UNDERSTANDING CHANGE
Modern Evolutionary Theory

CHAPTER CONTENTS

Chapter 2 ended with a discussion of scientific creationism and intelligent design. We said that the theory of evolution, unlike creationism and intelligent design, is supported by a massive amount of interrelated evidence gathered and interpreted according to the scientific method.

What is the evidence for evolution as we understand it today? How does evolution work? How do the processes of evolution apply to the human species?

Genetics

As we noted in Chapter 2, neither Charles Darwin nor Alfred Wallace understood the biological variation that was so crucial to their theory of natural selection. They knew that variation existed and was maintained even after a species had undergone years of natural selection, but as Darwin admitted, such variations "seem to us in our ignorance to arise spontaneously" (1898:239). Nor did Darwin understand how traits were passed on from parent to offspring—another important factor in natural selection.

Why did Darwin fail to understand these principles? The answer is that he was operating without knowledge of **genetics**. Adhering to the notion current in his day, he thought inheritance worked through some sort of "blending"—a mixing of parental substances in the offspring. There seemed to be ample evidence for this idea from plant and animal breeding, where offspring often exhibited traits that appeared to be 50:50 mixtures of their parents' traits—pink flowers from a cross of red-flowered and white-flowered parents, for instance.

Plenty of characteristics, of course, don't show this equal mixture of parental traits. Organisms inherit their sex from their parents, and, with few exceptions, they are not 50:50 blends but either male or female. Traits that seem to blend, though, like flower color, appear to have been more influential in early thinking about the mechanism of inheritance.

Ironically, at about the same time Darwin was writing *The Origin of Species*, an Augustinian monk named Gregor Mendel (1822–1884), working in a monastery in what is now the Czech Republic, established that inheritance did not operate by blending but rather was **particulate**. By conducting breeding experiments with the pea plants in the monastery garden (the culmination of many years of experimentation with plants and mice), Mendel showed that an organism's traits are passed from generation to generation by individual particles, which Mendel called *factors*—what we now call **genes**.

As a further irony, Mendel's work was neither widely read nor fully appreciated by those who did know about it. They failed to see any implications beyond some interesting facts about pea plants. After Mendel's death, his work fell into obscurity, and it was not until 1900 that it was rediscovered. By then the implications of his experiments were clear, and so the stage was set for the series of discoveries that led to our modern understanding of genetics.

genetics The study of the mechanism of inheritance.

particulate The idea that biological traits are controlled by individual factors rather than by a single hereditary agent.

gene The portion of the DNA molecule that codes for a specific protein.

protein The family of molecules that makes cells and carries out cellular functions.

enzyme A protein that controls chemical processes.

The Genetic Code

We now understand that a gene is actually a set of chemical instructions for the production of a **protein**. Proteins serve many functions in a living thing. Some are structural, shaping the cells (Figure 4.1); supporting the

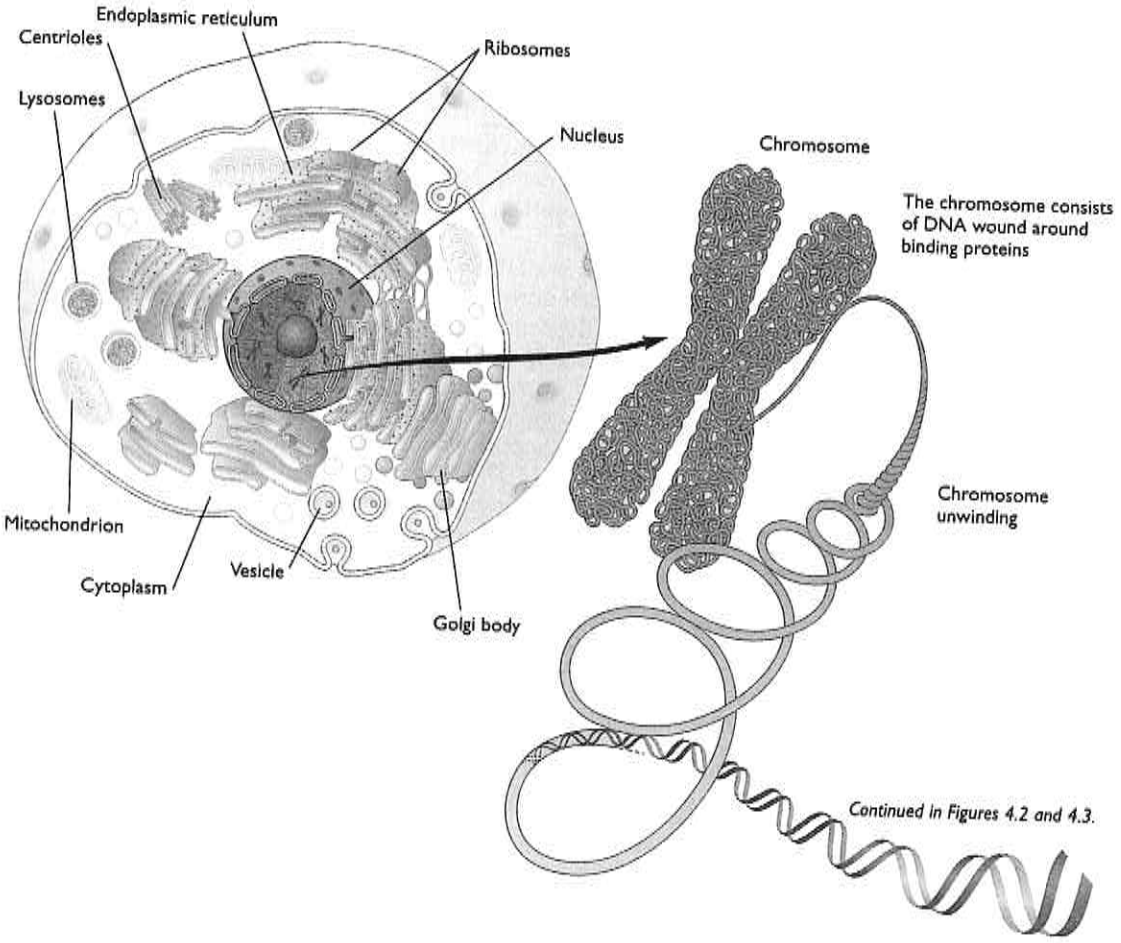


FIGURE 4.1 A typical cell and its important parts. This cell represents all types of cells, from the more complex single-celled organisms such as amoebas to the cells in the human body. The ribosomes are the sites of protein synthesis (see Figure 4.3). The mitochondria are the cells' energy factories, converting energy stored in nutrients into a form the cells can use to perform their various functions.

cells' internal structure; linking the cells together; and building membranes, muscles, and connective tissue. Collagen, a key constituent of skin, tendons, ligaments, and cartilage, is an example of the latter. Some proteins are **enzymes**, catalysts that speed up the body's chemical reactions. Still others transport chemicals through the body; hemoglobin, which we will discuss further below, is a protein that carries oxygen to the cells. A few proteins are hormones, chemicals that send messages among cells. Thus, it could be said that, in a sense, living things *are* proteins. About half an animal's dry body weight is protein. Humans can produce at least 100,000 different proteins.

The genetic code is made up of a variable sequence of bases (a family of chemicals) that are part of a long chemical strand called **deoxyribonucleic acid (DNA).** The strands of DNA are themselves wrapped around series of protein cores to make up **chromosomes,** found in the nuclei of all cells. Four bases are involved in the code: adenine (A), thymine (T), cytosine (C), and guanine (G). Chemical bonds between these bases hold the DNA molecule together, but the bases are bonded only in A-T or T-A and C-G or G-C pairs.

The first function of this pairing is to enable the DNA molecule to make copies of itself during cell division. The DNA molecule is shaped like a ladder with the ends twisted in opposite directions, a configuration called a double helix. During cell division the helix unwinds, and each strand, with its now unpaired bases, picks up the proper complementary bases, which are in solution in the cell. This is called **replication.** Thus, when the whole cell divides, each new daughter cell has a complete set of DNA base pairs (Figure 4.2).

deoxyribonucleic acid (DNA) The molecule that carries the genetic code.

chromosome A strand of DNA in the nucleus of cells.

replication The copying of the genetic code during the process of cell division.

protein synthesis The process by which the genetic code puts together proteins in the cell.

messenger ribonucleic acid (mRNA) The molecule that carries the genetic code out of the nucleus for translation into proteins.

codon A section of DNA that codes for a particular amino acid.

transfer RNA (tRNA) RNA that lines up amino acids along mRNA to make proteins.

amino acid The chief component of proteins.

A gene can be thought of as a portion of the DNA molecule that carries a code that instructs the cell to manufacture a particular protein. Our current understanding of how proteins are manufactured according to the genetic code is a process known as **protein synthesis** (Figure 4.3). Follow along in the diagram as you read the description.

During protein synthesis only a portion of the DNA molecule is unwound (in contrast to the complete unwinding seen in replication). **Messenger ribonucleic acid (mRNA)** is assembled against one strand of this unwound DNA. (Only one strand carries the code; the other is structural.) The mRNA transcribes the gene by matching complementary bases to those exposed in the coding strand of DNA, except that uracil (U) replaces thymine (T). We refer to each consecutive sequence of three DNA bases as a **codon.** Think of them as three-letter words. A gene, then, is a sequence of codons, a sentence made up of many words.

After mRNa has transcribed the code, it leaves the nucleus of the cell and moves to specialized structures in the cell called ribosomes where the message is decoded and translated into an actual protein. Another type of RNA called **transfer RNA (tRNA)** reads the three-letter codes as instructions for assembling a chain of **amino acids.** Each set of three exposed RNA bases codes for one amino acid. Thus, for example, a sequence of 300 bases codes for a sequence of 100 amino acids. A sequence of amino acids is a protein. Although there are only about 20 or so types of amino acids, it is possible to arrange them in a nearly infinite variety of sequences and lengths. In this way, the sequence of DNA bases represents a sequence of amino acids, and it is this sequence that determines the shape and function of the protein.

The measurable, observable chemical or physical traits of an organism are the results of the actions of proteins that have been manufactured by the cells according to genetic instructions. Some traits are coded for by

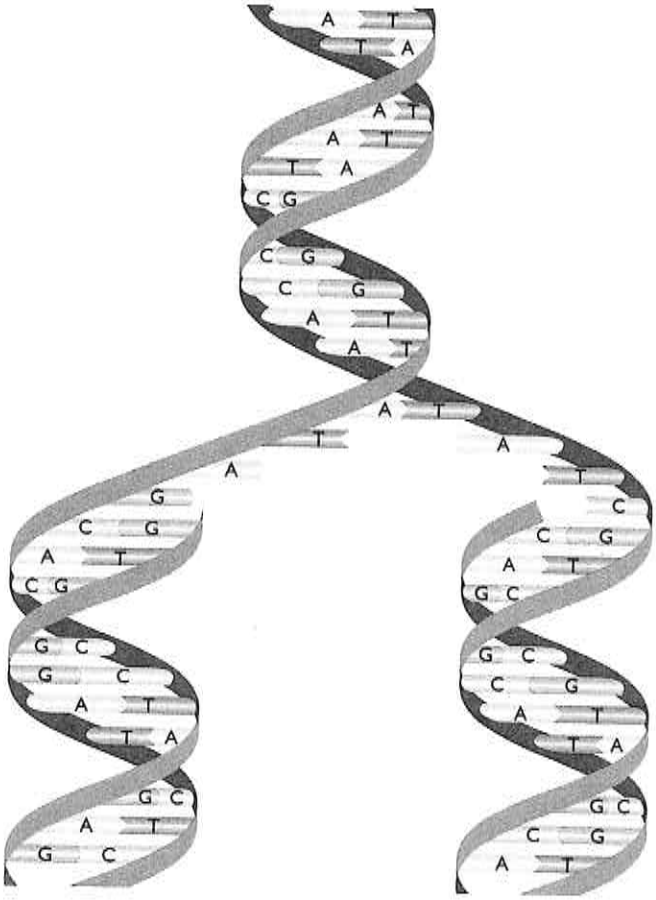


FIGURE 4.2 The DNA molecule. This molecule is shown in the process of unwinding and copying itself prior to cell division.

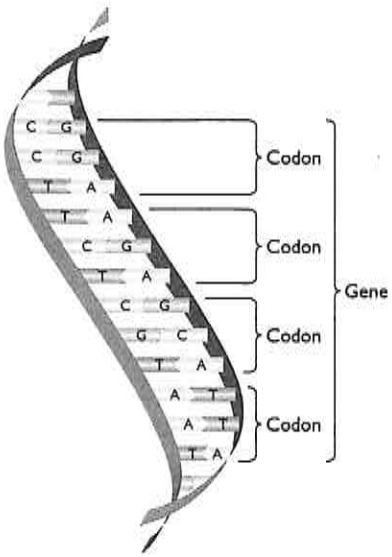
one or just a few genes. The blood component hemoglobin is a protein made up of two paired amino acid chains and thus is determined by two genes. Skin color is made up of many proteins and is thus coded for by many genes.

Each species has a characteristic number of chromosomes. Bacteria have a single chromosome. Humans have forty-six; chimpanzees, forty-eight; wheat, forty-two; and dogs, seventy-eight. In sexually reproducing species, chromosomes come in pairs. An organism inherits one chromosome of each pair from each parent and thus gets half its genetic material from each parent. It follows that the genes also come in pairs.

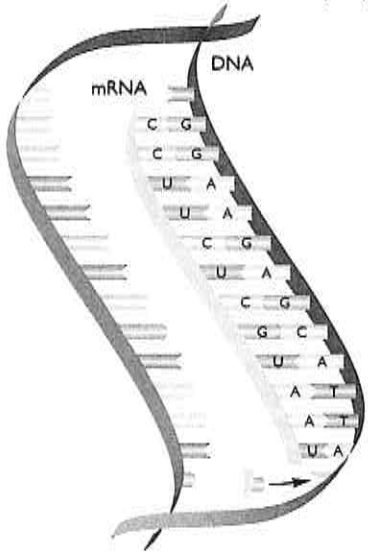
The catch is that a gene can have variants, called **alleles**, each with a slightly different set of codons. The alleles of a gene influence the same trait but may produce different expressions of that trait. For example, for

allele A variant of a gene.

1. Section of DNA molecule with base pairs



2. DNA molecule temporarily separates at bases. mRNA lines up its bases (with U replacing T) with their complements on the coding side of the DNA. (The other strand is structural and is not copied.)



3. mRNA moves out of cell nucleus to ribosomes. As ribosomes move along mRNA, tRNA picks up amino acids and lines up along mRNA according to base complements. Each tRNA transfers its amino acid to the next active tRNA as it leaves, resulting in a chain of amino acids.

4. This chain of amino acids forms a protein.

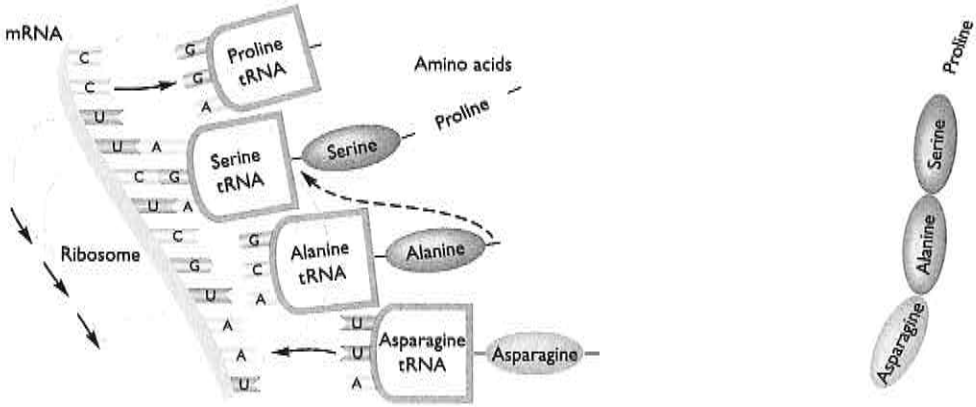


FIGURE 4.3 Protein synthesis as described in the text. In reality, no protein is only four amino acids long, but the process works exactly as shown.

blood type in the ABO system, there are three possible alleles, A, B, and O. Whether you have blood type A, B, AB, or O depends on which pair of alleles codes for the amino acid chain determining blood type. Each allele codes for a version of the chain with a different specific sequence of amino acids. Your allele pair is your **genotype.**

An Overview of the Human Genome

As of spring 2003, nearly the entire human **genome** had been sequenced. What this means is that we know the sequence of the 3.1 million base pairs (the As, Ts, Gs, and Cs) of two representative humans' DNA. (There were two organizations working on the genome sequence, each primarily focusing on a different participant's DNA.) We now have a baseline from which scientists will be able to further research the genome and compare other people and populations.

There is still much to learn. We need to figure out just where in that 3.1-billion-base-pair sequence the genes are, what proteins they code for, what those proteins do, and what functions the other nongene DNA serves. In other words, we still need to discover how the DNA "builds" an organism. But in the last few years, some remarkable new information has come to light.

It turns out that most of the genome is not composed of genes. That is, most of the genome—possibly 98 percent—doesn't code for proteins. We refer to this nongene material as *noncoding DNA*. Although this DNA used to be referred to as "junk DNA," we now know that much of it is not *without* function. Some of it acts as punctuation, marking the beginnings and ends of coding sequences. Some serves to regulate gene function and activity level. Some jumps around carrying other DNA with it, allowing the genetic code to reshuffle its elements; this provides a partial explanation for why a surprisingly small number of genes (20,000 to 40,000 by current estimates) can produce such a huge variety of proteins (around 50,000) in an organism as complex as a human being. Some noncoding DNA is made up of repetitive sequences, some hundreds of thousands of base pairs long, that may do nothing. Some of our DNA may be very ancient, from a remote common ancestor, and some may have been transferred from microbes.

Moreover, we have learned that the coding sequences are not lined up neatly together but are scattered and interrupted by noncoding sequences. A single coding sequence might code for more than one protein, depending on just which part is transcribed.

And recently it was discovered that RNA is more than just the means of converting the DNA code into proteins. Some classes of RNAs have other functions, such as turning on or off some genes, blocking the action of mRNA in producing a protein (which may be important in disease research), shutting down genes and thus operating as a defense against

genotype The alleles possessed by an organism.

genome The total genetic endowment of an organism.

harmful DNA or viruses, and even shaping the genome itself by keeping and discarding certain genes. Realizing that at least some of these noncoding RNAs are produced by the DNA genome has expanded the definition of a gene. Some genes (that is, coding DNA sequences) produce proteins as their end products, but others have noncoding RNAs as end products. By this definition, then, the estimated number of genes in the genome would increase.

Thus, the nice neat view we've had of the genetic code and how it works, even until recently, has radically changed. In the words of Lewis Carroll, the nature and operation of the human genome keeps getting "curiouser and curiouser" and will be an important area of study for years to come.

The Inheritance of Characteristics

An organism has two of the same allele—i.e., is **homozygous**—when both parents have contributed the same allele of that gene. An organism has two different alleles—is **heterozygous**—when the genes contributed by the parents carry different codes. Alleles are products of **mutations**, genetic mistakes that alter the code in a cell of reproduction and thus may transform an existing allele, say, one coding for brown eyes, into a new and different allele, say, one coding for blue eyes.

homozygous Having two of the same allele.

heterozygous Having two different alleles in a pair.

mutation A change in an organism's genetic material.

The expression of a genotype—the trait that results from the genetic code—is called the **phenotype.** In homozygotes both alleles are the same, so the way the trait is expressed is simply in accordance with these alleles. There is no alternative. In heterozygotes, the situation is more complex. In many, the influence of both alleles is expressed in the phenotype. This is what gives rise to the appearance of blending. But on occasion, the expression of one allele in heterozygotes may be hidden. Such alleles are said to be **recessive.** The other member of the pair, the one expressed, is **dominant.** The words dominant and recessive carry no implications of value, no significance for adaptation. Dominant alleles are not necessarily better or more common than recessive alleles.

phenotype The chemical or physical results of the genetic code.

recessive An allele of a pair that is not expressed.

dominant An allele of a pair that is expressed.

When an organism reproduces, it obviously cannot pass on both alleles of each pair to its offspring. If this were the case, the offspring would end up with twice the normal number of genes. Instead, organisms produce reproductive cells that are different from the cells that make up the rest of the organism. These are the sex cells, or **gametes** (sperm and egg, for instance). Gametes are produced through the process of meiosis, which splits the chromosome pairs—and thus the allele pairs—so that each gamete only has one of each gene (Figure 4.4). Mendel called this effect **segregation.**

gamete The cell of reproduction.

segregation The breaking up of allele pairs during gamete production.

When a sperm from the male parent fertilizes an egg from the female, the resultant **zygote** once again has pairs of chromosomes and thus pairs of each gene. But because the members of each pair have two different sources, the combination of genes in each pair will necessarily be different from that of either parent. This produces genetic variation among individuals of the same species and, in fact, among offspring of the same parent.

zygote A fertilized egg before cell division begins.

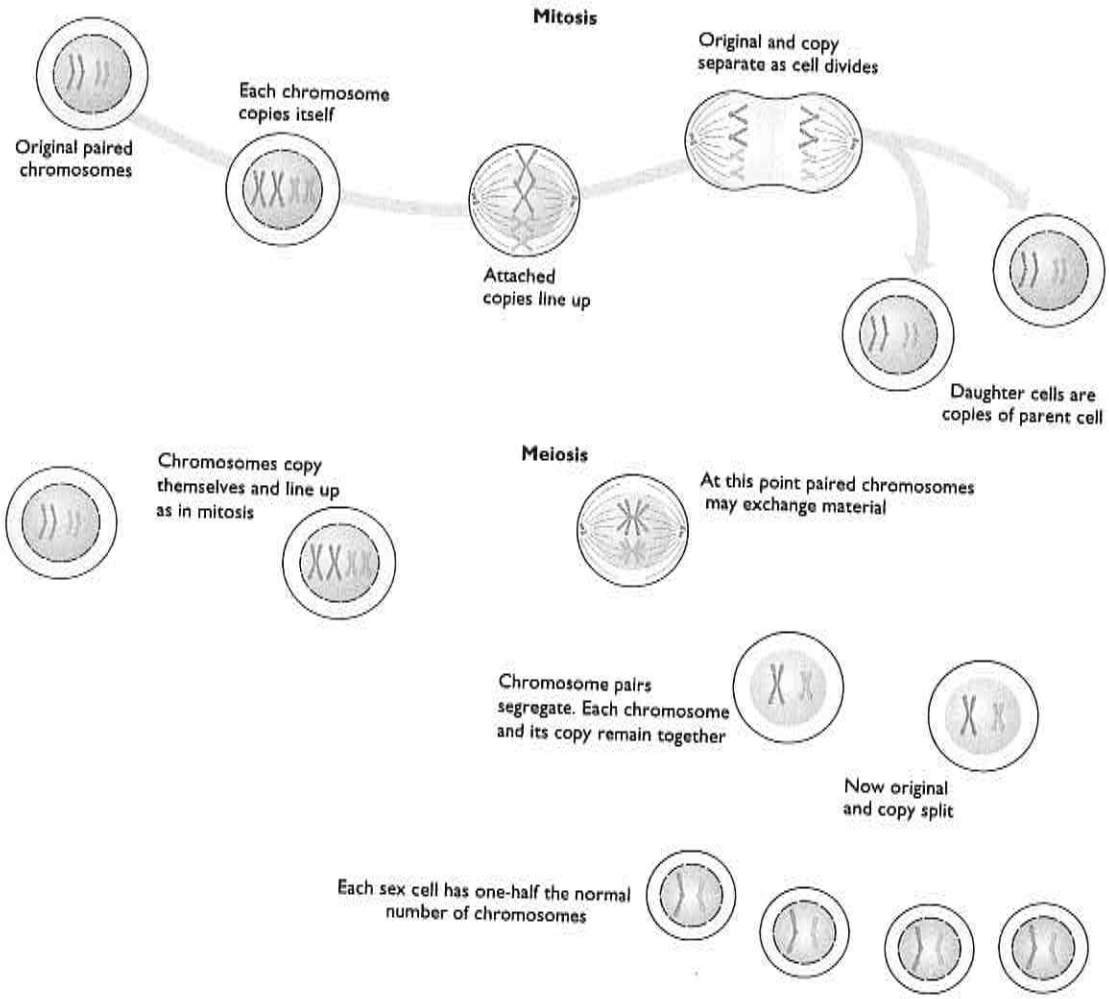


FIGURE 4.4 Cell division occurs in two ways. Mitosis produces exact copies of the parent cell and is the most common form of cell division. Meiosis results in four daughter cells, each with one-half the genetic content of the parent cell. Meiosis is the process by which gametes, or sex cells (sperm and egg), are manufactured. It ensures that when fertilization occurs, the new individual has a complete set of genes, one-half from each parent.

These principles can be demonstrated in a concrete example. Sickle cell anemia is a genetic disease of the blood often associated, erroneously, only with African Americans. The association results from the high frequencies of the disease found in a band across central Africa, a region from which most American blacks trace their ancestry. But sickle cell anemia is also found in southern Europe, the Middle East, India, and Southeast Asia.

Sickle cell anemia, the result of a mutation of a gene on chromosome 11, affects hemoglobin, the protein on the red blood cells that carries oxygen from the lungs to the body's tissues. Hemoglobin is made up of two paired amino acid chains, an alpha chain of 141 amino acids and a beta chain 146 amino acids long. If, through a mutation, the amino acid

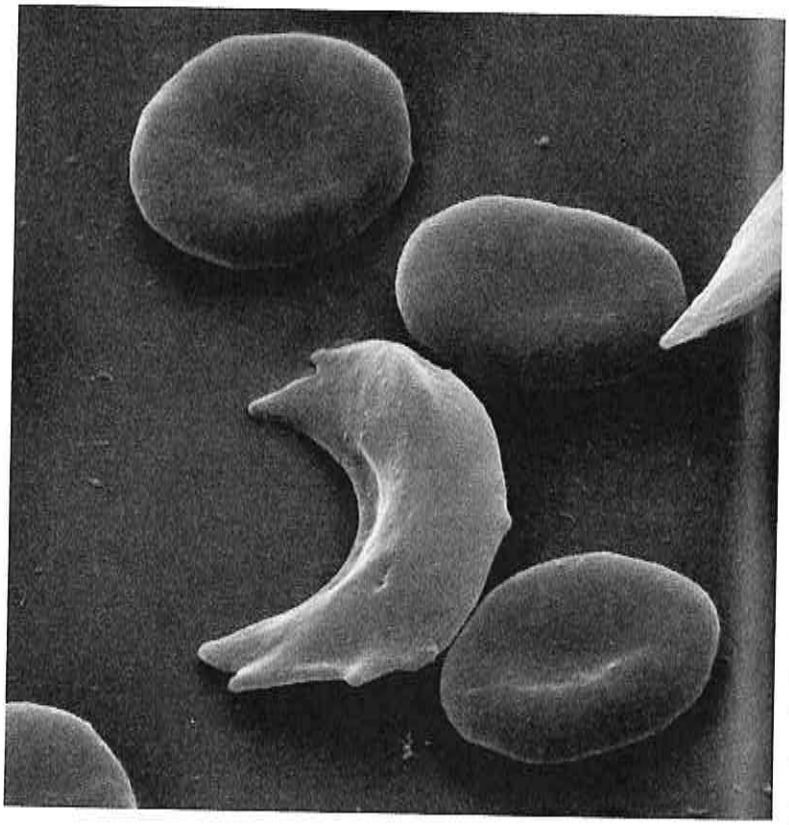


FIGURE 4.5 Normal red blood cells and one showing the abnormal shape characteristic of sickle cell anemia, which result from the presence of hemoglobin with one incorrect amino acid. Such cells fail to transport oxygen properly to the body's tissues. *(© Meckes/Ottawa/Photo Researchers, Inc.)*

valine is substituted for glutamic acid at position 6 on the beta chain, the disease results. In terms of the genetic code, this means that the mutation is one wrong "word"—a mistake in one codon, often a single incorrect base—in a sentence of 287 words. This is a **point mutation.**

When the abnormal hemoglobin is present and stress, high altitude, or illness lowers an individual's oxygen supply, the red blood cells take on peculiar shapes. Some resemble sickles (Figure 4.5). In this condition, they cannot carry sufficient oxygen to nourish the body's cells. Results can include fatigue, retarded physical development in children, miscarriage in pregnant women, fever, severe pain, and increased susceptibility to infection. People with sickle cell anemia frequently die before their twenties; even if they live longer, they have a very low reproductive rate. In terms of evolutionary success, sickle cell anemia has been considered nearly 100 percent lethal. (It should be noted that there have been recent breakthroughs in the treatment of the disease, including a new means of repairing the RNA message with the result that normal hemoglobin is produced.)

point mutation A mutation of a single letter in a codon.

The abnormal allele for sickle cell acts like a recessive in the sense that a person must have the homozygous abnormal genotype to be afflicted with the disease. Heterozygotes, however, still carry the sickle cell allele. When two individuals carrying one allele each for sickle cell mate, they have a one-quarter chance of producing an offspring homozygous for sickle cell anemia. A device called a Punnett square shows how this process works (Figure 4.6).

Sickle cell anemia also demonstrates some of the complexity of genetics and its intricate relationship to the environment and to evolution. The sickle cell allele is not completely recessive. Heterozygotes possess about 40 percent abnormal hemoglobin, and, under extreme conditions of low oxygen, they experience sickle cell symptoms, although not as severely as homozygotes. Indeed, complete dominance and complete recessiveness are the exception; the different alleles of most genes are both expressed to some degree. A heterozygote may be somehow intermediate between either homozygote or may show the phenotypes of both alleles. Alleles that exhibit these characteristics are said to be **codominant**.

The general rule is that the phenotype is usually not a clear-cut indication of the genotype. Besides various relationships among alleles, most phenotypic traits are simply not coded for by a single gene **(monogenic)** but by many genes **(polygenic)**. This effect becomes clear in complex traits like stature or skin color, where numerous individual cellular and chemical actions operate together to make up those phenotypes.

Moreover, not all genes are always in operation. Some are switched on by others in response to environmental changes or some internal timing. In other words, not all genes are equally influential in producing phenotypic traits. In addition, many genes can influence several seemingly unrelated traits. Think of the many symptoms of sickle cell anemia—highly variable specific expressions all resulting from a point mutation.

Finally, the relationship between genotype and phenotype can be influenced by environmental factors, that is, any factor outside the codon-to-trait process just outlined. Your skin color, for example, though coded for in your DNA, can change noticeably depending on your health, how much sun you get, and even your emotional state. All these factors can be considered environmental in the broadest sense of the word. Heterozygotes for sickle cell anemia, who all have the same genotype, nonetheless vary greatly in the degree to which they exhibit the symptoms of the disease and in how easily those symptoms are triggered. This variation occurs because of a complex interaction of numerous factors that affects the relationship between genotype and phenotype.

With the rediscovery of Mendel's work in 1900, an understanding of the mechanism of inheritance and of the basic source of variation was added to Darwin's framework for a theory of evolution by natural selection. From this synthesis, over the next century, our current knowledge both of genetics and of the processes of evolution has developed.

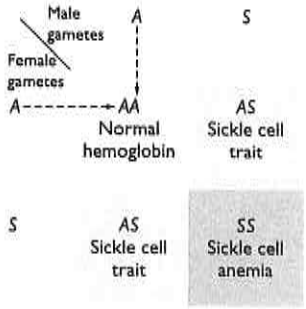


FIGURE 4.6 Punnett square showing sickle cell inheritance. (*A* is the normal allele; S is the sickle cell allele.) Two individuals, each heterozygous at the hemoglobin locus, produce gametes with normal and abnormal alleles in about equal numbers. When they mate, they have a one-quarter chance of producing a child with normal hemoglobin, a one-half chance of producing a heterozygote like themselves, and a one-quarter chance of producing a child who will exhibit the symptoms of sickle cell anemia.

codominant The expression of both alleles of a gene pair.

monogenic A trait coded for by a single gene.

polygenic A trait coded for by more than one gene.

The Genetics of Populations

The physical evidence for evolution is change in the phenotypic features of organisms over time. If evolution manifests itself as phenotypic change and if the genetic code is responsible for generating phenotypic traits, then evolution may be accurately regarded as genetic change over time. Because the overall genetic makeup of an individual does not change (except for isolated mutations), individuals don't evolve. The unit of evolution is the **population**, defined generally as a group within which mates are normally found.

Technically, a whole species could be treated as a genetic population because members of an animal species, by definition, can only reproduce within that species. (Some plants *can* reproduce across species.) But most species are unevenly distributed within their range and so contain subunits of interbreeding individuals, often further defined by a particular locality and perhaps even by particular adaptations and physical characteristics. These groups are called **breeding populations**, or **demes**. The evolution of a species can be seen as the collective evolution of the demes within that species as those populations change independently and as they interact by exchanging genetic material.

A breeding population is characterized genetically by identifying how often a certain allele appears in the population relative to the other alleles of the same gene. This is called **allele frequency**. Thus, the genetic definition of evolution is change in allele frequency over time. The processes of evolution, then, are all those factors that bring about changes in allele frequency.

To understand how this concept can help us study and explain the nature and operation of evolutionary processes, let's use sickle cell anemia in a hypothetical breeding population (Park 1999 from Relethford 1997). In a population of 145 individuals under study, the following numbers were found:

population A reproductive unit.

breeding population A population with some degree of genetic isolation from other populations of the species.

deme Generally, the same as a breeding population.

allele frequency The number of times (in percentage) that a particular allele appears in a population.

Phenotype	Genotype	Number
Normal	*AA*	35
Heterozygote	*AS*	100
Sickle cell	*SS*	10
Total		145

To study the population, it is necessary first to find the allele frequency. Because individuals with normal hemoglobin have genotype *AA*, there are twice as many *A* alleles as there are "normal" individuals. In addition, all heterozygotes possess one *A* allele, so this must be added to the total *A* allele count. Thus,

$$\text{Number of } A \text{ alleles} = (35 \times 2) + 100 = 170$$

Similarly, for the *S* allele,

$$\text{Number of } S \text{ alleles} = (10 \times 2) + 100 = 120$$
$$\text{Total number of alleles} = 290$$

Now, in order to calculate the frequency (the percentage) of occurrence of each allele, divide the number of each allele by the total number of alleles in the population. Thus, for the *A* allele,

$$170/290 = 0.59$$

Similarly, for the *S* allele,

$$120/290 = 0.41$$

In a population of 145 individuals, the allele for normal hemoglobin occurs 59 percent of the time, and the allele for sickle cell occurs 41 percent of the time. These are the allele frequencies for that population.

Given those allele frequencies, we can now calculate the expected genotype and phenotype frequencies under conditions where no evolution is in operation. This procedure is known as *testing the null hypothesis.* If you can state the condition under which nothing occurs, you can then compare it to situations where something *does* occur, observe the nature and direction of the difference, and possibly discern factors that are responsible. With reference to the genetics of populations, the null hypothesis is specifically known as the **Hardy-Weinberg equilibrium** (named after the mathematician and physician who independently derived it in 1908).

Using our two alleles, *A* and *S*, we designate the frequency of *A* as p and the frequency of *S* as q. (These letters are used because of a mathematical convention.) The probability of creating each of the possible genotypes is the product (the result of multiplication) of the frequencies of the alleles of that genotype. Thus,

Genotype	Product of Frequencies
AA	$p \times p = p^2$
AS	$p \times q = pq$ } $2pq$
SA	$q \times p = qp$ }
SS	$q \times q = q^2$

Because all genotypes are now accounted for,

$$p^2 + 2pq + q^2 = 1$$

(that is, 100 percent of the genotypes).

We can now return to our hypothetical population to see what its genotype frequencies would be if they were based solely on the frequencies of the alleles, if, in the terminology of population genetics, the population were in Hardy-Weinberg equilibrium.

Hardy-Weinberg equilibrium The formula that shows genotype percentages under hypothetical conditions of no evolutionary change.

Genotype	Expected Frequency	Expected Number	Observed Number
AA	$p^2 = 0.59^2 = 0.3481$	$0.3481 \times 145 = 50$	35
AS	$2pq = 0.59 \times 0.41 \times 2 = 0.4838$	$0.4838 \times 145 = 70$	100
SS	$q^2 = 0.41^2 = 0.1681$	$0.1681 \times 145 = 24$	10

The observed numbers are not in equilibrium. The allele frequencies have changed relative to the null hypothesis situation. Evolution, by definition, is taking place; there are fewer "normal" individuals than expected, more heterozygotes, and fewer sickle cell homozygotes. An evolutionary trend seems to favor heterozygotes. Given what we know about sickle cell anemia, this makes perfect sense. (We'll see why in the next section.) Indeed, data similar to this hinted at the nature of the disease and led to our understanding of it.

In real life, we would still have to run certain statistical tests on the above results because even if the expected and observed numbers do not match, they could still result from simple chance. One such test, called *chi-square*, showed that the results are probably not a matter of chance. They are, in mathematical terms, statistically significant.

What processes, then, can bring about changes in allele frequency in populations and thus alter the phenotypic nature of the group?

The Processes of Evolution

Natural Selection

As Darwin explained, from the physical and behavioral variation within a species, nature selects the characteristics best adapted to a particular environment. The measure of nature's selection is the relative reproductive success of the individual organisms that possess those characteristics. This is called **differential reproduction.** Individuals with the most adaptive traits tend to produce more offspring on the average, thus relatively more often passing on the alleles that code for their advantageous traits. In this manner, the better adapted traits accumulate over time and the poorly adapted ones become less frequent—even disappearing if their possessors fail to reproduce at all. The result is that the species as a whole stays adapted to its environmental **niche**—the particular set of environmental circumstances with which it comes in contact and to which it must adjust.

differential reproduction The differing reproductive success of individuals within a population.

niche The environment of an organism and its adaptive response to that environment.

The traits that make an individual better adapted will, of course, vary with the species in question and with that species' particular niche. Bigger size, smaller size, bright colors, dull colors, speed, stealth, intelligence, reliance on built-in instincts—each can be adaptive depending on the species and niche.

In addition, selection of mating partners takes place in many species. Males may directly compete with one another for access to females. The famous head-clashing duels of bighorn sheep are an example. In other cases, females choose mates based on things such as the establishment of a nesting site or colorful feather displays, as with the peacock (although the adaptive benefit of these displays is still not fully understood). This form of selection is called **sexual selection**, and as we'll see it may have played a part in early human evolution.

Thus, by ensuring that the more advantageous traits are passed to more offspring, natural selection maintains a species' adaptation to its environment. As we have seen, however, environments change. When this happens, traits that were once adaptively neutral or even poorly adapted may actually become better adapted. These traits will begin to occur in higher frequencies because their possessors become increasingly successful reproductively. At the same time, traits that once were adaptive may become increasingly rare and perhaps even nonexistent. Even under new environmental conditions, then, the species can remain viable and adapted, though its adaptive features may be different.

A striking example of natural selection in changing environments comes from pioneering work by Rosemary and Peter Grant and their colleagues (Grant and Grant 2000, 2002; Weiner 1994) among the famous birds of the Galápagos Islands collectively known as Darwin's finches (Figure 4.7). Among the important adaptive features of this group of birds are their beaks, which have evolved to help each species of finch exploit particular food sources. In 1977 there was a severe and nearly yearlong drought on one of the small islands that the Grants' team was using as a study area. Insects virtually disappeared, and the only plant seeds available were larger than average and had tougher than average exteriors to preserve their moisture. The finches on the island suffered a serious food shortage.

The next year, when the rains returned, the researchers found that just one finch in seven (a mere 14 percent) had made it through the drought. Moreover, the surviving birds of one species common to the island (the medium ground finch) were 5 to 6 percent larger than those that had perished and had beaks that were slightly (in fact, less than a millimeter) longer and deeper than the average before the drought. It's not a big difference on a human scale, but the size difference allowed some of the finches to more easily crack open the larger, tougher seeds during the drought, enabling them to survive; males survived much better than females because they are about 5 percent larger.

Now, however, because evolution takes place across generations, it had to be seen if this change would be passed on to the offspring of the surviving finches. This was, indeed, the case. It is the female finches that select males with which to mate. The males that the few surviving females selected were the largest and had the deepest beaks. As a result, the finches

sexual selection The active, rather than random, selection of mating partners by individuals within a population.

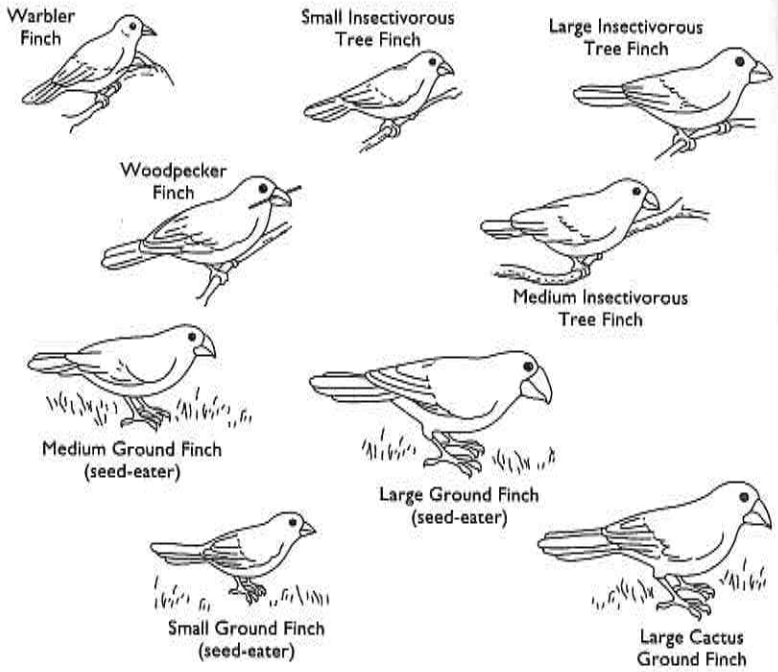


FIGURE 4.7 Some of the species of Darwin's finches found on the Galápagos Islands with indications of their basic adaptations for acquiring food.

of the next generation were both larger and had beaks that were 4 to 5 percent deeper than the average before the drought. Moreover, when conditions, and thus food sources, returned to normal for a time, the average beak size decreased over several generations toward its previous dimensions. Larger beaks were no longer a distinct advantage and so were no longer selected for. These changes showed natural selection in action. Because of the severity of the situation, it took place rapidly enough for human observers to measure and record it.

It is important to note that in this case, as in any example of natural selection, the variation that proved useful under changed circumstances was already present. It did not appear when it was needed or because it was needed. The finches that survived *already* had larger body and beak size; they did not develop these after the drought altered their food source. It was, in other words, already an aspect of their variation, although only a small number of finches would possess large beaks and bodies since, under usual conditions, they conferred no distinct advantage and may even have been disadvantageous. This is the essential difference between Lamarck's inheritance of acquired characteristics and Darwin's natural selection (Figure 4.8).

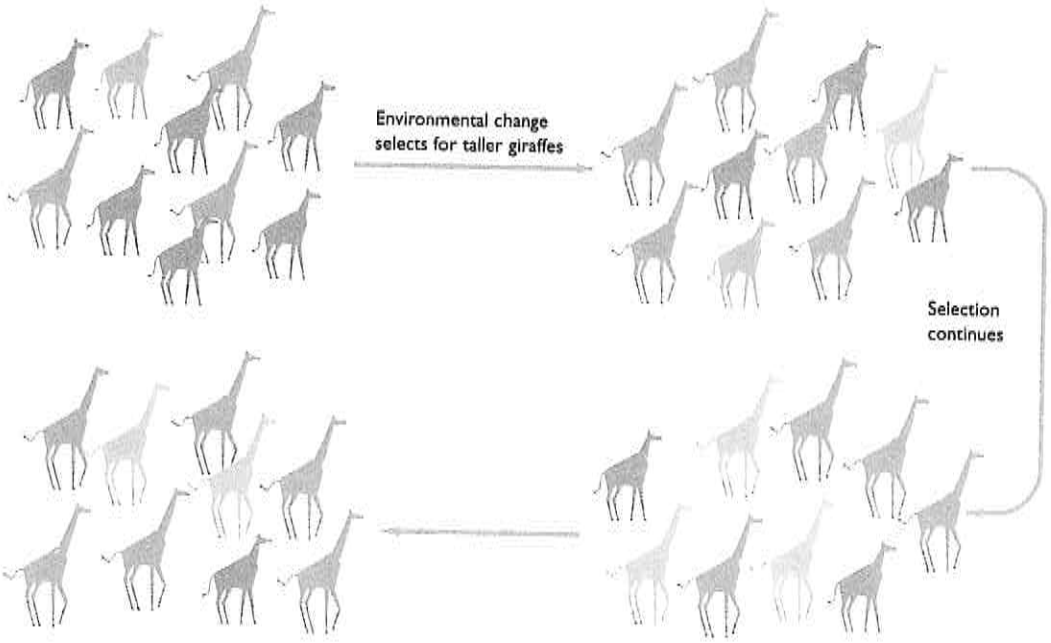


FIGURE 4.8 Schematic diagram of Darwinian natural selection. As in the case of the finches, an environmental change makes the larger individuals more reproductively successful, and they become the most common representatives of their species. Compare with Figure 2.6, Lamarck's concept of evolutionary change.

A more complex example comes once again from sickle cell anemia. Despite the fact that it is so disadvantageous, sickle cell anemia is found in high frequencies in certain areas of the world (Figure 4.9). One would expect a lethal allele to disappear quickly or, in evolutionary terms, to be *selected out*. But in some parts of Africa, the sickle cell disease is found in at least one in every sixty-four people. There's clearly more to the persistence of this disease than first meets the eye.

The reason for the high frequency of sickle cell disease is that the heterozygous condition conveys an adaptive benefit. Besides not usually having severe symptoms of sickle cell anemia, heterozygote individuals also have a resistance to malaria, a potentially fatal disease caused by a parasitic single-celled organism and transmitted by mosquitoes. This resistance comes about because red blood cells with abnormal hemoglobin (remember, heterozygotes have 40 percent abnormal hemoglobin) take on abnormal shapes when infected by the malaria parasite and die, failing to transport the parasite through the system. Sickle cell is found in highest frequencies where malaria is found in highest frequencies (Figure 4.10). In no environment is there any advantage in being homozygous for the abnormal allele. In malarial environments, however, heterozygotes do have an advantage. As you saw in the Punnett square, when two heterozygotes

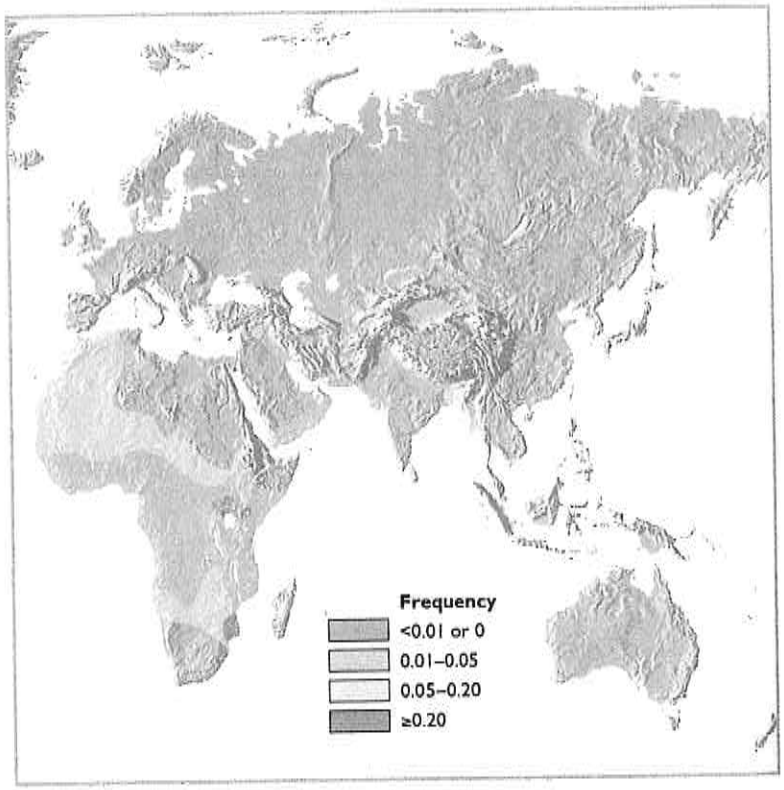


FIGURE 4.9 The distribution of high frequencies of sickle cell anemia. Compare this with the map of high frequencies of malaria (Figure 4.10).

mate, they have only a one-quarter chance of producing a homozygous child who will probably die at an early age from sickle cell whereas they have a one-half chance (twice as good) of producing more heterozygotes who carry the defense against malaria.

Adaptive fitness, then, is relative to particular environmental conditions. What is adaptive in one environment may not be adaptive in another. What is adaptive at one time may not be at another. A lethal allele (such as sickle cell) may actually be adaptive in certain genotypic combinations within certain environments. As we will see later, a genetically determined ability to, say, walk on two legs more frequently and with greater ability might provide an advantage to those members of a species that possess those characteristics and abilities, but only under certain environmental conditions. The gene-environment relationship is a complex one.

This is something to keep in mind as we discuss the various phenotypic evolutionary changes our species has undergone. Few evolutionary events are as simple as they first seem. When we discuss the evolution of

FIGURE 4.10 The distribution of high frequencies of malaria shows a correspondence with high frequencies of sickle cell anemia (Figure 4.9).

our upright posture or our large brains, you should appreciate the complex genetic changes that must have occurred, as well as the complex interactions between genotype and phenotype and between phenotype and environment.

It is also important to remember that because it operates on variation already present in existing traits, natural selection is not always successful in maintaining the viability of a species. When some environmental change is too severe or too rapid, there may simply not be any variation within a species that enables some of its members to reproduce in quantities sufficient to perpetuate the species. Extinction is the result—indeed, it has been the fate of over 90 percent of all species that have ever existed.

For example, dinosaur species occupied a great diversity of niches and were around in some form for over 100 million years. Yet a rapid and substantial environmental change occurred to which none of the dinosaurs (or too few to matter) had sufficiently adapted traits. As a result, the dinosaurs died out in a fairly short period of time. Human activity also constitutes a

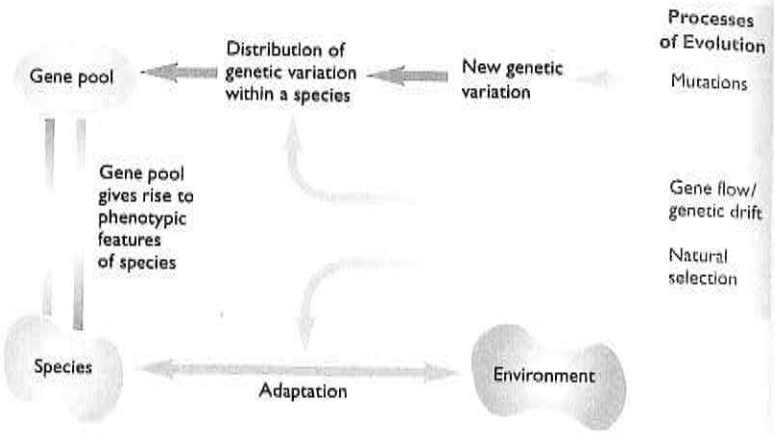


FIGURE 4.11 Processes of evolution. A species is in an adaptive relationship with its environment. This relationship is maintained by natural selection. Environments, however, are constantly changing, so the adaptive characteristics of species change over time. In addition, the gene pool of a species is always changing, altering the phenotypes on which selection acts. Processes that alter a species' gene pool are also, by definition, processes of evolution because they change allele frequency. Mutation provides new genetic variation by producing new alleles or otherwise altering the genetic code. Gene flow and genetic drift mix the genetic variation within a species, continually supplying new combinations of genetic variables.

form of environmental change and can bring about the same kinds of results. Overhunting of passenger pigeons in North America, coupled with the felling of forests that provided their habitat, resulted in the extinction, by 1914, of a species that once numbered in the billions.

Natural selection is not magic, nor is it the only process that changes allele frequency and thus contributes to evolution. Whereas natural selection produces change in the direction of better adaptation, other processes have no adaptive direction. But since they cause the frequency of alleles to change over time, they are considered evolutionary processes (Figure 4.11), and they also provide variation on which natural selection acts. These processes are mutation, gene flow, and genetic drift, which we will examine next.

Mutation

Mutations are random—that is, unpredictable—changes in the material of inheritance. Mutations may affect individual genes, as in the case of sickle cell anemia, the result of one wrong codon in a sequence of 287. Or they may affect a whole chromosome, or a portion of a chromosome, and therefore many genes. Mutations may occur spontaneously as a result of mechanical errors during the processes by which the genetic code either copies itself during cell division or is translated into working proteins. Mutations may also result from certain outside stimuli such as cosmic or nuclear radiation, various chemical pollutants, and some insecticides.

Mutations are frequent. Some have taken place in cells somewhere in your body since you started reading this page. The only ones that matter to the evolution of sexually reproducing species, though, are those that occur in the sex cells or the cells that produce the sex cells. These are the

mutations that can be passed on and can thus change the allele frequencies of a population over time.

Because mutations are sudden, random changes, they may logically be considered mistakes. As mistakes, many have deleterious effects. Such mutations tend to disappear because individuals having them are less reproductively successful. In other words, these mutations are selected against. Other mutations may produce alleles that are neutral in terms of adaptation—that is, at the time they occur they are neither more nor less adaptive than the original allele. And some mutations may even be more adaptive than other variations. In this case, natural selection is provided with new raw material that may be selected for.

Mutation, then, is a source of variation on which natural selection can act. It is also itself a process of evolutionary change because it alters the hereditary material of a species. The effect of a mutation on the species depends, of course, on just what traits are affected and on how important those traits are to the relative reproductive success of individuals possessing them. A small, inconsequential mutation has little or no effect on the individual. As a result, it may or may not be passed to a proportionately large number of offspring; that depends on the success of the individual based on other traits it possesses. A large mutation, or a small one with extensive effects (such as the sickle cell allele), will be passed on to decreasing numbers of individuals if it is deleterious. A trait that kills carriers before they become old enough to mate is unlikely to become common in a population because those with the trait are not able to pass it on. On the other hand, a genetic trait may spread rapidly through the species in subsequent generations if it confers a distinct advantage on those who carry it.

Mutations are changes that affect the hereditary material itself. Natural selection operates on the physical manifestations of the hereditary material based on the adaptiveness of those manifestations. Two other processes of evolution, **gene flow** and **genetic drift**, work at a level between selection and mutation. These processes change allele frequency by altering the frequencies of genotypes, that is, allele combinations. They work, however, without regard to their specific adaptive characteristics.

Gene Flow

Members of a species interbreed with one another. But species tend to be divided into breeding populations, or demes, that are delimited by geographic distance, a specific environmental range and niche, and social organization. These breeding populations within a species may undergo natural selection for their particular environmental situations and may therefore exhibit minor differences among one another. So when members of different populations do interbreed with other populations as a result of migration or the exchange of genes with neighboring populations—when

gene flow The exchange of genes among populations through interbreeding.

genetic drift The change in allele frequency by random fluctuations.

the genes of one "flow" into the **gene pool** of another—the offspring have new genetic combinations. New physical manifestations appear in the mixed population and provide even more raw material for natural selection on the species level.

When flow is extensive among populations within a species, it has the effect of reducing the genetic variation among those populations. A perfect example is our own species; our mobility and tendency to interbreed have blurred physical distinctions among individual populations—the reason why our species cannot be divided into biologically meaningful racial groups (see Chapter 10).

Genetic Drift

Several distinct processes fall under the heading of genetic drift. **Fission** is the opposite of gene flow. When a population within a species splits, the new subpopulations will differ from one another and from the original population in the average phenotypes and genotypes. This may not seem obvious at first, but an example can demonstrate the point. Suppose you calculate the average stature of members of your anthropology class to be 5′ 9″ with a range from 5′ 1″ to 6′ 7″. If you divide the class population into two samples without considering height, do you think the average stature and range for each new group will be the same as the original? The two samples might have a similar mean height and range, but, by chance, they may not; in fact, they may end up being very different. The same applies to genes in natural populations. Any population split—a common enough occurrence—will provide evolutionary change as well as new gene pools for selection to operate on (Figure 4.12).

gene pool All the genes of a population.

fission The splitting up of a population to form new populations.

founder effect Differences in populations caused by genetic differences in the individuals who establish the populations.

gamete sampling The genetic change caused when genes are passed to new generations in frequencies unlike those in the parental population.

This effect is enhanced when the split is uneven—when, for example, 10 percent of a population splits from the original and founds a new population. It is virtually impossible for that 10 percent to possess the same average physical traits, gene combinations, and allele frequencies as the original. This is known as the **founder effect**. The founder effect and fission together form one type of genetic drift.

The other form of genetic drift is **gamete sampling**. When fertilization takes place in sexually reproducing species, the genetic material from two parents is mixed. The potential number of new genetic combinations in the offspring is enormous. You may resemble your parents, but you are not a carbon copy of either, and your specific genetic makeup is absolutely unique to you (unless you have an identical twin). With sexual reproduction, then, change occurs every generation, based solely on the laws of probability applied to the recombination of parental genes in their offspring. This change is not related to the adaptive fitness of the traits involved because it is produced at the time of fertilization, before the environment has a chance to act on the physical traits.

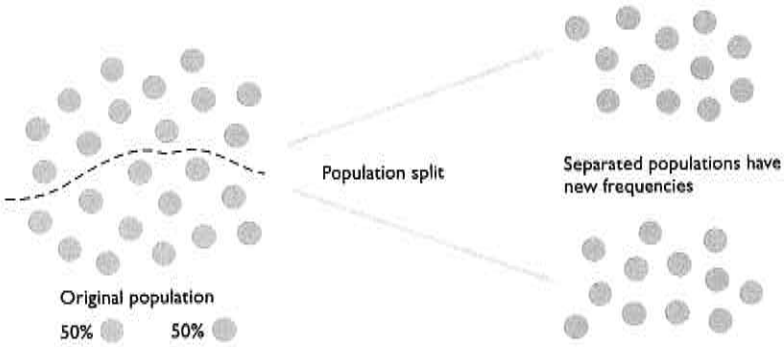


FIGURE 4.12 Diagram of fission, where a population split produces new populations with distinct gene pools. Gene flow, which produces one new genetic population from two or more, can be pictured by reversing the direction of the arrows.

As described in the preceding paragraph, the process of gamete sampling affects only the offspring of one set of parents, but the combined effects of this process at the population level, in many sets of parents and offspring, can bring about a great deal of change from one generation to the next. Especially if the phenotypes coded for by the new genetic combinations are adaptively unimportant, the specific expressions may change at random across generations, "drifting" in whatever direction chance takes them—hence the name genetic drift.

Suppose, for instance, two parents are both heterozygous for a certain locus, say, *Aa*. A Punnett square would reveal that they stand a one-quarter chance of producing an *AA* offspring, a one-half chance of producing an *Aa* offspring, and a one-quarter chance of producing an *aa* offspring. These figures, however, are probabilities, not certainties. Each fertilization is an event independent of all previous fertilizations. They may, for example, produce nothing but *AA* offspring. In that case, all their *a* alleles are lost. In a large population, there is a good chance that two other parents of genotype *Aa* will produce only *aa* offspring, and it will all balance out. But in a small population—of, say, under 100 individuals—the chance of such a balance is very small. In fact, all forms of genetic drift have greater effects in small populations. In this way, some alleles may ultimately be lost and others may reach a frequency of 100 percent all with no necessary relation to adaptation.

As with the other processes, drift produces evolutionary change as well as new population variation on which natural selection may then operate. It may not, however, always be a positive process. A further threat to species already on the brink of extinction because of low population size is the fact that what little genetic variation they have left may be further depleted by the drifting of some alleles to high frequencies and others to low frequencies. The less genetic variation, the less chance a species has of containing enough individuals well enough adapted to reproduce in sufficient numbers. This is part of the current plight of cheetahs, African lions, gorillas, condors, and other endangered species.

The Origin of Species

Although natural selection was the cornerstone of Darwin's theory of evolution, it was not the phenomenon he ultimately sought to explain. What interested Darwin, and Wallace, was the question of where all the species of plants and animals had come from in the first place. Natural selection was the mechanism they proposed as the answer. Darwin, in fact, felt it was *the* answer. Natural selection, he said, brought about "the accumulation of innumerable slight variations, each good for the individual possessor" (1898:267). He added,

> What limit can be put to this power, acting during long ages and rigidly scrutinizing the whole constitution, structure, and habits of each creature,—favoring the good and rejecting the bad? I can see no limit to this power, in slowly and beautifully adapting each form to the most complex relations of life. (1898:267)

So, according to Darwin's view, one way new species arise is as a direct result of constant adaptive change within existing species. Eventually, a species will change so much it evolves into a new species.

In addition, such constant selection, Darwin said, will also produce variation among populations within a species in response to slight differences in their environments. He referred to these populations as "varieties." Eventually, selection brings about such marked distinctions in varieties that two or more new species branch from the old one. These species, said Darwin, "are only well-marked varieties, of which the characters have become in a high degree permanent" (1898:285).

Darwin, then, saw natural selection as more than a mechanism for the origin of species. To him, it was the driving force behind the origin of

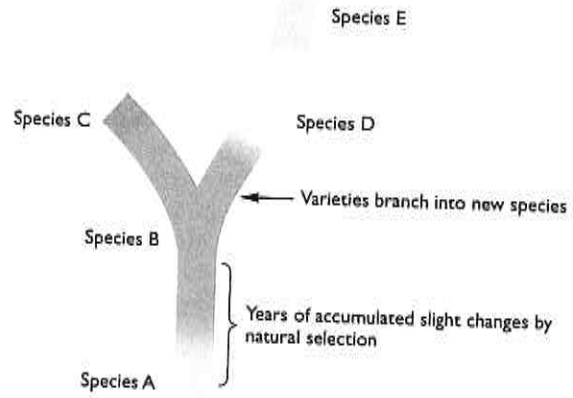


FIGURE 4.13 Darwinian gradualism.

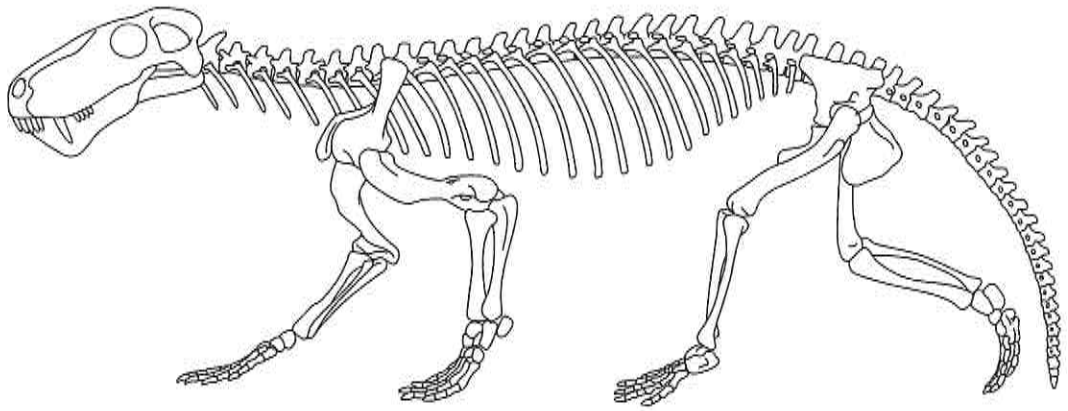


FIGURE 4.14 *Lycaenops*, a mammal-like reptile from 240 mya. Its legs were long and under its body, allowing it to keep its body continually off the ground, like mammals in general but unlike earlier reptiles. *Lycaenops* also had long canine teeth like mammals, though its other teeth were reptilian. *(Neg. #2A3387. Courtesy Department of Library Services, American Museum of Natural History)*

species, constantly choosing from among innumerable small variations those best suited to an environment. The result is the turning of one species into another, whether it occurs gradually but inexorably or produces first new varieties within the species and finally a new species, forming in the process "an interminable number of intermediate forms . . . linking together all the species in each group by [fine] gradations" (1898:271–72). Evolution, according to Darwin, is a long string of small adaptive changes over time. This model of evolution is called **gradualism**, or sometimes **Darwinian gradualism** (Figure 4.13).

Darwin was challenged, however, on one aspect of his model of speciation. If evolution were slow and gradual, where, some of his contemporaries asked, were all those "intermediate forms" in the fossil record? Darwin's answer was that the fossil record was "imperfect," that more time and study would reveal all the transitions. He was overly optimistic.

Although the last 140 years have brought to light fossils representing the transitions between major forms of life (dinosaurs to birds and reptiles to mammals, for example; Figure 4.14), transitional forms between individual species have, for the most part, failed to appear. Moreover, species, for the most part, seem to remain fairly stable throughout their tenure on earth. The concrete evidence does not fully support Darwin's model.

Moreover, Darwinian gradualism presents a theoretical problem. If each small variation selected for is "good for the individual possessor," then we must account for the adaptive benefit of each small step toward the development of some completed characteristic. Could one-tenth of a wing, or one-hundredth of an eye—like a millimeter in those finches' beaks—*always* convey to its possessor a reproductive advantage over the members of its species that lack this trait?

It would seem, then, that evolutionary change over great spans of time is not the result of gradual change *within* species (individual species changing

gradualism The view that speciation is slow and steady with cumulative change.

Darwinian gradualism The same as gradualism.

so much they become a new species) but, rather, the result of new species branching from existing species. Indeed, natural selection is not the creative "scrutinizing" force that Darwin envisioned, but a more conservative force, simply eliminating what doesn't work adaptively and allowing to reproduce what does. Change *within* a species is limited.

This does not mean that natural selection has *no* effect on a species over time. Traits that are important may change back and forth as environmental conditions change. Recall the changes in beak size among the Darwin's finches studied by the Grants and their team. The data indicate that a slight difference in beak size among members of one species—as little as a millimeter or two—can be of adaptive importance during sudden, prolonged, or radical environmental changes such as droughts. In fact, small variations made the difference between life and death, and so, after such an environmental episode, the average beak size of an affected finch species could be significantly altered. When conditions—and, thus, food sources—returned to normal, the average beak size often returned to its previous measurement. The average expression of important traits of a species, then, may change back and forth as environmental conditions change. This is called **oscillating selection.** But it is adaptive variation—a sort of fine-tuning—around a norm, rather than continual change in a particular direction.

Speciation, however, is a frequent process in the sense that new species of some sort evolve all the time (although most events of speciation involve smaller species with short generations). It is also a relatively rapid process in a geological time frame where thousands of years constitute a few seconds on our cosmic calendar. Speciation occurs when a portion or portions of a species are isolated from the species as a whole. If sufficient variation exists to allow this new population to survive and reproduce in its new environment, it may accumulate enough differences from its parent species to become a separate species, reproductively isolated from the parent species. This isolation may be physical. For example, the thirteen species of Darwin's finches that inhabit the Galápagos were descended from a single South American species. Over a long span of time, individuals from the original population were blown or floated out to sea from the mainland, and a few managed to end up on the dozen or so major Galápagos Islands, where they adapted to the various niches on the islands. Natural selection to those niches, along with the relative isolation of the islands and long periods of time, allowed the finch populations to diverge to such a degree that they are now considered separate species, characterized by such features as differences in size and other features such as their beaks. Subsequent movement has resulted in a dispersal of the various species among the islands. Some of the larger and more ecologically varied islands of the group support as many as ten finch species (Figure 4.15).

The isolation may also be genetic. Mutations were once thought to involve only slight alterations. Now, however, we recognize that not all genes

oscillating selection Adaptive variation around a norm, rater than in one direction, in response to environmental variation in a species' habitat.

speciation The evolution of new species.

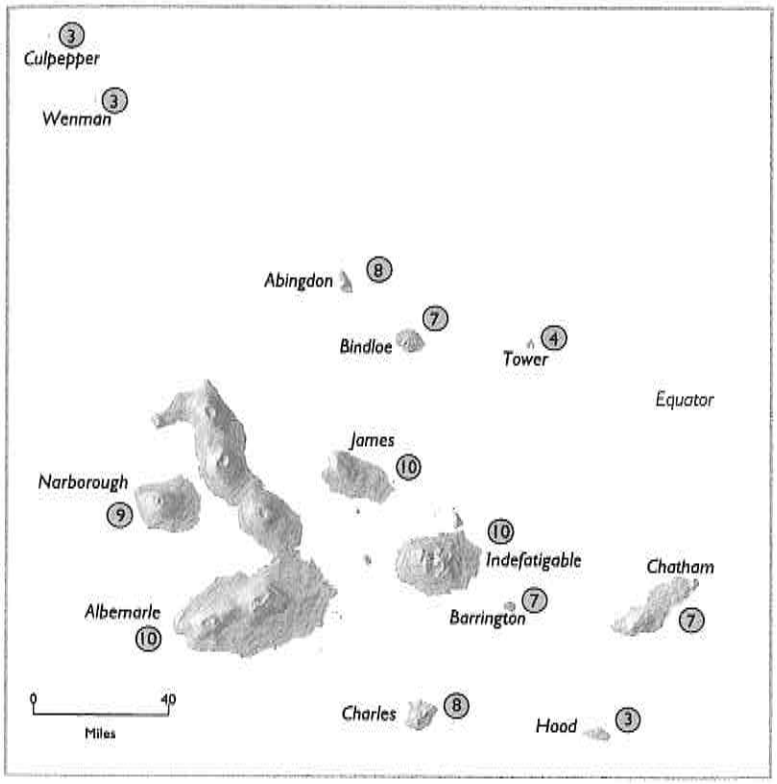


FIGURE 4.15 The various species of Darwin's finches evolved when small groups from an original species underwent adaptation to the varying environmental conditions found throughout the Galápagos Islands. The numbers on the map represent the number of finch species found on each major island.

are of equal importance and that a mutation of large numbers of genes or of genes that code for important phenotypic traits can produce a major change in the offspring of the organism that passes that mutation on.

There are, for example, genes influencing developmental changes that act on the individual at an early age but may have important consequences for the structure and function of the adult organism. There appears to have been one such change, more than 140 mya; that altered the development of dinosaur skin to produce featherlike structures, thus beginning the evolution of the birds (Figure 4.16). Thus, if multiple members of an existing species (say, all the members of a litter or all the hatchlings in a nest) share such a mutation, with the result that they cannot interbreed with the rest of the species, or their differences severely limit interbreeding, speciation may take place very rapidly.

This model of evolutionary change, first proposed in its modern form in 1972 by paleontologists Niles Eldredge and Stephen Jay Gould (1972), is called **punctuated equilibrium**. It says that the evolutionary histories of species are marked by equilibrium—long periods of little change—with

punctuated equilibrium The view that species tend to remain stable, with evolutionary change arising fairly suddenly through the breaking off of a new species.

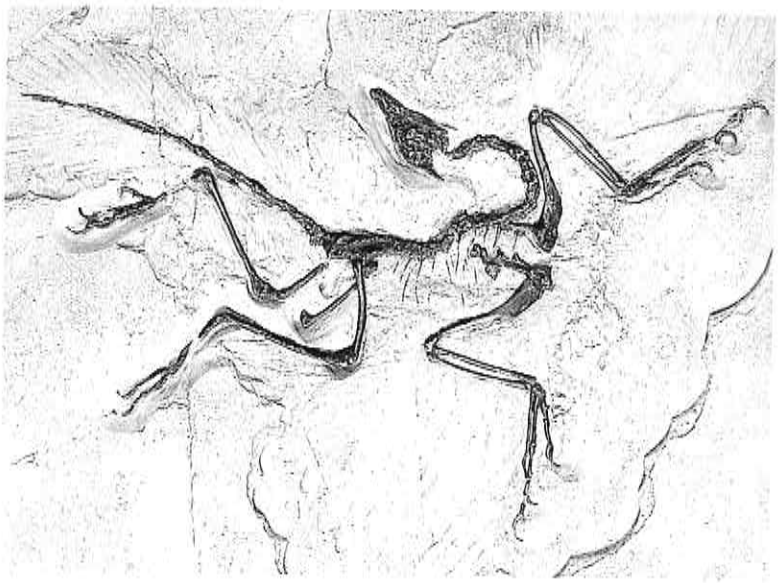


FIGURE 4.16 The fossil remains of *Archaeopteryx* ("ancient bird"), about 150 million years old. It is actually a small, bipedal dinosaur with feathers. These feathers, modifications of dinosaurian scales, are an example of a sudden but sizable alteration in a species' genetic makeup that eventually gave rise to a whole new group of organisms. *(© Tom and Therisa Stack/Tom Stack & Associates)*

natural selection acting largely in a conservative way to maintain the species' adaptation to its environment, mostly by selecting against maladaptive variations but also by bringing about, if possible, small changes back and forth as adjustments to environmental alterations (oscillating selection). This equilibrium, however, is punctuated by bursts of change in the form of speciation events. These events occur when a portion of a species is isolated, either physically or genetically. In time, a new species may evolve. This is not an instant process; it may take thousands of years. Indeed, although we recognize thirteen species names for the Galápagos finches, some of these species can interbreed when altered environmental circumstances cause their niches to overlap. In other words, they might be considered as still somewhere in the process of becoming true species. But speciation is not the gradual evolution Darwin had proposed, marked by a long series of small transitional steps. It begins with a big step—the isolation of a portion of a species or a macromutation—providing natural selection with something brand new and very different to work with (Figure 4.17).

So the evolution of life on earth cannot be depicted as a ladder or chain representing a steady march of progress toward complexity, as

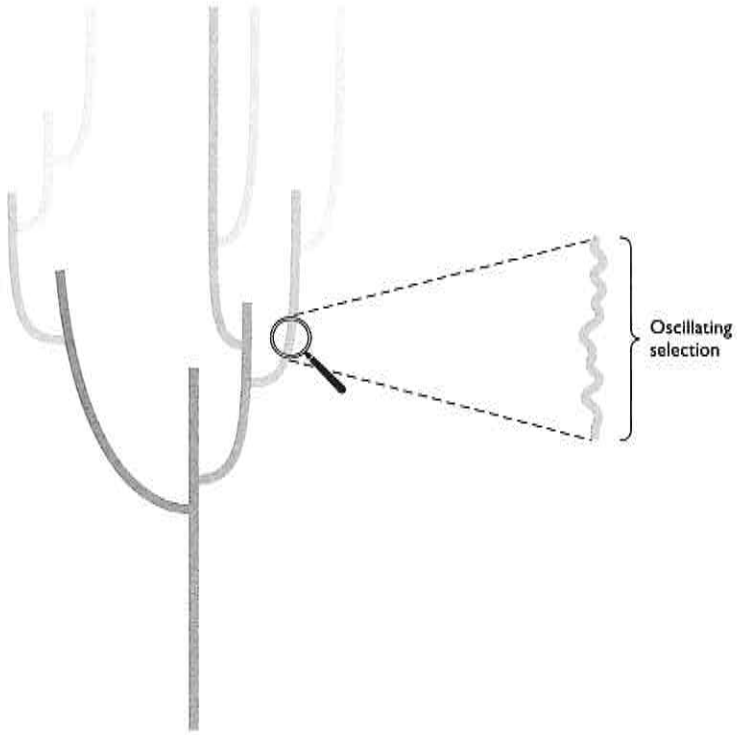


FIGURE 4.17 The shape of evolution according to the punctuated equilibrium model. Major evolutionary change is the result of speciation, the branching of new species from existing ones. Individual species change relatively little over time, although natural selection still acts constantly to maintain a species' adaptation to its environment.

Lamarck and other early scientists believed. Nor is it a gracefully branching tree, as Darwin pictured it. Instead, evolution is, in the words of Stephen Jay Gould (1994:91), a "luxuriant bush," more complex than we will probably ever know—or, as Gould puts it in another work (1985:355), a "blooming and buzzing confusion."

Summary

To fully account for the evolution of species, an understanding of the workings of genetics is vital. Genes code for the traits that make up the physical organism by giving instructions for the synthesis of proteins. Proteins make cells and control their functions, and cells, in turn, make up the tissues that make up the organism.

The gene pool of a species changes over time because of four processes that alter the frequencies with which the alleles of genes appear. Mutations produce new alleles or, on the chromosomal level, new sequences or combinations of alleles. Gene flow shuffles the alleles of populations within a

CONTEMPORARY ISSUE

What Is Genetic Cloning?

Since advances in genetic technology enabled the cloning of Dolly the sheep in 1997, that ability and its potential applications have been areas of both great scientific interest and heated controversy, much of the latter due to a poor general understanding of what the term *cloning* means. What immediately comes to mind are the plots from popular science fiction novels and movies. For example, in Ira Levin's *The Boys from Brazil*, a Nazi scientist produces clones of Hitler, each, of course, with Hitler's personality.

Not only is something like that impossible, but it doesn't even come close to exemplifying the scientific meaning of cloning. The term *clone* simply refers to an exact or nearly exact copy of a biological entity, whether an individual or a cell. Identical twins, by that definition, are natural clones because they began life as a single fertilized egg cell and are, essentially, genetically the same.

In terms of artificial cloning, there are two main types. And the distinction is important. Dolly and members of at least seven other species are examples of *reproductive cloning*. Here, the goal is the production of a genetic duplicate of an organism. Motivations range from increasing productivity of food animals to producing organs for transplantation to replicating pets. Not only are the functions of reproductive cloning open to ethical considerations, but at present there are severe limitations on its success, including the fact (probably mercifully) that it doesn't seem to work with primates (Simerly et al. 2003).

Specific techniques differ, but in the most widely used method, the chromosomes are removed from an unfertilized egg shortly after ovulation, when it would be ready to develop if stimulated by the sperm. The donor cell, the one to be copied, is a somatic (body) cell, often a skin cell or a mammary cell. It is fused with the egg cell with the help of electric pulses, which also mimic the stimulation of fertilization. After about four or five days in a chemical solution, if all goes well, the fused cells will have developed into an embryo of several hundred cells. At this point, the embryo is transferred to the uterus of a surrogate mother of the species in question with the hope that it will develop normally. If it does, as was the case with Dolly, the new organism will be an

species to produce new genotypic frequencies. Due to genetic drift, new genotypic frequencies occur each time a population splits and each time a new generation is produced. And natural selection affects allele frequencies via the differential reproduction of the carriers of adaptive phenotypes.

Given sufficient change and the right circumstances, new species can evolve from existing ones. Charles Darwin thought this occurred as a result of the gradual yet inexorable force of selection slightly changing each species every generation. We now understand that natural selection is more a conservative force than a creative one, largely acting to maintain the adaptation of a species to its environment. Speciation occurs when a portion of a species is isolated, physically or genetically, and, if it gains an adaptive foothold, starts off on its own evolutionary path, becoming different enough to be reproductively isolated from its parent species.

exact genetic copy of the individual from whom the donor cell was taken.

One misconception about this technique is that the clone will be a phenotypic duplicate of the donor. To be sure, clone and donor will be very much alike. But genes do not act alone in producing phenotypic traits, and many variables are involved in the path from single egg to multicellular organism. The more complex the trait, the more variable the potential results. So a clone of me would look like me but would not have all my personality traits. Even if we could clone humans, we would not be making exact copies.

The second type of genetic cloning is *therapeutic cloning*, in which cells and eventually tissues are grown *in vitro* (in a chemical culture) for medical purposes. No reproduction of individuals is involved. The idea is to make copies of stem cells. These are cells, such as a fertilized egg and cells from early in embryonic development, that have the potential to become all the different cell types in the body. In a few days, cells begin to specialize. At this stage (which is the same stage an embryo is implanted into a surrogate in reproductive cloning), certain cells are removed from the embryo in the hope of differentiating them into specific types of tissues to replace or repair a patient's tissue damaged by certain diseases, including Parkinson's disease, muscular dystrophy, and diabetes.

An objection to stem cell cloning is that an embryo—a potential life—is intentionally created only to be destroyed when the wanted cells are removed. But a new technique can "trick" an egg cell into "thinking" it has been fertilized so that it clones itself into an embryo. Such embryos are nonviable and could not lead to a pregnancy. In addition, there are stem cells still present in adult animals, and these could potentially be cloned and artificially differentiated. Ethical objections to therapeutic cloning seem easier to overcome than those to reproductive cloning, and the benefits to human health are of potentially inestimable value.

Both research and debate will continue on cloning, as well they should. But the former should be done with clear and beneficial goals in mind and the latter with sound, accurate knowledge of the science involved.

Study Questions

1. What are genes, and through what steps do they produce the traits that make up a living organism?
2. What are the basic laws of inheritance; that is, how are traits passed on from parent to offspring?
3. How may we study the genetic makeup of populations as well as genetic change in populations through the use of mathematics?
4. What are the processes of evolution? How do they interact to bring about evolutionary change?
5. How do existing species give rise to new species?
6. How is sickle cell anemia an example of all aspects of modern evolutionary theory?

Key Terms

genetics
particulate
gene
protein
enzyme
deoxyribonucleic acid (DNA)
chromosome
replication
protein synthesis
messenger ribonucleic acid (mRNA)
codon
transfer RNA (tRNA)
amino acid
allele
genotype
genome
homozygous
heterozygous
mutation
phenotype
recessive
dominant
gamete
segregation
zygote
point mutation
codominant
monogenic
polygenic
population
breeding population
deme
allele frequency
Hardy-Weinberg equilibrium
differential reproduction
niche
sexual selection
gene flow
genetic drift
gene pool
fission
founder effect
gamete sampling
gradualism
Darwinian gradualism
oscillating selection
speciation
punctuated equilibrium

For More Information

Many excellent books on evolution and evolutionary processes are available. We recommend Mark Ridley's *Evolution*, second edition. The history of evolutionary thought, with excerpts from original sources, is covered in C. Leon Harris's *Evolution: Genesis and Revelations.*

A must for anyone interested in genetics—or, for that matter, the nature of scientific inquiry in general—is *The Double Helix* by James D. Watson, about the race to discover the nature of the genetic code and thereby win the Nobel Prize. The author was one of the winners. For an up-to-date piece on new discoveries in genetics, and their applications, see "Secrets of the Gene" by James Shreeve in the October 1999 *National Geographic.*

For the original announcement of the completion of the human genome, see the "Science Times" section of the February 3, 2001 *New York Times*, the February 15, 2001 issue of *Nature*, and the February 16, 2001 issue of *Science* (in the last publication, see page 1163 for a list of Web sites on the genome project and data).

The reseach on Darwin's finches and the people who conducted it are the subjects of Jonathan Weiner's Pulitzer Prize–winning *The Beak of the Finch: A Story of Evolution in Our Time.* For an update, see Peter and Rosemary Grant's article "Non-Random Fitness Variation in Two Populations of

Darwin's Finches," in the January 22, 2000 issue of the *Proceedings of the Royal Society of London: Biological Sciences* and their "Unpredictable Evolution in a 30-Year Study of Darwin's Finches" in the April 26, 2002 issue of *Science*.

Cloning technology is summarized in "Cloning for Medicine," by Ian Wilmut (who cloned Dolly), in the December 1998 *Scientific American*.