For example: “In Africa, millions of years ago, there were animals that resembled modern okapis, long-legged herbivores with small heads and short necks. These early okapis survived because they ate tremendous amounts of leaves from bushes and short trees. But the environment cooled, the bushes disappeared, and
leaves on trees were the only source of food. Browsing animals ate the lower-growing leaves, leaving more leaves higher up in the trees. The okapis, faced with a need to get food and a will to solve their problem, stretched their necks just enough to get to those elusive leaves. Neck-stretching okapis acquired slightly longer necks and were able to pass on this trait to their offspring, who then had slightly longer necks than their parents. This continued until the okapis eventually became giraffes with very long necks.”

End of story. In other words, Lamarck believed that a characteristic acquired during individual lifetimes, like a slightly longer neck gained through stretching, could be passed on to offspring. Given what little was known at that time about the causes of biological change, his idea was not ill founded, just incorrect.

Nineteenth-Century Ideas

By the nineteenth century scholars believed that the Earth was considerably older than 6,000 years. Some suggested it was at least 75 kyr old. Geologists embraced the principle of uniformitarianism, the doctrine that the processes that form and change the Earth today are the same processes that were operating in the past. This principle replaced catastrophism, the doctrine that life on Earth was the result of many catastrophic events, each one wiping out the remains of the previous event and replacing older life with newly created life.

Two major nineteenth-century scientific thinkers, Charles Darwin and Gregor Mendel, were each surrounded by like-minded colleagues who were prepared to accept explanations for biological change. Charles Darwin (1809–1882), a member of the English upper middle class, discovered the principles of natural selection. Gregor Mendel (1822–1884), an Austrian monk working in the garden of a monastery in what is now the Czech Republic, discovered the principles of inheritance. The work of these two men, along with the development of the theory of mutations by the Dutch botanist Hugo de Vries (1848–1935), formed the three cornerstones that explain evolution: inheritance, natural selection, and mutations. The next section looks at these principles in the chronological order of their discovery, as each builds on knowledge of previous discoveries.

Modern Synthesis

Although the basic principles of evolution were known by the beginning of the twentieth century, the details and the synthesis of inheritance, natural selection, and mutations have occupied the thinking and experimental world of many scientists for the last hundred years. The modern synthesis took place in two parts, the first a necessary precursor to the second. In the first round, R. A. Fisher (1890–1962), a British statistician and geneticist, J. B. S. Haldane (1892–1964), a British biologist, and Sewall Wright (1889–1988), an American statistician and geneticist, wrote technical papers that were vital to establishing evolutionary principles and theory. However, they were so technical that no one could read them! It took a second threesome—Russian-born geneticist Theodosius Dobzhansky (1900–1975), German-born ornithologist Ernst Mayr (1904–2005), and American paleontologist George Gaylord Simpson (1902–1984)—to expand the mathematical predictions and explain them so that they were understandable (Ridley 2004). These three population-minded biologists/geneticists gave us the modern synthesis in the mid-1950s.

Although the modern synthesis has had its critics, it did remove a good deal of “mythology” about evolution that was rampant in the first half of the twentieth century. The study of evolution also became far more field oriented as a result of these critics. For example, skeptics had a right to wonder if X-ray induced mutations on fruit flies in a laboratory were sufficient evidence of mutations as a major cause of evolutionary change.
What Is Evolution?

Mendel's Heredity

Before scientists could accept the causes of biological change, they had to understand the principles of heredity, or inheritance, and the reasons why populations are conservative, changing relatively little from one generation to the next. Mendelian inheritance is the first cornerstone of our modern understanding of evolution. Let's look at the context of Gregor Mendel's pioneering discovery of this major key to understanding what makes evolution work.

When Mendel was working in his monastery's garden, he was surrounded by fellow clergy who were also involved in scientific research, because monasteries were both scholarly and religious institutions at that time. In addition to theological training, Mendel had studied university-level math and botany; this scientific training was very pertinent to his eight years of experiments. (See Highlight 3.1, on pp. 44–45.)

Mendel chose to work with the common garden pea, contrasting seven pairs of highly visible traits. Figure 3.1 visually contrasts those traits: seed texture (round or wrinkled), seed interior color (yellow or green), seed coat color (gray or white), ripe pod shape (inflated or constricted), unripe pod color (green or yellow), flower position on stem (axial or terminal), and flower stem length (long or short). When there are two and only two forms of a gene for a particular trait, each is called an allele. (There are a few exceptions to this “two and only two” rule, such as the genes that determine ABO blood types.) For example, long and short are the alleles for the stem length gene. Mendel started with 70 true-breeding plants that showed the same singular form of each trait, generation after generation; for example, plants with long flower stems, when allowed to self-fertilize, produced only long flower stems every generation. In succeeding

Figure 3.1

Mendel's Seven Pea Plant Traits. The seven traits Mendel used in his famous hybrid experiments are shown with their possible variations. What traits will the offspring show when a yellow, wrinkled pea is crossed with a green, round pea? What traits will the next generation show when those offspring plants are cross-pollinated?

Source: Adapted from Park (2003).
In Mendel’s Garden

GREGOR MENDEL (1822–1884) WROTE AN ARTICLE in 1866 titled “Experiments in Plant Hybridization,” not knowing that his work would eventually become the basis for understanding heredity. Farmers at the time knew how to hybridize plants and animals, but they did not understand the underlying principles of heredity. Mendel, a self-avowed plant hybridizer, discovered those principles from doing experiments on ordinary pea plants and outlined the principles in his 1866 article. He did not know that the principles he discovered were applicable to most of life, giving science a generalizing principle to equal that of Darwin’s natural selection. It was 34 years later that three scientists independently rediscovered the principles Mendel outlined.

Mendel’s choice of experimental plants was just plain good luck, because peas are normal sexually reproducing plants with the equivalent of sperm and ova that produce seeds. When Mendel later repeated his experiments on another plant, the hawkweed, the results were very different, and he might have deduced that each plant species was different in hybrid behavior. What he didn’t know at the time was that hawkweed is an aberrant plant with respect to reproduction.

Born into a peasant farming family in 1822 in what was then Austria but is now the Czech Republic, Mendel learned about fruit tree and plant improvement during his childhood. He went into the priesthood because he knew he would not inherit the family farm and because it meant freedom from financial troubles in the outside world. In addition, in those days, monasteries were places of intellectual camaraderie, with priest-scientists as common as priest-philosophers. Both the monastery and the high school where Mendel taught biology had libraries with standard books on agriculture, horticulture, and botany, so to some extent he was a self-taught botanist. Before launching his experiments, he spent two years taking university-level courses on artificial pollination, physics, and math (including what we now call statistics). At that time plant hybridization as an aca-

The location of the garden in which Mendel conducted his famous hybrid pea experiments is indicated by an X on this depiction of the monastery. The monastery has been restored and made into a museum celebrating its famous former resident. Contrast this “laboratory” (the garden and a small inside room) with botanical laboratories today.

Source: Based on Orel, V. 1996. Gregor Mendel, the first geneticist. New York: Oxford University Press.
demestic field focused on discovering the origins of particular species. People thought that one way to form new species would be through hybridizing two existing ones. Mendel's work with hybridization was in line with the interest in the origins of particular species. His finding of regularities of trait inheritance was an unexpected bonus.

Mendel's experiments began in 1856, three years before the publication of Darwin's *The Origin of Species*. He began with a small garden, where he ultimately planted almost 28,000 peas, and a greenhouse where he grew a few plants in each series of experiments to guard against insect pollination. Mendel's journals do not indicate precisely why he first planted peas, though some historians of science claim he was looking specifically for the range of traits that offspring of hybrids showed. Because his papers were burned after his death, we do not know much about his thinking at this time. After carefully counting the variations that occurred during seven generations and crossing thousands of plants using the chosen traits, he discovered that the "units of heredity" obeyed statistical laws. These numbers must have convinced him of the logic of the principles he deduced.

During eight years of experimenting, Mendel found some traits remained stable throughout (we would call them "fixed"), but that 14 traits showed alternative forms. He chose 7 of these traits for crossing and recrossing. (See Figure 3.1.) In his first experiment he crossed the two seed shape alleles, round and wrinkled. The shapes of the pea seeds in the next generation were uniformly round. But the next generation, of self-fertilized round-seeded hybrids, produced 5,474 round seeded peas and 1,850 wrinkled-seeded peas, or the now famous 3:1 ratio. Mendel referred to the round seeds as "dominating" and the wrinkled seeds as "recessive." He ran seven generations of experiments with each cross, crossed different traits (color, stem length, etc.), and combined traits and crosses. In each step he counted traits and reduced each to a ratio of dominant versus recessive.

Why did 34 years lapse between the time Mendel's paper was published and the rediscovery of his principles? Granted, the paper was in an obscure journal; but about 140 copies were exchanged with other local journals, and Mendel was cited 11 times during this period, suggesting his work was not totally unknown. Several factors seem to have caused the long delay. Mendel was a priest working alone in a monastery, not a professor of biology at some mainstream university. Most scientists come upon earth-shaking ideas not while reading journal articles, but rather in talking to fellow scientists. Mendel did not have this luxury. Also, although Mendel was able to duplicate the ratios and principles established for peas in four-o'clocks (a flower), he was not able to duplicate them in other plant species; this may have made him wonder about possible generalizing principles established in the pea study. Several years after his pea work, Mendel was appointed head of his monastery, and he had no more time to do experiments. So, in essence, he stopped being a scientist. Some historians of science also point to the impact of Darwin's work after 1860 and suggest that there was no room in scientific circles for another "harebrained" radical theory. But perhaps most important is the fact that Mendel wrote the original paper for horticulturalists, and even those interested in heredity and generalizing principles saw his paper as being on plant hybridization. In other words, Mendel may have seen the generalizing principles, but others did not, perhaps because they were not looking for them in a paper on plant hybrids. His discoveries were not a revolution within a discipline, however, but the beginning of a new discipline—genetics. By 1900 three scientists working independently rediscovered the laws of heredity, discovered Mendel had worked out the laws 34 years earlier, and gave him credit for it. By then hybridization was no longer an interesting topic to study; heredity was.

The last half of the nineteenth century may have witnessed two harebrained theories, but once Hugo de Vries found that corn genes behaved the same way pea genes did, once Darwin's ideas were accepted as the molder of evolution, and once Thomas Hunt Morgan found that chromosomes in fruit flies behaved the same as chromosomes in peas, what can be called the "genetic theory of natural selection" became the generalizing principle of evolutionary studies.

**Sources**
Mendel discovered four principles (sometimes called laws) that established the genetic basis of biological inheritance:

1. The units of inheritance are discrete particles, remaining discrete generation after generation; they do not result from blending of units from parents, nor will they blend in offspring, though they may look as if they do. In Mendel’s day it was widely accepted that hereditary units from parents mixed so that offspring received an amalgam of traits: A tall male mating with a short female would produce medium-sized offspring; a light-skinned female mating with a dark-skinned male would produce offspring with medium-shaded skin. Mendel showed that although the theory of blending seemed logical, it was incorrect. For example, the inside seed color of peas occurs in two varieties, yellow and green; but no matter how many hybrids or back-crosses Mendel bred, they were never chartreuse. Mendel called these units of inheritance “factors”; today they are called genes.

2. Individuals inherit one discrete unit, or gene, from each parent. The units are located on paired chromosomes; the pairs separate during sex cell replication, and each chromosome lines up with the appropriate chromosome from the other parent during fertilization. Mendel called this the principle of segregation (see Figure 3.2). For example, the inside seed color trait is located on a particular spot on each of two paired chromosomes. After undergoing replication, the two paired chromosomes separate, each carrying one seed color gene. One of these chromosomes will then pair with the chromosome carrying the seed color gene from the other parent.

3. The genes for different traits are inherited independently, as long as the genes are on different chromosomes. In other words, according to the principle of independent assortment, whether a pea plant has a long or short stem has no effect on whether it has green or yellow seeds, because the genes for these two traits are located on different chromosomes.

4. Individuals receive one gene from each parent for each trait, even though some genes may be recessive. In the case of each of the seven pairs of genes in Mendel’s pea experiments, one form (allele) often masked the form of the other in subsequent generations. This is the principle of dominance and recessiveness. Relative to the two possible pea seed colors, yellow and green, a plant’s seeds will be green if it gets two green alleles from its parents, yellow if it gets two yellow alleles—and yellow if it gets an allele for each color. Why? Because yellow seed color is dominant over green. However, even if a plant with two different seed color alleles has yellow seeds, it may pass on some green alleles to its offspring in the next generation. The effects of recessive genes are often masked, but the alleles do not disappear.

Through experimentation Mendel found firm support for the principles he originally deduced. By the end of his experiments, he had used an estimated 28,000 pea plants, constantly hybridizing and back-crossing every combination of alleles. His meticulous observation and his mathematical skills were the backbone of his study; he could document each generation with ratios of trait occurrences. In 1866 Mendel published his findings in a fairly obscure journal. Soon after, he became the head of his monastery and ceased doing scientific work. His findings were not known to the scientific world until the turn of the twentieth century, when three independent researchers rediscovered his principles. One of those researchers was Hugo de Vries, who is better known for his early work on mutations. Other important principles
of genetics and inheritance soon followed Mendel’s: In the late nineteenth century August Weisman (1834–1914) refuted Lamarckian theory by demonstrating that hereditary material is passed on to offspring only through sex cells (ova in females, sperm in males) and not through body/somatic cells; in 1911 the word *gene* was coined to refer to the outer manifestation of specific instructions, such as “genes for short wings”; scientists discovered that alleles carried on the same chromosomes are linked, or inherited by offspring as a unit; and the principles of inheritance were found to apply to humans as well as to peas (Grant 1991). And in 1953, with the discovery by James Watson (b. 1928) and Francis Crick (1916–2004) of the actual structure of the genetic material (DNA), modern genetics, not just the study of heredity, had been launched.

**From Peas to Humans**

Once the principles of inheritance were established for all life forms, geneticists looked for simple Mendelian traits in humans that would be analogous to green versus yellow seed color in peas. They searched for traits that required the interaction of only two alleles for the trait’s manifestation (one dominant or both codominant), that could be observed, and that would appear in the same ratios as Mendel’s pea variations. Eventually
researchers discovered many such traits—among them, for example, the presence or absence of hair on the mid-digits of fingers, the ability of the thumb to bend backward at a 50-degree angle (called hyperdexterity or hitchhiker’s thumb), and the ability or inability to taste a chemical called phenylthiocarbamide (PTC). The familiar AOB blood system is one step more complicated, with three alternative alleles—A, O, and B—where A and B are dominant over O and co-dominant with each other.

Experimenters often use the ability to taste the chemical PTC to illustrate simple Mendelian genetics in humans because PTC is neutral (people don’t care if they can taste it or not) and because culture is not known to mask or change the effect. In PTC-tasting tests each participant puts a strip of paper that has been soaked in the chemical, then dried, into his or her mouth. The taste will be bitter/salty or nonexistent. Every human individual either tastes or does not taste PTC, and groups differ in the frequency of taster or nontaster genes. For example, 97 percent of African Americans taste the chemical, but only 70 percent of European Americans are tasters (Allison and Blumberg 1959). Tasting or not tasting is observable (in its tasting sense), measurable (yes or no), and is the outer expression or phenotype of the effect of two alleles. Every human receives an allele for tasting or nontasting from each parent. The taster condition is dominant over the nontaster condition. By tradition, a capital letter (T) represents the dominant form and a lowercase letter (t) represents the recessive form. Neither taster nor nontaster condition has subjective superiority or inferiority, nor does the dominant form necessarily occur more often than the recessive form. (See Figure 3.3 for the genetic possibilities for this trait.)

If an individual receives a taster allele from each parent, the individual can taste PTC and the individual’s genotype is written as TT. A genotype is the genetic endowment that individuals receive from their parents for particular traits. If an individual receives a nontaster allele from each parent, the individual cannot taste the chemical and the genotype is tt. In both cases, because the genotype is composed of the same form of the allele, the individual is considered homozygous for this condition. If the individual receives a taster allele from one parent and a nontaster allele from the other, the genotype is Tt and the individual is considered heterozygous for the condition. A person needs only one taster allele to be able to taste the chemical, so all heterozygotes are tasters.

For each simple Mendelian trait, the phenotype can be observed; the genotype of the recessive form (e.g., nontasting) is obvious, because each parent must contribute a recessive allele for the individual to be recessive; but because the effects of dominance mask the effects of recessive alleles, the genotype of the dominant form can be either homozygous (with each parent contributing one dominant allele) or heterozygous (with each parent contributing a different form of the allele, the effect of the recessive allele being masked or hidden). Mendel’s principles work for humans as well as for peas.

Geneticists have found more than 4,000 of these simple Mendelian traits in humans. Some harmful genes are inherited as dominants, such as those producing achondroplasic dwarfism and brachydactyly (having very short fingers); some are inherited as recessives, such as
those producing cystic fibrosis and sickle-cell anemia. For the trait to be manifest, a person needs to inherit only one of the harmful genes if it is dominant, but two if it is recessive. Geneticists have also learned that the vast majority of traits are more complex than this. Very seldom does a gene make a trait by itself. Traits often result from the action of more than one set of alleles. Or the same set of alleles can affect two or more traits. Finally, environment can affect traits, as in the cases of certain childhood diseases and sunlight: Disease during an individual’s growing years often results in shorter stature than genetically expected, and skin color can be darkened by the sun. Skin color, brain size, stature, and head shape are traits that have complex heredity. Some exceptions to Mendel’s principles have been found as well. For example, as implied earlier, linked traits on the same chromosome usually are not subject to the principle of independent assortment.

**DNA, Genes, and Cells: From Atoms to Populations**

Once the units and principles of inheritance were identified, scientists turned to studying genetic material at both the micro/molecular level (the gene/allele or smaller) and the macro/population level (the species or larger). Principles at both levels are now relatively well understood. Here we look at genetics at the molecular level. In Chapter 15 we’ll briefly consider the population level of genetics, focusing on gene pools of modern human populations and how they change over a few generations.

A discussion of genetics can either start with atoms and end at populations (moving from smallest to largest units) or start with populations and end at atoms (largest to smallest units). The major steps from the smallest to the largest units in a linear genetic model are: atom to molecule (amino acid to protein or nucleic acid to DNA) to gene to chromosome to cell to individual body to population.

All substances are made up of atoms. The most common of the 116 kinds of atoms are the familiar carbon, hydrogen, oxygen, and nitrogen. Atoms join to become molecules, and the molecules common to living things are carbohydrates, lipids, proteins, and nucleic acids. Proteins are long chains of amino acids; in all of life on Earth, there are only 20 amino acids, but they differ in type, amount, and sequence for each species. Through a complex process of protein synthesis, humans become humans and chickens become chickens.

It is molecules of DNA that provide protein synthesis codes for all biological organisms. DNA exists in all plants and animals. Twenty-seven different kinds of ancient DNA were recently extracted from soil in Siberia and New Zealand, originating from plant roots and animal feces/urine deposits (Stokstad 2003). DNA directs protein synthesis within cells, and all cells in a species carry the same basic codes. For example, human skin, liver, and blood cells have the same basic DNA. It has been discovered that although the DNA sequence is the code that specifies which amino acids produce which proteins, messenger RNA (mRNA) provides the intermediate one-way transfer of that information. Because proteins are the major constituents of all body tissues, protein synthesis is vital to life. Proteins vary in function: Some form the structure of tissues, such as collagen in bone, while others form connective tissues. Hormones and enzymes are other kinds of proteins; hormones stimulate various kinds of cellular activity, and enzymes begin or enhance chemical reactions.

The cells of all living organisms can be called the basic units of life, because cells are the smallest units that perform all necessary life functions. It is in the nucleus of the cell that the units of inheritance reside. By using special microscopic techniques, scientists can see chromosomes in cell nuclei as long, narrow bodies with alternating light and dark bands (see Figure 3.4). Each species has a specific number of chromosomes in each cell; mammals, the taxonomic order to which humans belong, have between 40 and 80 chromosomes. Humans have 46, often referred to as 23 pairs because at replication they can be matched according to shape, size, and genetic material. After replication each pair moves apart, and one chromosome from each pair becomes part of a new cell.
DNA consists of very long strands, twisted around each other in a double helix shape. By contrast, an RNA molecule has only one helix. Figure 3.5 shows a simplified version of the famous Watson and Crick (1953a, 1953b) model of DNA. As the figure illustrates, the DNA strands are made of alternating sequences of phosphate and sugar units. Attached to each sugar is one of four bases: adenine (A), thymine (T), guanine (G), and cytosine (C). Biochemists had long known what DNA was made of, but in 1953 James Watson and Francis Crick figured out how the bases were connected to the sugar and phosphate units. Although their first attempt at modeling was totally wrong, they persevered until they lighted on the famous double helix. The two DNA strands are held together by chemical bonds that connect the bases: T bonds only with A, and G bonds only with C. When scientists speak of DNA sequences, they are referring to sequential arrangements of the four bases, such as AAGGTATCCAGACA. It is the order of the bases that gives DNA its specificity. It has been estimated that there are 3.2 billion base pairs in the human genetic code (Deloukas et al. 1998). There are 64 possible three-letter combinations of the four bases that provide the genetic code for the 20 amino acids needed for protein synthesis; CTT codes for glutamic acid, for example. This may sound like a very small amount of possible variation for all of life—but, by analogy, although the English language uses only 26 letters, a large dictionary contains a million words and a large library houses many millions of books (Relethford 2001a). The fact that there are only four kinds of bases in any DNA sequence (A, T, C, and G) means that the sequences of any two species can differ on average by no more than 25 percent. Human DNA and daffodil DNA are 35% identical, and human DNA and chimpanzee DNA are 98 percent identical, all because of common ancestry and the fact of only four bases (Marks 2000b). (See Highlight 3.2, on pp. 52–53, for a description of the Human Genome Project, the Iceland study, and the role of anthropologists in this venture.)

A gene is a segment of DNA on a chromosome; it gives certain instructions that will eventually be expressed as a trait in the individual who carries it and possibly in that individual’s progeny. For example, the interaction of two alleles on a specific spot on the
ninth chromosome determines a person’s blood type (Marks and Lyles 1994). The average number of genes on each human chromosome is about 1,500, but this number varies by the size of the chromosome and the density of the genetic material (Klein and Takahata 2002). Of the estimated 3.2 billion base pairs of the human genetic code only around 25,000 (an educated guess) are genes, giving instructions that will eventually be manifest (Wade 2003a). Genes are separated by long stretches of DNA with no role in coding proteins. The label junk was assigned to this noncoding part of DNA before its extent was known. Many now believe it has necessary functions, such as repairing or turning on genes. Researchers estimate that as much as 97 percent of nuclear DNA is noncoding, meaning that it is not building proteins and is not susceptible to natural selection (Groves 2001; M. Jones 2001; Klein and Takahata 2002). In humans each chromosome contains about a meter of DNA, tightly coiled upon itself many times; humans have 23 pairs of chromosomes, so every human cell is estimated to contain 46 meters of DNA (Margulis and Sagan 1997).
Highlight 3.2

In the News: Iceland and the Human Genome

THE HUMAN GENOME PROJECT (HGP) officially began in 1990 and has been a high-profile and high-interest enterprise ever since. Everything from the politics, the skirmishes between public and private labs, and the sheer magnitude of the project to the potential scientific results has attracted attention and controversy. But at least some of the waiting is over, because the first stage—sequencing the estimated 3.2 billion base pairs—is completed. Pithy phrases like “the book of life” and “nature’s genetic instruction manual for making and maintaining human beings” and “a glimpse of an instruction book previously known only to God” were among those in the air in the summer of 2000 at the announcement of the project’s “first stage conclusion.” As of July 10, 2003, the first human chromosome—number 7—had been completely sequenced. Its 153 million base pairs represent 99.4 percent of the sequence. Researchers tackled this chromosome first because it is thought to be associated with various diseases.

From the beginning, there was a difference of opinion on whether the research should be public, funded mainly by the U.S. government but internationally supported and with results free for anyone to use, or private, funded by private enterprise and with the database restricted to those who paid for its use. The HGP is the public version. So far it has cost about $3 billion and involved 62 scientists from 13 labs all over the world, each sequencing a particular part of the genome. The private organization is Celera Genomics in Rockville, Maryland; it reportedly spent $330 million in private funding and used its own method of sequencing.

What is this project, and what does it mean to science and to the ordinary person? In simple terms, the human genome is a map of the genetic makeup of the human species. In every cell in every human body (except in the sex cells), there are 23 pairs of chromosomes, and on each chromosome there are genes that have certain functions, the genes being made up of thousands of base pairs. Though not every base pair is identical throughout the species (we each have our own unique genome), the sequencing of the genetic material is the same. The data came from 1,056 individuals in 52 world populations. Stage two will locate genes and determine their functions. Until this stage is completed, scientists do not know where one gene begins and then ends on a chromosome.

Rather than reinvent the wheel, once the genome is sequenced for humans, scientists will use the genomes from other species that have been completely sequenced so far—genomes for roundworms, fruit flies, mice, rats, mosquitoes, and several fish—as a start in determining gene function. (The chimpanzee genome is finalized, and the gorilla genome has begun to be sequenced.) To put it simplistically, the mouse genome can be laid next to the human genome, and where mouse functions are known for individual genes, scientists can hypothesize about parallel locations and functions in humans. Some genes have already been located and their functions identified.

Once scientists have located specific genes and ascertained their functions, stage three, applications using the new knowledge, can begin. Many people hope that the new knowledge will reinvent medicine, allow physicians to predict diseases, correct disease-carrying genes, and eventually eliminate diseases in individual patients, perhaps forever. Some diseases, such as sickle-cell anemia, can be explained by simple Mendelian genetics. Other conditions, such as Down syndrome, occur when there is an extra chromosome. Still other diseases are additive and need the interaction of many genes before they are manifest. These kinds of diseases are common, yet their causes have been elusive so far because scientists don’t know where the genes are and which ones they are, much less how many are involved. Diabetes, heart disease, and some types of cancer are on the list. If a physician working with a geneticist who is a specialist in genome reading can identify genetic susceptibility to a particular disease in a patient, the physician can counsel the person about environmental factors (stress, overweight, lack of exercise, smoking, etc.) and perhaps forestall the onset of the disease. Or, a bit down the road, genetic replacement or removal of the faulty gene might be successful.
For some time to come, gene therapy will be exceedingly expensive, and this is where anthropological ethics will come into the picture. Think of the problems that would develop if everyone in the world survived until age 200. If only certain people could have gene therapy, who would they be? Who would make the decisions? Anthropologists also need to be involved in discussions about the ethics of making individual genomes public. If a physician has a patient’s genome for consultation purposes, can insurance agencies and employers demand that information? Sequencing the human genome has resulted in so much complicated data that a new “index” to the “book of life”—called the HapMap—will allow geneticists and physicians to narrow down entry points. This strong biomedical focus is encouraging for the application of the results of the years of pure research.

One interesting and highly debated project involving the use of a database resulting from a genome project is under way in Iceland. In December 1999 an Icelandic company began to collect DNA from all 270,000 Icelandic citizens to link their genetic profiles with their health and family tree records; the parliament had approved the creation of the database the year before. The stated goal is to make predictions about diseases caused by faulty genes. Because Iceland’s citizens are relatively homogeneous, being mostly descended from Scandinavian and Norse groups and having experienced little immigration over the years, Iceland is an excellent laboratory for studying genes.

But there is fear that the genetic information will not be used solely for the stated intention. Some argue that the safeguards protecting privacy are not adequate and that the database might be sold to pharmaceutical companies with the company that collected the data making a profit. At least 5 percent of citizens have asked that their data be excluded.

Iceland may be the first country to “go genetic” in terms of a biological database, but others may follow. Each country must decide whether the ethical problems are worth the potential rewards.

organisms grow or cells replace themselves, the DNA strands unwind from each other by breaking at the bonds between the bases. Each strand replicates itself by picking up nucleotides (chemical compounds of salt, sugar, and bases) within the cell; normally the replication is exact, and whatever genetic material is on each strand is inherited as a unit.

During regular body cell replication, called mitosis, all 46 chromosomes come together at the equatorial plane of the cell (the analogy is with the equator on a globe) and replicate themselves, temporarily forming 92 chromosomes. The cell then divides into two, each daughter cell receiving a full complement of 46 chromosomes. It takes 30 to 90 minutes to complete this division. Cells are at rest most of the time, doing what they are supposed to do as muscle cells, skin cells, etc.

The process of replication differs in several ways between body cells and sex cells. In human sex cell replication, or meiosis, there are two cycles of cell division. In the first cycle, all 46 chromosomes line up at the equatorial plane and replicate themselves, again temporarily forming 92 chromosomes; the two sets of paired chromosomes then separate from each other and move to opposite sides of the nucleus, and the cell splits into two cells, each having 46 chromosomes. The second meiotic division separates newly paired chromosomes so that each newly formed sex cell (ovum or sperm cell) has 23 chromosomes. These will combine with the 23 from the other parent to form a zygote, a single cell from which a human being develops. That is, the zygote that results from fertilization has 23 pairs, or 46 chromosomes. If it were not for the second reduction division, a sperm and ovum would produce a zygote with 92 chromosomes. (See Figure 3.6.) Because of random assortment and segregation during this complex process, a zygote never has the same genotype as either parent. In fact, it has been estimated that the total number of possible genetic combinations from any human mating is in the trillions.

The last important difference between body cells and sex cells is that although all body cells in each organism are identical (unless there have been body cell mutations), all sex cells are different from one another. Once a zygote is formed, however, it divides by mitosis over and over to produce the millions of cells in the individual's body.

Geneticists classify two basic types of genes: structural and regulatory (Jacob and Monod 1961). Structural genes give instructions that eventually are expressed in observable traits, such as protein-coding instructions for skin tissue. Regulatory genes function only to control some structural genes, governing the timing of certain events in an organism's life by turning on or blocking effects. For example, in human females, regulatory genes are responsible for triggering female sex cells (eggs) that were originally formed before birth. In males, regulatory genes trigger penis and testicle growth, the development of facial hair, and lowering of the voice. On the species level, many experts believe that much of the difference in structural genes between humans and chimpanzees or bonobos was caused by mutations on regulatory genes, particularly as they affect brain activity, at the time the last common ancestral group split into separate lineages (King and Wilson 1975; Pennisi 2002).

**Darwin's Natural Selection**

After finding he had little talent and no taste for either medicine or the ministry, the 22-year-old Charles Darwin signed on as the gentleman naturalist on HMS *Beagle*, a ship that was exploring the coast of South America to establish longitude measurements. While on the 5-year voyage from 1831 to 1836, Darwin read books by the British economist Thomas Malthus (1766–1834) on the problems of overpopulation and Volume 1 of Charles Lyell's (1797–1875) *Principles of Geology* (1830) on uniformitarianism and the great age of the Earth. Apparently Lyell's Volume 2 arrived by mail boat when Darwin arrived in Montevideo (Gould 2002). The ideas of both Malthus and Lyell influenced Darwin’s later biological observations and thinking, because at the time of the voyage, he believed in Lamarck’s theory of acquired characteristics. The long voyage allowed Darwin to observe the variability that existed within many of the species he encountered, variability that became the anchor for his lifelong effort to explain evolution. In
particular, he noted tortoise shape and size variability and finch beak size and shape variability on the Galapagos Islands, even if at the time he did not understand the significance of these phenomena. His findings can be summarized in the phrase identified with him, natural selection, the second cornerstone of our understanding of evolution. Natural selection is so straightforward and easy to understand that Thomas Huxley, a scientist and contemporary of Darwin, wrote that he felt “extremely stupid not to have thought of that” (Huxley 1901).

Darwin was hampered by a lack of knowledge that genetics, heritability, and mutations comprise the ultimate source of all variation (as well as by his belief in Lamarckian evolution and blending). He wrote about unusual traits never seen before, calling them “sports,” but he did not understand their nature or importance. The thinking that led Darwin to his theory of natural selection went something like this: (1) All species are capable of producing more offspring than their food resources can sustain. (Here, he was obviously influenced by Malthus, who wrote that animals were able to increase their
numbers exponentially while food resources increased only additively.) For example, Darwin reasoned that a single breeding elephant pair would have 19 million descendants after 750 years, even though elephants are slow breeders, unless something stopped the exponential increase (Morris 2001). (2) Biological variation occurs within all species, and this variation is inherited. (3) Because there are more individuals born than there is food to sustain them, there is competition between individuals for sustenance. (4) Some individuals possess variations that are favorable for survival, such as additional speed to escape predators, resistance to disease, or good vision to locate food resources. These individuals are more likely to live longer and produce more offspring than those who possess variations less favorable to long life and offspring production.

To understand Darwinian natural selection and its role in evolution, we need to make a distinction between selection’s operation and its effects. Selection operates through net reproductive differential and survival, or differential reproductive success—that is, through the differing numbers of offspring that survive among all individuals in any given species. If an individual has more than the average number of surviving offspring, then that individual has positive reproductive differential. By contrast, if an individual has fewer than the average number of surviving offspring, then the individual has negative reproductive differential. Most biologists use the criterion of “offspring surviving until the age of their own reproduction” to measure net numbers. Because individuals within any species vary reproductively, they differ in their genetic contribution to the next generation. If, for example, a particular wolf produces twice as many offspring as the average wolf, then that wolf’s genetic contribution to the next generation is double the contribution of the reproductively average wolf. If a wolf has no offspring, then it makes no genetic contribution to the next generation’s gene pool.

What causes animals—wolves, to continue the example—to have positive or negative reproductive differential? Differential depends on the total phenotype of each individual wolf at any particular time. The unit of consideration is each individual “total wolf,” relative to all other wolves, and it is usually not possible to pick out one specific trait that is responsible for the reproductive differential. Factors in positive reproductive differential may include looking attractive to the opposite sex, being able to get and maintain a place to raise a brood, having the ability to produce twins instead of one offspring at a time, or, in the case of humans, being able to make people of the opposite sex believe you can support a spouse and numerous children by virtue of intelligence or wealth. Behavioral traits, as long as they are variable, heritable, and can affect differential reproductive success (i.e., have a biological base), are subject to natural selection as well. So a wolf who inherits the ability to be a better mother than other wolves will likely raise more of her cubs to maturity and thus will tend to pass on her talent for mothering. Traits that enhance reproductive success differ through time as circumstances change, however. Also, what favors reproductive differential may be very different from one species to another.

The effects of natural selection are often difficult to observe and/or measure. Darwin often wrote of adaptation, suggesting that in the long run, populations would become better adapted to their existing environments due to natural selection. He believed that the finches on the Galapagos Islands had beaks that varied relative to different island environments, and he argued that specific beak shapes were adaptations. He also pointed to the fact that many animals that live in cold climates have thick fur that makes them well adapted to that environment. If there are two wolves in northern Canada during a particularly cold winter, one with a thick pelt of fur and the other with a scraggly coat, the wolf with the thick fur will probably live longer and produce more offspring than the scraggly-coated wolf. In time, an entire population of thick-furred wolves that are better adapted to the cold Canadian winter will evolve. Theoretically, all traits that lead to better adaptation enhance survival and confer subsequent reproductive advantage. Some adaptations have clear benefits: Bats use echolocation to find insects, legs and feet evolved for walking, and eyes and ears evolved for seeing and hearing.
These are, of course, the large-scale macroevolutionary adaptations that Darwin was attempting to explain. More recently, scientists have found that much small-scale micro-evolutionary change is neutral, showing no correlation to differential reproductive success (Stearns and Hoekstra 2005). In many cases it is not clear what is being adapted to or just how the adaptation is “better.” Because natural selection acts on many traits at the same time, effects on some traits will often mask effects on others. Scientists can measure complexity by counting genes or traits, but it is subjective to claim that something is better just because it happened. And since more than 90 percent of all species that have ever lived have become extinct, natural selection does not always better adapt populations to existing environments.

Darwin believed in another kind of selection, sexual selection. A lion’s mane, a peacock’s tail, a human female’s large breasts—all are expensive to develop and to carry around; but if the reproduction of that lion or peacock or human female is enhanced by having a bit more of that trait, perhaps members of the opposite sex will prefer that individual, the gaudy show as being a sign of good health and a worthy mate. In a recent experiment one scientist used swallows, cutting off the tails of one third of the males at mid-section. On another third of the swallows, he pasted the cut-off tail ends; thus, there were one-third long-tailed, one-third medium-tailed (normal), and one-third short-tailed male swallows in the experimental group. At breeding time the females preferred the long-tailed males. It has been said that natural selection adapts organisms for survival in their existing environment, but sexual selection adapts them to the needs of obtaining a mate and reproducing (Lewin and Foley 2004).

This relatively new idea about sexual selection is that females are selecting males (as sex partners) on the basis of their “good genes,” as represented by morphological features such as long tail feathers or beak size that might translate as “good health.” But one interesting experiment by Nancy Burley on the zebra finch suggests there is more to it than “good genes.” In her experiment she made different colors of paper hats and fitted them on male birds. The female birds preferred mating with the red-hatted males. Paper hats would not occur naturally on male zebra finches so what females were selecting in this case was color, perhaps an aesthetic choice (Schulzhuitzen 2001).

Darwin was a populationist, principally because he observed so much variability in species. Although it is often difficult to know the range of variability in a species when there only a few specimens, Darwin’s dictum was that scientists should think in terms of variable groups rather than of one example or one fossil somehow being the group. Darwin was also a holist, believing selection operated on whole or total individuals relative to others in the population. After about 1900 most scientists focused on the gene as the unit for selection, but in the mid-1970s science returned to the concept of the entire individual (Mayr 2000). To understand the impact of natural selection, however, you must keep in mind that although selection operates on individuals through reproductive differential, it is the population or species that evolves and adapts. The unit of selection is the individual, but the unit of evolution is the population.

Darwin wrote a short outline of his ideas in 1842, but he did not publish the full treatise until 1859. He apparently thought he had not collected enough data to support his hypothesis and had fears about addressing such a controversial issue. Indeed, his ideas were not met with universal delight—mainly because they did not include the notion of progress and they involved extinctions. Yet at least one other scientist, Alfred Russel Wallace, came to very similar conclusions about natural selection at approximately the same time as Darwin. In 1858 both men received credit for their work when their joint paper was read at the Linnaean Society of London (Desmond and Moore 1991).

Thomas Hunt Morgan (1866–1945), an American biologist, was initially critical of both natural selection and Mendelian inheritance. But after testing the mechanisms of both heredity and selection on fruit flies in his laboratory, he reversed his judgment and became a staunch proponent of modern evolutionary theory. It was Morgan who combined Mendel’s and Darwin’s famous laws in 1916 (Marks 2002).
Darwin’s foremost contribution to our understanding of evolution was his explanation of how it works through natural selection, but he made additional contributions. He denied any notion of “progress,” so beloved of previous scientists, instead focusing on trends. He also believed that evolution was branching or bushy. From Aristotle to Lamarck and beyond, scientists who believed in change (evolution) had thought of it as linear, a kind of “teleological march toward greater perfection” (Mayr 2000). Finally, Darwin rejected “typological thinking” in favor of population thinking, believing that it was the population that would show the effects of natural selection. He could observe that no two individuals were exactly alike in any population, but he saw that it was the population that evolved, not the individual. Finally, as a last word on Darwin, it is interesting that he used the word evolution only once in The Origin of Species, and it was the last word in the book.

The last of the three cornerstones of the science of evolution is the concept of mutations, the ultimate cause of change in hereditary materials.

**De Vries’s Mutations**

Mutations were discovered in 1886 when the Dutch botanist Hugo de Vries saw in evening primroses in an abandoned field traits that he knew he had never seen before. Darwin’s “sports” became “mutations.” Unfortunately, primroses are genetically odd, and what de Vries thought were mutations were just primrose irregularities. He was not seeing new species at all. Most of what scientists know about mutations they have learned either through experiments on laboratory animals or, in the case of humans, through funded research on medical abnormalities. The word mutation may bring to mind mishapen individuals or spontaneous abortions, but without mutations, evolution would not occur as it does. It has, however, been estimated that deleterious mutations are at least 100,000 times more frequent than beneficial ones simply because organisms are well adapted and any change is more apt to be deleterious than beneficial. Mutations should be thought of as the raw material of evolution because they are the only way new genetic material can be introduced into populations. Every biological trait that exists today in every species had its origin in a mutation somewhere in the species’ evolutionary history. Without mutations, the first life would have been the only life on Earth; there might be more of it, but it would be the same today as when it began.

Most genetic material replicates itself exactly during cell replication. A mutation is any change that occurs in the genetic material when it is undergoing replication. Partial duplication, missing chromosomes, chromosomes with missing chunks, repeated sections, and broken chromosomes that are spliced onto others are all mutations; the effects of such chromosomal mutations are usually systemic and almost always fatal in humans, because in each case so many genes are involved. However, most mutations, including the nonfatal mutations that can become part of the evolutionary record, occur at the base pair level on the DNA and are analogous to one wrong note in an entire musical score. For example, a single instance of base C could mutate to base G. In relation to the entire number of replications, mutations are rare. Expert estimates of the mutation rate for humans vary from 1 to 4 to 200 in a single individual. Most mutations do not manifest in subsequent generations (Klein and Takahata 2002; McKee 2000). However, since males produce sex cells throughout their lifetimes, while females produce and store their sex cells before they are even born, males may contribute 50 times more mutations than females do and throughout their lifetimes. Most genetic change (good, bad, neutral) can therefore be laid at the feet of males (S. Jones 2003).

What causes mutations? Under laboratory conditions fruit fly genes can mutate when exposed to extreme heat or cold, X-rays, radioactivity, or chemicals. For ethical reasons scientists do not purposely cause mutations in humans, but human genes can mutate on their own when exposed to human-related factors such as cigarette smoking, chemical wastes in the air or water, chemicals added to foods and medicines, air pollution, or human-caused radioactivity. Mutations also can result from natural factors such
as cosmic radiation, radiation from under the Earth’s crust, working with certain elements such as radium, increases in temperature, and the instability of DNA itself. Instability means that mutation patterns are unpredictable (Lowenstein 1992). Also, experiments on fruit flies have shown that radiation causes mutation rates to go up, yet fears that children of survivors of the atomic bombing of Japan would show increased expressions of radiation-caused mutations have not been borne out.

**The Tempo of Evolution**

There are two prominent models of the tempo of evolution. Darwin’s name is usually associated with the tempo of evolution called **phyletic gradualism**: the view that in general, evolutionary change occurs slowly and continuously over time. Darwin believed that natural selection operates on all variable traits, constantly favoring or rejecting one variation or another. He believed that this process results in species’ changing slowly and, in his words, becoming better adapted. The other model of tempo is **punctuated equilibrium**, a concept once called saltation and repopularized in the 1970s by Stephen Jay Gould and Niles Eldredge (1977). According to the punctuated equilibrium view, the majority of evolutionary lineages show long periods of stasis, or little change, followed by short bursts, or punctuations, of evolutionary activity. (See Figure 3.7.) There are two reasons why punctuated equilibrium became popular again. First, the two scientists who supported it saw what they believed to be stasis and punctuation for the species in which they specialized: Eldridge saw 8 myr of no change in trilobites before they became extinct, and Gould found the same lack of slow and continuous change in snails (Morris 2001). Second, many scientists were bothered by the lack of transitional fossils between major groups of plants and animal. If the tempo of evolution were fast, that would explain why those fossils were missing. Even Darwin was conscious of the large gaps in the fossil record, but he believed, as many modern scientists do, that they were due to a lack of preservation or
that the fossils just had not been found yet. This “absence of evidence” argument has been recently augmented by the fossil discovery of a number of transitional fossils, such as those of the earliest land animals.

An intensive and extensive look across the fossil record at different types of animals over a long amount of time does not point to only one tempo throughout time, place, or species. For example, certain mollusces change only during speciation events but not at all during stasis, while rodents—and some other animals—change as much between as during speciation (Stearns and Hoekstra 2005). And in fact neither Darwin nor Gould and Eldredge claimed that all evolutionary events were in only one mode (Gingerich 1984). So what tempo is more common? Current evidence concludes that the general tempo is slow and continuous change for some lineages, and stasis and bursts for others. Michael Rosenzweig further suggests that “it depends on the scale at which it is measured” (1997, 1622). Both at a microevolutionary level over a small number of generations and from a very broad perspective of millions of years, species appear to show a pattern of slow and continuous change. At the speciation level, evolution often appears to occur in a punctuated equilibrium mode. Many paleoanthropologists are not convinced the tempo has always been in only one mode for the entire human lineage (Foley 1987), and one researcher even uses the term “rapid gradualism” (Dawkins 1994). Finally, many experts contend that given the nature of the fossil record and its preservation problems, evolution will always look punctuated whether it is or not (Ridley 2004).

The Causes of Evolution

For almost 100 years scientists have understood that evolution is the result of the interaction of heredity (providing the continuity and consistency) and change (resulting in variability). Scientists have also identified the major causes or factors that interact to produce change over time: mutations, natural selection, gene flow, and genetic drift. (Whether to speak in terms of “causes,” “factors,” “forces,” or “mechanisms” is a matter of choice.) Mutations and natural selection are responsible for the vast majority of macroevolutionary events, mutations introducing the new genetic material and natural selection producing the variability that evolution works on; but in the shorter run genetic drift or gene flow may be the major cause of change in some lineages. In this section we look at the role each factor plays individually, but it is their interaction that causes evolution. Chance plays an important role in evolution through several key mechanisms: mutations are chance (random) events; the exact combination of parental chromosomes is a matter of chance; which specific individuals meet to pass on gene flow is chance; and genetic drift is due to chance and small numbers (McKee 2000).

Evolutionary Mutations

For a mutation to become established in the evolutionary record, three conditions must be met: It must occur in the right place, be the right size, and be operated on positively by natural selection or be neutral—that is, not bestow negative reproductive differential (Klein and Takahata 2002). Each condition is a kind of filter, as most mutations do not occur in the right place, most are lethal because of their size, and very few of them enhance reproductive differential. Thus, starting with a relatively large number of mutations, only a few become established in evolution. Nonetheless, mutations must be considered the raw material of evolution because they are the only known way that new genetic material can potentially enter a population. All variability was at one time introduced as a mutation.

Mutations must occur in the sex cells to have evolutionary significance. Most cells are body cells rather than sex cells, so most mutations occur during body cell repli-
The Causes of Evolution

...cation and have no effect on evolution. Because there is no connection between body cell mutations and sex cells, there is no way for one to affect the other. Estimating mutation rates for humans is very difficult, but they occur in perhaps 5 percent of sex cell replications.

Large mutations, which are by definition lethal, have no chance to become evolutionary because they do not go beyond a single generation, whereas small mutations may accumulate small effects. Mutations that are systemic and cause change in developing organs often cause spontaneous abortions; mutations that cause early death also are considered lethal. It has been estimated that of the 20 percent of known conceptions in modern human females that end in spontaneous abortions, half of these outcomes are caused by sex cell mutations (Bogin 2001a). Such an estimate is probably low, however, because many spontaneous abortions occur early in pregnancy and are probably not noticed, reported, or counted.

Evolutionary Selection

Only when mutations bestow positive reproductive differential on the individuals that initially carry them can the mutations be passed on to at least the next generation. Mutations that bestow negative reproductive differential on individuals who carry them (which is far more common) will disappear, usually in a generation or so. The action of positive selection on mutations is through individuals via their reproductive differential, but given time, the effect can change a population.

Specific small mutations that occurred originally in sex cells and conferred positive reproductive differential are all around us, given that all major biological change has taken place through this process. For example, a mutation that allowed an individual to see better in dim light than its conspecifics would likely allow that individual to feed longer, live longer, avoid predators better, and produce more offspring—who themselves could see better than their conspecifics in the next generation. In time the better-eyesight mutation would likely become fixed in the population. Even a recessive gene that began as a mutation can become all but fixed (0.997) after as few as 200 generations (Relethford 2001a). But again, most mutations neither bestow positive reproductive differential on an individual nor become established in successive generations. Most neutral mutations do not survive and even the seemingly advantageous ones merely have a somewhat better chance to survive (Klein and Takahata 2002). Mutations are not wish lists; they happen randomly. When particular mutations fortuitously occur, pass through the positive reproductive differential filter, and appear to be beneficial to a population, the population can be said to be better adapted to its environment. Not all traits in all species at any particular time are necessarily adaptive, however. Some may be linked to traits that are adaptive and may merely be “along for the ride”; these traits can be thought of as neutral, because if they were truly maladaptive, natural selection would eliminate them in subsequent generations.

One of Darwin’s more important points about natural selection was that it operates on the variability found within any species. Establishing the fact of considerable variability within all species was one of the most difficult parts of his hypothesis to establish, but once it was accepted, it became a key part of natural selection. Two types of selection operate on the existing variability in a population long after the original variations evolve as mutations. In directional selection, the mean of a trait’s variation moves in a constant direction, with succeeding generations showing short- or long-term directional change. For example, scientists observed and measured beak length in finches during drought years (1976–1977) in the Galapagos Islands (Grant 1986). Finch beak length increased during each generation, so it can be considered directional. Because beak length is showing existing variability, the change is directional selection—but perhaps of a short-term nature, given that drought is an abnormal condition in the Galapagos. (See Highlight 3.3 for a further description of
When Darwin arrived in the Galapagos, he undoubtedly saw finches; he did collect about 100 specimens. Later he wrote of their variability relative to specific islands, but he thought they were all one species and all ate the same food—he never understood their variability or the importance of their beak shape differences. On the other hand, he never claimed to understand their variability or evolution. But the Galapagos were actually an excellent place to gain insights into natural selection, and perhaps Darwin intuited more than he put in writing. The Galapagos, by being isolated, showed...
the results of one likely migration of finches from the mainland, with secondary migrations to other islands. In their new homes the finches found themselves in different geographic areas that called upon parts of the variability that existed in the original migrating group. Darwin would not have seen such island specificity in rural England, because English finches interbred on their borders, keeping the species open and variable. The Galapagos were a unique laboratory for identifying the subtle and elusive effect of natural selection, that of adaptation.

Biologists now know a good deal more about finches and the Galapagos than any scientist did in the mid-nineteenth century. It is known, for example, that finches vary in color and body size as well as in the size and shape of their beaks, which are usually described in terms such as “sharp and pointed,” “long and slender,” “short and thick,” “stout and straight,” and so forth. Finches eat different kinds of foods, such as seeds, cactus spines, mangoes, twigs, leaves, insects inside trees, and ticks from tortoises. When you see a finch with a long slender beak poking into cactus spines and another finch with a short strong beak tearing apart bark to expose insects, it is tempting to hypothesize a direct correlation between beak shape and food taken. However, because no species of finch eats only one food, the relationship is considerably more complex. Nonetheless, beak size and shape are affected by food exploitation methods.

As Darwin did not correctly classify the finches, evolutionary studies had to wait until taxonomic and distribution problems were overcome before biologists could hypothesize about the birds’ evolution. The modern model postulates three steps in finch evolution: (1) An initial group colonized one island from mainland South America, 600 miles away. DNA evidence suggests that this first group arrived about 2 to 3 myr ago. (2) When the carrying capacity of the original island was reached, finches dispersed to nearby islands and encountered different environmental circumstances, such as food resources. Beak mutations, acted on by natural selection, changed the population; this occurred on several different islands. (3) Reproductive isolation resulted in 14 species of finches in the Galapagos.

If these steps sound familiar, it is because scientists believe this is the normal way speciation occurs when geographic barriers (such as ocean and distance) effectively isolate populations. This finch speciation event may have taken only half a million years to complete.

Taking this research one step farther, Peter Grant, a contemporary American biologist, has studied the Galapagos finches for several decades, often with his biologist wife, Rosemary. Grant suggests that rainfall is the only important variable in Galapagos life. The amount of sunlight and the temperature do not change much from year to year, because the islands are on the equator. But rainfall can be quite variable, ranging from El Niño excess to normal to drought.

During five normal rainfall years, the Grants studied individual finches on Isla Genovesa, focusing on morphological and reproductive variability. A normal year has four wet and eight dry months. Finches breed only during the wet months and stop breeding when there are no more caterpillars or spiders for their fledglings. The Grants found that reproductive success was variable even in a normal
Fernandina, a Galapagos island, is home to many unusual birds, land animals, and sea creatures.

Highlight 3.3 (continued)

year; 15 percent of the population produced no fledglings, leaving 85 percent to contribute to the next generation. The key to reproductive success during normal rainfall years was not morphological variation (body size, beak shape) but experience. Experienced birds (birds in their second or later year of laying eggs) already had mates from the previous year and started breeding early, thus producing multiple clutches. Inexperienced birds had to find mates; by the time they did, they were lagging behind the experienced birds in producing first clutches. Females chose their mates on the basis of courtship behavior, which is based more on experience and age than on genetics. The upshot of these factors was that, on average, experienced birds produced twice as many fledglings as inexperienced birds.

During very wet or very dry years, in contrast, body size and beak shape were more important in reproductive success than experience was. In very wet years the cacti were smothered and died, so long-beaked finches that fed on cactus spines did not produce as many offspring as wide-beaked finches that fed on insects. During the drought of 1976–1977, the Grants witnessed little food production at all, and no finches bred. Obviously, population declined, but survival rates varied, with survival depending on birds’ ability to exploit difficult food resources. Only those who survived could become parents during the next breeding season. Large birds with strong beaks had a better chance of survival than small birds. The small seeds went first, to all sizes of birds with any beak shape; but when the small seeds were depleted, only large birds with wide beaks could exploit the large seeds. When the Grants compared the finches’ average beak depth and size in the years before and after the drought, they saw an increase of 4 percent. Because there was no breeding during the drought year, this change was due to differential mortality. If either wet or dry conditions had continued over many years, beak traits would have changed directionally. Grant suggests that if 4 percent change per year had continued for as little as 46 years, it could have led to more speciation.

The Galapagos Islands were and continue to be a natural outdoor laboratory. One lesson to be learned is the value of variability. Although body size and beak shape were stable during normal rainfall years, beaks and body size changed after a single abnormal season. Because rainfall in the Galapagos is quite variable from year to year, survival of individual finch species depends on variability. That Darwin may have not used finches to formulate his theory of natural selection is really not very important; this lesson is.

post-Darwinian observations on Galapagos Islands finches.) If we look at human evolution, increase in mean brain size over the past 2 myr and development of lactose tolerance in some populations are examples of long-term directional selection (Olson 2002). Although Darwin assumed directional selection to be a slow process, it has been seen to operate in less than 10 years on the wing length of wild North American fruit flies.

The other type of selection is called **stabilizing or balancing selection**. This form of selection stabilizes the existing variability of a trait in a population generation after generation. For example, one familiar species, the house cat, varies in hundreds of traits: size, color and texture of hair, whisker length, and tail length, to name a few. Although house cats vary in size, they are never as large as lions or as small as mice; stabilizing selection limits the size of cats to magnitudes within the range of the species’ existing variability as long as no new mutations occur to change that range. Stabilizing selection would have been very familiar to Darwin, as it is constantly operating on the extremes of any trait’s variability. If every variation of every biological trait of every species that ever existed is considered, the effect of stabilizing selection on species’ variability is staggering.

### An Example of Directional Selection

It is fortuitous when we can observe natural selection operating directly on mutations, because the process normally occurs too slowly for us to see it. Nonetheless, scientists and others have observed and reported on more than 100 cases of natural selection occurring in nature. The peppered moth of Industrial Revolution fame in central England is the classic example of observed and noted directional selection on an initial mutation. Before the Industrial Revolution (particularly around Manchester), people often saw a certain species of moth resting on light-colored lichens on trees. (Lichens are symbiotic fungi and algae.) Because most of the moths also were light colored, they were camouflaged and not easy prey for birds. Roughly 1 percent of the moths were dark colored (a result of an initial mutation or mutations), but birds ate most of these uncamouflaged moths before they had a chance to reproduce. A small number of dark moths continued to appear during the pre–Industrial Revolution period, however, either because occasionally a dark moth avoided predation and reproduced or because mutations reoccurred on the color gene(s). After the Industrial Revolution began, chimney soot killed the lichens and exposed the dark-colored tree bark. Given the principles of natural selection, it is not surprising that the frequency of dark moths increased to 90 percent and the frequency of light moths decreased accordingly. Dark color became the camouflage color, and the light moths, now easily seen by birds on the dark tree bark, were picked off. After England passed the Clean Air Acts in 1956, pollution lessened considerably; the light-colored lichens returned to the trees; and the light-colored moths evolved back to their original high frequency (Grant 1991).
Although the peppered moth is a well known and often cited example of natural selection, it occurred only after the Industrial Revolution, a human-caused event. Studies of melanin (dark pigment) developing in spiders as a result of industrial activity also indicate directional natural selection. A recent case of directional natural selection occurred without human interference, showing natural selection in operation in modern times. The “normal” color for pocket mice in Arizona is light, and, living on sandy surfaces, they are well camouflaged. Some pocket mice have mutated to a dark color, making them well disguised on nearby dark lava rocks (Yoon 2003).

The peppered moth example provides interesting insights. First, it indicates that directional selection works on the biological variability that exists within a species—in the moth, on its colors. It also demonstrates that the vehicle of selection is the relative reproductive differential or survival value of the two forms of moth color under two different environmental conditions. What was good under one set of conditions was bad under another. Finally, better adaptation to existing environments can be noted in this case. (Part of the research for this classic case has come under criticism recently, but it is difficult to get two different colored moths to sit on the same piece of bark and spread their wings at the same time for a photo opportunity!) Mutations and natural selection form the causal core of evolution: Certain small, randomly occurring mutations in sex cells that bestow positive net reproductive differential on individuals are the major reason why ancestral populations change over time to become descendant populations.

**Gene Flow**

Gene flow is of secondary importance in macroevolutionary change, but it is often of primary importance in microevolution. Gene flow is the movement of genes between populations through the sexual reproduction of organisms that do not normally interbreed. It can occur through true migration, when populations move into new areas and interbreed with existing populations, or through the exchange of genes on reproductive borders without significant migration, when interbreeding occurs between the two populations. In populations that inhabit areas of large extent, individuals are more likely to mate with those in close proximity than with individuals living far away. Often, natural geographic barriers such as rivers, mountain chains, or deserts exist between populations. These barriers limit random mating within the species and are one reason why subspecies can exist. But when members of populations interbreed after migration of one population into the breeding area of another or interbreed at their borders, the result is that genes flow from one population to another through offspring. Gene flow has three effects. First, it can spread beneficial mutations from one population to another. Second, it can prevent breeding isolation that would result in speciation by cladogenesis (splitting), a topic we’ll discuss shortly. And third, it reduces the sometimes deleterious effects of inbreeding in small populations.

**Genetic Drift**

Sewall Wright discovered in the 1920s that populations could change over time for a reason other than mutations, natural selection, or gene flow. He discovered that numerical size alone can cause populations to change via genetic drift in a single generation—and that the smaller the population size, the greater the possible effect. A population that numbers more than 10,000 (considered “infinite”) is not susceptible to genetic drift because its large size ensures that each generation will be representative of its predecessor unless mutations, natural selection, or gene flow are causing change. A population that numbers fewer than 1,000 individuals can show up to 1 percent change due to chance alone in any two generations. But a population can show real change if the number is under 100 (up to 10 percent change per gene per generation), and even more if the number is under 10 (up to 60 percent change per
Gene per generation). Obviously, the possible genetic drift effect increases as population size decreases. Another variable effect of gene drift is somewhat controversial, but there is some evidence that gene drift has a greater effect on neutral mutations and can “fix” them quickly (Klein and Takahata 2002).

Genetic drift gets its name from the fact that alleles can “drift” away or be lost. How does genetic drift work? Consider the nontaster gene in human groups. Until 10 kyr ago local human populations likely ranged between 50 and 25 individuals. In such groups up to 20 percent change per gene per generation is statistically possible. The change is totally random, because it is due to chance; so the change can be directional, with traits becoming fixed or totally lost, or gene frequencies can randomly increase and then decrease to end up where they began, or there may be no change at all. Figure 3.8 shows four computer simulations to show the effect of genetic drift on a very small population of 15 individuals; each simulation begins with an allele frequency of 50 percent for the nontaster gene. In one simulation the allele becomes fixd in the population after 10 generations; in another the allele is lost after 17 generations; and in another the frequency ends as it started 25 generations earlier. Statistical probabilities are just that—probabilities—and which path chance will take cannot be predicted. More than 99 percent of human groups have been (and some still are) susceptible to the effect of genetic drift, in which traits can become fixed or lost very quickly. Theoretically a small but extremely beneficial mutation could even cause speciation in relatively few generations (Relethford 2001a), although this idea is controversial.

**Figure 3.8**

**Genetic Drift.** Shown here are four computer simulations of 25 generations for a small (N = 15) human population. Note that each began with 0.5 (50 percent) allele frequency for the nontaster gene. In one simulation the allele became “fixed” in the population, in one it “drifted” or became lost, and in one it ended up at the level where it started.
What Really Evolves?

The population or species evolves, not the individual or any higher taxonomic unit. The terms *species* and *population* are often used interchangeably, but although a species is a population, a population is not necessarily a species. By the definition of evolution, traits also can be said to evolve, as in the case of brain size or bipedalism—but always at the population level. At a micro level alleles evolve as well, because their proportions fluctuate in a population, whether directionally or more randomly, over time. This section considers the species level of evolution. Over a million and a half species have been formally named and described, but it is estimated there may be as many as 50 million species in existence today. Many of the undescribed groups live in the forest canopy where they are difficult to study; most are insects.

The Species

There are certain concepts in the scientific literature that are particularly contentious: Cultural anthropologists argue over how to define culture, political scientists over the definition of democracy, and biologists over the best way to define “species,” with over 22 definitions discovered in the literature as of 1997. The International Code of Zoological Nomenclature is the official body that accepts or denies new taxa, but there is no way to enforce its decisions (Winston 1999). The biological species concept (BSC), formalized by Ernst Mayr in 1942, seems the best definition to use here because it says that *species* define themselves by who mates with whom. The English naturalist John Ray (1627–1705) was the first to recognize that the ability to produce viable offspring was a way to classify animal groups, and Ray began the tradition of assigning species names on that basis (Young 1992). Most biologists define modern species on the basis of reproductive isolation; that is, a species consists of individuals that mate with each other and produce viable offspring but do not mate with members of other species. Reproductive isolation between species can be caused by geographic barriers, seasonal mating differences, lack of appropriate sexual attractants, incompatibility of reproductive organs, the inability of cells to fertilize and form offspring, and/or the inability of offspring to produce functional sex cells. Although the BSC appears to concentrate on the reproductive element, it does not ignore morphological or genetic similarities (Rennie 2002). The BSC is the best definition for sexually reproducing living species.

Real and theoretical concepts of species do not always coincide, however, and hybridization and the ability to recognize species in the fossil record remain problematic. If we observe members of a named species mating with members of another named species on a relatively regular basis, does it mean they are “hybridizing” (mating between species)? Or do our observations merely indicate that we are observing a population through the lens of stasis, when in reality species are always in the process of evolving? When two populations live in close proximity, human observers may give them different species names because they consider their morphological differences sufficient to warrant different species status; but if the populations interbreed and produce viable offspring, by definition, they are but one species. Modern baboons are an example. Numerous morphological differences in size, hair color, and behavior led taxonomists to classify baboons as five different species. But because baboon populations interbreed on their borders and produce viable offspring that often back-cross to the parent population, baboons are but one “superspecies,” *Papio papio*. Similarly, two “species” of macaque monkeys have been observed to interbreed in Indonesia for at least 20 years (Bynum 2002), and humped zebu and the nonhumped nonzebu cattle differ morphologically but are totally interfertile in mixed herds in East Africa.

**biological species concept (BSC)**
A definition of *species* that focuses on reproductive capabilities: Organisms from different populations are in the same species if they can interbreed and produce fertile offspring.

**species**
A group whose members interbreed naturally and produce fertile offspring.
There are cases of true hybridization between species, but humans are almost always involved in the process: Donkeys and zebras do not mate normally but can be made to produce “zebronkeys” for sideshows, lions and tigers can be made to produce “ligers” and “tiglons,” and mules (horse–donkey hybrids) can be created but are apparently always sterile. Although people love stories about hybridization between dogs and cats or between humans and gorillas, such interbreedings do not happen. Interestingly, however, many folk taxonomies match scientific ones quite well. One human group in New Guinea, for example, recognizes several hundred species of vertebrates and only four of them aren’t found on Western scientists’ lists of species (Ridley 2004).

George Gaylord Simpson, an American paleontologist who specialized in establishing the evolution of the horse, added the time dimension to Mayr’s BSC in 1961 by defining species in evolution as “independently evolving lineages.” He called his model the evolving species concept (ESC). Simpson realized that it would always be problematic to identify individual species in evolving lineages and claimed there was no nonarbitrary way to divide up a continuum. Noting these difficulties, some experts have looked to major adaptive changes as clues to speciation and subsequent taxonomic delineation. Hominid bipedalism and primate arboreal life are examples of major adaptive changes. The premise underlying this approach is that such major changes would result in rapid morphological changes that could be noted in the fossil record. This approach does work for major changes but does not help with separating evolving lineages once the major change is fixed (Kimbel and Martin 1993).

The Genus and Subspecies

Unlike the species, neither the genus nor the subspecies evolves. The genus is a taxonomic collection of species that has evolutionary significance only in the probability that all species assigned to a genus once had a common ancestor. Some species contain
**subspecies**, groups whose individuals share certain characteristics and genes in higher frequencies with one another than with the species as a whole. In theory all members of a species should be able to interbreed and produce viable offspring; in actuality, however, sometimes they are unable to. Dogs are a good example. At one time probably all members of *Canis familiaris* could breed successfully, but humans using artificial selection have made large dogs larger and small dogs smaller. The result can be a physical mismatch, such as between a female Chihuahua and a male Saint Bernard. The concept of the subspecies is not at all universal; many species occupy small geographic areas where subgroups do not form.

**Speciation**

When a species either changes over time and forms a new species or splits into two or more species, the process is referred to as **speciation**. In **transformism**, also called **anagenesis**, one species transforms itself as a whole unit into a new species over time. This time relationship between ancestral and descendant species makes them parts of a **chronospecies**. Through accumulated mutations, natural selection, and sufficient and periodic gene flow, the chronospecies becomes different enough in time from its ancestral condition that it is given a new taxonomic name. **Splitting**, also called **cladogenesis**, occurs when an effective isolating barrier, geographic, genetic, or behavioral, keeps members of two (or more) segments of a species from interbreeding. (See Figure 3.9.) Mutations that occur in one population have no way to spread to the other

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**SPECIATION.** One species can speciate into two by splitting, or cladogenesis, as a result of some kind of isolating barrier between segments of the species. This figure depicts a geographic barrier. What would happen to the isolating barrier (the river) if the animals depicted could swim? What other isolating barriers could cause speciation by splitting?
population if gene flow is reduced to zero; and given that mutations are random, identical mutations are not likely to occur in both populations. Also, natural selection is likely to operate somewhat differently on each population, because each occupies a somewhat different environment. With or without genetic drift, the two populations in time will split—will become so different that even if the isolating barrier were removed, interbreeding could not occur.

The length of time it takes for speciation to occur by either transformism or splitting varies from species to species and from time to time. Theoretically, it takes only one important trait difference for a population to form a new species—a group incapable of mating back with the parent population (Wilson 1999). Although no one has ever seen speciation occur in nature, two American geneticists recently sorted two groups of fruit flies by their environmental preferences and bred each group separately for 35 generations. The resulting populations refused to interbreed (Rennie 2002). In nature, because most mutations do not affect reproduction directly, it is common for large land-based species to take half a million years or so to speciate. Predictably, a lineage that has high mutation rates, exists in differing environmental niches, and has numerous geographic barriers is likely to speciate more often and more rapidly than a lineage that has low mutation rates, exists in a homogeneous environment, and has no known geographic barriers.

Paleospecies and the ESC

Most evolution experts use Simpson’s ESC to define species. It is, of course, impossible to define paleospecies, extinct species, by reproduction criteria, because past breeding population boundaries are not known. So scientists use their knowledge of the range of variability in modern species and apply it to closely related past populations to try to identify where one species ends and a new one begins. If the basic question is “How much variation can there be within a past population for it to be considered a single species?” the answer is “the same kind and amount of difference as there is within a modern, closely related species” (Rose and Bown 1993). Many scientists use the 5 to 10 percent variability range as a kind of magic number for hominids. That is, if a group of recently discovered hominid fossils shows 5 percent variability in all comparable traits, it is plausible that there are two species; if that group shows 10 percent variability, it is probable that two species are present (Groves 1997). However, humans today show 12 percent variability in cranial capacity, so the rule is not ironclad (Henneberg 1997). It has been suggested that if a modern taxonomy specialist were to see the bones of all known 300 breeds (subspecies) of dogs, about 20 species (not subspecies or breeds) would be identified (McKee 2000). There continue to be numerous difficulties in using the ESC with paleospecies, yet there is no viable alternative. For example, if chronospecies could not be divided up, even stratomolites dating to 3.5 billion years ago would have to be *Homo sapiens*, because humans came from an unbroken line of life forms extending back to those stromatolites. Other difficulties in identifying paleospecies include small, biased samples and scientists’ inability to know whether a given fossil represents “average” traits or whether it is from the beginning, middle, or end of its species’ time (Foley 1991). Because scientists will always be able to arbitrarily divide up evolving lineages into taxonomic chunks, there will always be disagreements. And, unfortunately, the “observer effect” (or bias) is always present (Eckhardt 2000).

**splitting (cladogenesis)**

A mechanism of speciation in which an effective isolating barrier (geographic, genetic, or behavioral) keeps two (or more) segments of a species from interbreeding long enough for mutations and natural selection to make the separated populations no longer able to interbreed and thus separate species.

**paleospecies**

Species that are extinct, though their descendant species may still exist.
Before exploring the specifics of human evolution or the general lineage leading to humans, it is necessary to understand the principles that underlie evolution in general. Although most evolutionary knowledge is based on experiments with nonhuman animals, it is appropriate to use such knowledge to understand human evolution—because all animals share the basic stuff of life, the same amino acids. Once scientists understood the fundamentals of the three cornerstones of evolution—inheritance, natural selection, and mutations—they began looking for causes and seeking to answer why questions. There is still a lot to learn, however. Chapter 4 will focus on macroevolutionary happenings.

- Humans are animals and can be studied the same way as any other species of animal. Biological anthropologists focus on populations of humans rather than on individuals.
- Evolution is defined as biological change over time. Because change can be observed and measured, evolution is fact, not theory.
- Early ideas about evolution began with Anaximander, a nontraditional sixth-century BC Greek thinker who suggested that humans evolved from fish.
- Until the seventeenth century, mainstream thinking held that life was created in perfect form; thus, there was no need for change or possibility of change.
- By the eighteenth century, travel had begun to enlighten scientists and the lay public. The Lamarckian theory of acquired characteristics was central to early explanations for biological variations and change.
- Scientists in the mid- to late nineteenth century discovered the three cornerstones of evolutionary understanding: Darwin discovered the principles of natural selection, Mendel discovered the principles of inheritance (heredity), and de Vries named mutations as the ultimate source of variation. These understandings formed the basis for the modern synthesis.
- The basic principles of Mendelian inheritance are as follows: Discrete particles are the units of inheritance; after sex cell replication, chromosomes separate, taking their genes with them (segregation); as long as traits are on different chromosomes, they will assort independently (independent assortment); and some alleles show dominance over and mask the effects of other alleles.
- The principles of genetics are the same for all animal species. For each biological trait, there is a phenotype and genotype. For the more than 4,000 simple
ential), gene flow, and genetic drift. Not all mutations are evolutionary, and natural selection eliminates most mutations that occur in sex cells. Gene flow keeps species from splitting and passes new genetic material throughout the species. Traits can be lost or fixed in small populations through genetic drift that is due to chance.

- It is the species, not the genus or subspecies, that evolves; a species is self-defining.
- Speciation can occur (new species can form) when existing populations transform themselves over time into a quite different species or split into two species. Extinct species are called paleospecies.

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**SUGGESTED READING**


Grant, P. *Ecology and Evolution of Darwin’s Finches*. Princeton, NJ: Princeton University Press, 1986. Armed with more than a hundred years of biological research and discoveries since Darwin’s classic visit to the Galapagos Islands, Grant studied Darwin’s finches in great detail, fortuitously being able to take advantage of a severe local drought and its rapid effects on finch beaks. Nicely illustrated, the book puts flesh on Darwin’s more anecdotal study.

Marks, J. *Human Biodiversity: Genes, Race, and History*. New York: Aldine de Gruyter, 1995. A readable book that focuses on human genes and their relationship to modern humans as a population and as subpopulations ("races") and looks at how concepts of genes and “race” have changed over time.