anthropology

what does it mean to be human?

FOURTH EDITION

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What can evolutionary theory tell us about human variation?

Not everyone looks the same. Why is that? Does it make a difference? Do the differences cluster together? In this chapter, we will look at the way evolutionary theory explains patterns of human biological variation. In particular, we will show why anthropologists have concluded that these patterns cannot be explained by the concept of biological "race."

CHAPTER OUTLINE

Not everyone looks the same. Why is that, and what does it mean?

Chapter 4 looked at **macroevolution** as applied to the history of humans and their closest relatives. This chapter shifts the focus to **microevolution**, which devotes attention to short-term evolutionary changes that occur within a given species over relatively few generations. It is measured in what is sometimes called "ecological time," or the timescale experienced by organisms living and adapting to their ecological settings.

What Is Microevolution?

The Modern Evolutionary Synthesis and Its Legacy

In the 1930s and 1940s, biologists and geneticists worked to formulate a new way of thinking about evolution that combined Darwinian natural selection and Mendelian ideas about heredity. Until recently, this approach (called the "modern evolutionary synthesis" or "neo-Darwinism") dominated research and thinking in biology. As we saw in Chapter 2, contemporary evolutionary theorists have challenged, expanded, and enriched this neo-Darwinian research program, much the way the formulators of the modern synthesis had earlier challenged, expanded, and enriched the contributions made by Darwin, Mendel, and other early evolutionary thinkers. But some achievements of the modern synthesis remain fundamental to our understandings of living organisms. In anthropology, perhaps the most significant contribution of neo-Darwinism was the way it undermined the nineteenth-century anthropological concept of "biological race," refocusing attention on a new understanding of biological species. After World War II, anthropologists like Sherwood Washburn rejected the old, race-based physical anthropology of the nineteenth and early twentieth centuries and replaced it with a "new physical anthropology" or "biological anthropology." Research in biological anthropology took for granted the common membership of all human beings in a single species and addressed human variation using concepts and methods drawn from neo-Darwinism (Strum et al. 1999).

microevolution A subfield of evolutionary studies that devotes attention to short-term evolutionary changes that occur within a given species over relatively few generations of ecological time.

macroevolution A subfield of evolutionary studies that focuses on long-term evolutionary changes, especially the origins of new species and their diversification across space and over millions of years of geological time.

species A distinct segment of an evolutionary lineage. Different biologists, working with living and fossil organisms, have devised different criteria to identify boundaries between species.

Biologists have proposed alternative definitions of **species** that attempt to respect the purpose of Darwinian taxonomy, which is to represent scientists' best current understanding of the relationships between and among organisms. As biological anthropologist John Fleagle points out, "Most biologists agree that a species is a distinct segment of an evolutionary lineage, and many of the differences among species concepts reflect attempts to find criteria that can be used to identify species based on different types of information" (Fleagle 2013, 2). Neo-Darwinians defined a species as "a reproductive community of populations (reproductively isolated from others) that occupies a specific niche in nature" (Mayr 1982, 273). This definition, commonly referred to as the *Biological Species Concept*, has been useful to field biologists studying populations of living organisms. However, this definition of species has been less useful for scientists studying fossils. In fact, Fleagle notes that the Biological Species Concept has even been losing favor among field biologists because "as more and more 'species' have been sampled genetically, it has become clear that hybridization between presumed species has been very common in primate evolution" (Fleagle 2013, 1; see also Stringer 2012, 34).

As we saw in Chapter 3, many taxonomists working with living primates prefer to use the *Phylogenetic Species Concept*, which identifies species on the basis of a set of unique features (morphological or genetic) that distinguish their members from other, related species. Contemporary paleoanthropologists also often rely on this concept of species, as we saw in Chapter 4, although they also sometimes apply a Phenetic Fossil Species Concept. Users of the Phenetic Fossil Species Concept first attempt to calculate the measurable morphological differences between living species. They then assume that similar degrees of morphological difference may also be used to distinguish species in the fossil record. Fleagle observes that this concept can be a useful way to sort fossils in a continuously changing lineage "in which the endpoints may be very different but individual samples overlap" (2013, 2).

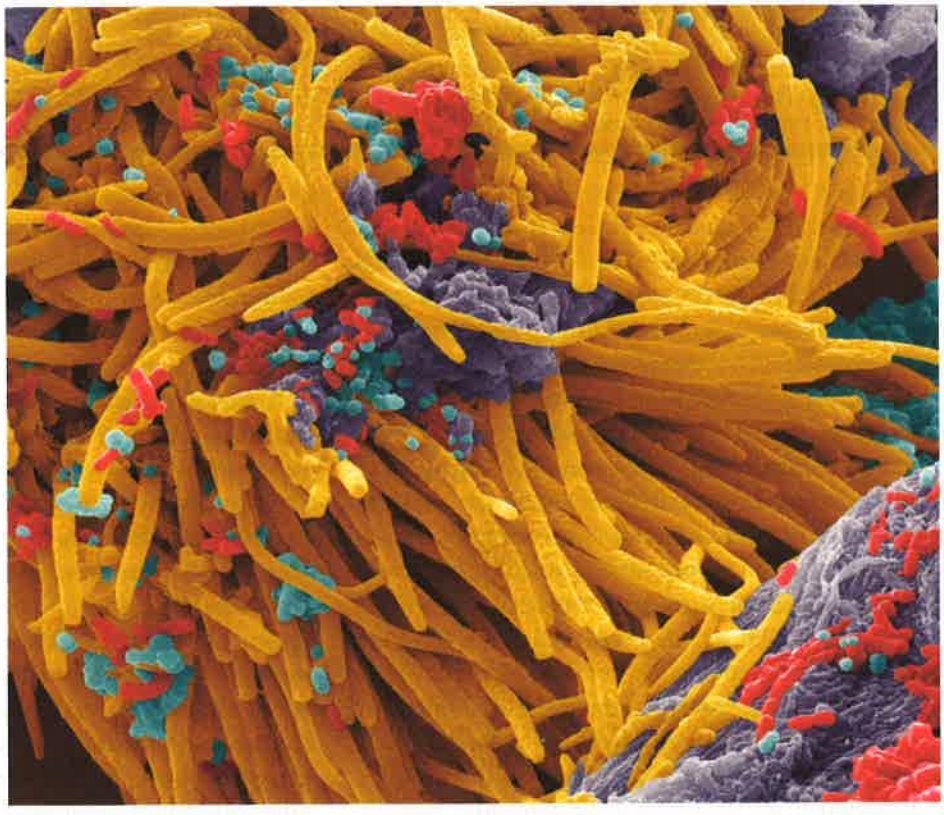
Species normally are subdivided into *populations* that are more or less scattered, although the separation is not complete. That is, populations of the same species (or individual members of those populations) may be separated at one time, but may merge together again, and successfully reproduce, at a later time. Evolutionary theorists Ian Tattersall and Rob DeSalle describe this process of species differentiation and reintegration as *reticulation* (Tattersall and DeSalle 2011, 50). They emphasize that reticulation takes place *within species* and that the "resulting weblike pattern of relationships is very different from the dichotomous pattern among species"

IN THEIR OWN WORDS

Have We Ever Been Individuals?

Evolutionary biologists committed to the Modern Evolutionary Synthesis, interested in carrying out microevolutionary studies of natural selection on genes, have worked hard to clarify distinctions between species, populations of a particular species, and organisms that belong to such populations. Now, the seemingly self-evident, taken-for-granted boundary distinguishing one individual organism from another is not looking so self-evident after all—even among mammalian species, such as ourselves. Recent research is showing that it is incorrect to assume that each biological individual (such as an individual human organism) is also a genetic individual; that is, in possession of just a single genome. On the contrary, each human organism contains within it multiple communities of different species of microbes, each with its own separate genomes, living with us in a mutually beneficial association called symbiosis. Biologists Lynn Chiu and Scott Gilbert explain that 90% of the cells in mammalian bodies belong to populations of different species of microbes that affect a range of chemical processes supporting our ongoing health and well-being. Some of these microbes contribute to our digestive processes; others to the construction of our bodies; others to our brain function; still others keep our immune system operating properly. And these symbiotic relationships are ancient. As Chiu and Gilbert put it, "Development is a multi-species project. The mammalian body requires its symbionts; it is not constructed properly if it does not have them" (2015, 193).

For these reasons, biologists suggest that the proper term to identify organisms such as ourselves is not "individual" but rather "holobiont," a label that acknowledges the fact that each of us contains within ourselves multiple communities of symbionts of different species. Thinking of organisms as holobionts reshapes the way we think about not only our relation to other organisms but also the way we understand our own life cycles. In particular, Chiu and Gilbert argue that thinking of humans as holobionts reshapes our understanding of what happens when we reproduce. We can no longer consider human reproduction to involve only a male individual and a female individual, whose individual genetic endowments are joined to produce an individual offspring. Rather, we



This scanning electron micrograph shows some of the bacteria living in your mouth. Humans and other animals have symbiotic relationships with microbes that are integral to various biological functions such as digestion.

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IN THEIR OWN WORDS

Have We Ever Been Individuals? *Continued*

need to reconceptualize human reproduction as "holobiont birth," in which individual persons and their symbiotic communities are all involved. Thus, they write, "There is never an autonomous mammal. . . . Symbiosis is a necessary condition for continued life. From the symbiotic perspective, birth is a transition from one symbiotic state to another. Remarkably, this transition appears to be mediated by the mother" (2015, 195). Indeed, they identify four processes through which "the mother creates conditions suitable for her own reproduction and the reproduction of symbiotic microbes" (2015, 196): the physiology of the pregnant women, including hormone levels, modify populations of helpful microbes in her gut and vagina; the mother transfers helpful bacteria to her fetus during gestation; further helpful bacteria are transferred to the infant during vaginal birth; and additional helpful bacteria are transferred to nursing infants via the mother's milk.

Recognizing the symbiotic relationships that characterize holobionts cannot be missed unless biologists pay close attention to developmental processes over time. In the case of holobiont birth, paying attention to processes requires rethinking the relationship between the human host and the multiple symbiotic communities of bacteria that live within it. That is, it is incorrect to conceive of the host as a static, self-interested, independent "habitat" colonized by static, self-interested, independent species. Rather, from the perspective of biological process, it becomes clear that different symbionts provide different niches for one another over time; that symbionts therefore support the ongoing life processes of one another. Even though each individual symbiont does not support every other symbiont all of the time, the overall network of interactions among all symbionts together supports and sustains the ongoing life process of the holobiont.

And this has implications for how we understand human reproduction. Rather than conceiving of the relationship between father, mother, and offspring (or between their genes) as competition for limited resources, the birth of the holobiont highlights the heterogeneous connections among host, symbionts, and offspring. As Chiu and Gilbert conclude,

> The past decade has brought about remarkable new discoveries about relationships between and within organisms. One of the most revolutionary of these discoveries has been the importance of symbiotic signals used to build, maintain, and protect a holobiont. Developmental symbiosis merges embryology and ecology in interspecies webs of mutual and reciprocal communication. Birth is seen not as the origin of a new individual, but as the perpetuation of these organizing webs of signals between animals and microbes. (2015, 205)

on which the Phylogenetic Species Concept is based (Tattersall and DeSalle 2011, 50). For example, prior to the rise of the great ancient civilizations, the human species was made up of widely scattered populations. Those populations living in North America had been separated from populations in Europe for thousands of years, until the European explorations of the Americas began in the fifteenth century. However, when Europeans and the native peoples of North America did come into contact, they were able to interbreed and produce viable, fertile offspring. From the perspective of the Biological Species Concept, this ability to interbreed and produce fertile offspring indicates that members of these different populations belong to the same reproductive community and hence the same species. Proponents of the Phylogenetic Species Concept can specify the set of unique features that distinguish all successfully interbreeding populations of the human species from populations of other, related species.

Finally, Darwinian population thinking requires biologists to recognize the distinctiveness of each individual *organism* that belongs to a particular population of a given species. It is variation among individual organisms in particular populations, in particular environmental circumstances, that engenders the Darwinian struggle for existence. To follow arguments made by evolutionary biologists, therefore, these three nesting concepts—*species made* up of *populations* made up of *organisms*—must be kept distinct from one another. It is also important to remember that even if individual *organisms* from *populations* of different *species* occasionally mate with one another, such matings do not necessarily dissolve the species boundary. For instance, horses and donkeys can interbreed to produce mules, but mules are infertile, so the species boundary between horses and donkeys is unaffected by these matings.

Neo-Darwinians were also concerned about the genetic makeup of species. They introduced the concept

TABLE 5.1 Example of Allele Frequency Computation

Imagine you have just collected information on *MN* blood group genotypes for 250 humans in a given population. Your data are as follows:
Number of *MM* genotype = 40
Number of *MN* genotype = 120
Number of *NN* genotype = 90
The allele frequencies are computed as follows:

GENOTYPE	NUMBER OF PEOPLE	TOTAL NUMBER OF ALLELES	NUMBER OF *M* ALLELES	NUMBER OF *N* ALLELES
MM	40	80	80	0
MN	120	240	120	120
NN	90	180	0	180
Total	250	500	200	300

The relative frequency of the *M* allele is computed as the number of *M* alleles divided by the total number of alleles: 200/500 = 0.4.
The relative frequency of the *N* allele is computed as the number of *N* alleles divided by the total number of alleles: 300/500 = 0.6.
As a check, note that the relative frequencies of the alleles must add up to 1.0 (0.4 + 0.6 = 1.0).

Source: Relethford 1996, 66.

of the **gene pool**, which includes all of the genes in the bodies of all members of a given species (or a population of a species). Using mathematical models, evolutionary theorists can estimate the **gene frequency** of particular genes—that is, the frequency of occurrence of gene variants or alleles within a particular gene pool. Measuring the stability or change of gene frequencies in populations over time allowed geneticists to trace short-term evolutionary change in a new field called **population genetics**. Once population geneticists had identified a target population, they analyzed its gene pool by calculating the frequencies of various alleles within that gene pool and trying to figure out what would happen to those frequencies if the carriers of the various alleles were subjected to particular selection pressures (Table 5.1). Some evolutionary geneticists tested these predictions on such organisms as fruit flies, but others concentrated on human beings.

The ability of human beings from anywhere in the world to interbreed successfully is one measure of membership in a single species. Comparing our genotypes provides additional evidence of our biological closeness. As we have seen, most alleles come in a range of different forms (i.e., are **polymorphous**), and known polymorphous variants fall into one of two groups. The first group, *polymorphic alleles*, accounts for most genetic variation across populations. Populations differ not because they have mutually exclusive sets of alleles but because they possess different *proportions* of the same set of alleles. An example is the ABO blood groups: the polymorphic alleles *A*, *B*, and *O* are found in all human populations, but the frequency of each allele differs from population to population. The second group, *private polymorphisms*, includes alleles that are found in the genotypes of some, but usually not all, members of a particular population. One example is a genetically determined blood cell antigen known as the "Diego antigen." The Diego antigen occurs only in Asian and African populations, but 60 to 90% of the members of the populations where it is found do not have it (Marks 1995, 165). This work leads to the inescapable conclusion that the traditional Western concept of "race" makes no sense in terms of genetics. Racial thinking is essentialistic. However, evolutionary geneticist Richard Lewontin demonstrated more than four decades ago that more genetic variation could be found within conventionally identified racial groups than could be found between them (Lewontin 1972). These results, based on population thinking, make it clear that "humankind . . . is not divided into a series of genetically distinct units" (Jones 1986, 324). Ian Tattersall and Rob DeSalle (2011) point out that Lewontin's claims have successfully withstood attempts to reject

gene pool All the genes in the bodies of all members of a given species (or a population of a species).

gene frequency The frequency of occurrence of the variants of particular genes (i.e., of alleles) within the gene pool.

population genetics A field that uses statistical analysis to study short-term evolutionary change in large populations.

polymorphous Describes alleles that come in a range of different forms.

them experimentally for more than forty years (141). This means that the boundaries said to define human races have been culturally imposed in shifting and unstable clusters of alleles (Marks 1995, 117).

It turns out that genetic variation in human populations is mostly a matter of differences in the relative proportions of the same sets of alleles. In fact, the distribution of particular phenotypes shifts gradually from place to place across populations as the frequencies of some alleles increase, whereas those of others decrease or stay the same. Moreover, the distributions of some traits (like skin color) do not match the distributions of other traits (like hair type). Such a pattern of gradually shifting frequency of a phenotypic trait from population to population across geographic space is called a **cline**. Clines can be represented on maps such as that presented later in Figure 5.4, which shows the gradually shifting distribution of differences in human skin color from the equator to the poles.

Phenotypic contrasts are greatest when people from very different places are brought together and compared, while ignoring the populations that connect them (Marks 1995, 161). This is what happened when Europeans arrived in the New World, conquered the indigenous peoples, and imported slaves from Africa to work on their plantations. But if you were to walk from Stockholm, Sweden, to Cape Town, South Africa (or from Singapore to Beijing, China), you would perceive gradual changes in average skin color as you moved from north to south (or vice versa). Evolutionary biologists argue that skin pigmentation is distributed in this way as a consequence of natural selection: individuals in tropical populations with darker skin pigmentation had a selective advantage in equatorial habitats over individuals with light pigmentation. By contrast, populations farther away from the equator faced less intense selection pressure for darkly pigmented skin and perhaps even selective pressures in favor of lighter skins. But *different* selection pressures would have been at work on other traits, such as stature or hair type, within the same population, which is why the geographical distributions of these traits do *not* match up neatly with the distribution of skin pigmentation. To make things even more complex, different genes may be involved in the production of similar phenotypic traits in different populations: for example, although different ancestral populations of humans living near the equator have dark skin, the identity and the number of alleles involved in the production of this phenotypic trait may be different in different populations.

cline A pattern of gradually shifting frequency of a phenotypic trait from population to population across geographic space.

Evidence for this gradual geographical intergradation of human phenotypes led biological anthropologist Frank Livingstone (1964) to declare more than 40 years ago that "There are no races, there are only clines" (279). Clinal variation explains why people searching for "races" have never been able to agree on how many there are or how they can be identified. *Clines are not groups.* The only group involved in clinal mapping is the entire human species. Each cline is a map of the distribution of a *single* trait. Why not, therefore, superimpose a grid over a particular geographical region, and then sample individuals randomly from the grid squares? As Peter Wade and his colleagues point out, "Starting with a grid tends to produce gradients or clines of gradual variation and reduces the impression of located genetic populations; the absence of boundaries suggests the continuous movement and biological mixture of peoples between populations" (2014, 23). Although many people may think that human population movement and mixture is relatively recent, studies of ancient DNA are now suggesting that human populations have been moving and mixing with one another for hundreds of thousands of years, if not longer (Bolnick et al. 2016, 328). And modern clinal mapping reveals similar patterns of movement and mixture.

Biologists might compare the clinal maps of trait A and trait B to see if they overlap and, if so, by how much. But the more clines they superimpose, the more obvious it becomes that the trait distributions they map *do not coincide* in ways that neatly subdivide into distinct human subpopulations; that is, clinal distributions are *not concordant*. Since the biological concept of "race" predicts exactly such overlap, or concordance, it cannot be correct. In other words, *clinal analysis tests the biological concept of "race" and finds nothing in nature to match it.* And if biological races cannot be found, then the so-called races identified over the years can only be symbolic constructs, based on cultural elaboration of a few superficial phenotypic differences—skin color, hair type and quantity, skin folds, lip shape, and the like. In short, early race theorists "weren't extracting races from their set of data, they were imposing races upon it" (Marks 1995, 132).

The Molecularization of Race?

During the 1960s and 1970s, anthropologists and others explained that there was no biological basis for race; in other words, all humans are part of a single species. Although there is internal variation within the species, it does not easily fall into the cultural categories of "race" as they had developed in the United States. In the past thirty years, however, we have witnessed in the United States and elsewhere a resurgence of attempts to explain

group differences in terms of race. Sometimes it is the powerful who engage in such practices, in controversial books such as *The Bell Curve* (Herrnstein and Murray 1994). Sometimes, however, it is members of politically and economically marginalized groups who do so, as a calculated move in political struggles with those who dominate them.

Perhaps no more complicated set of questions has been raised about race in the twenty-first century than those that have emerged following the completion of the Human Genome Project (HGP) in 2003. The goals of the project were as follows:

- to identify all the approximately 20,000–25,000 genes in human DNA
- to determine the sequences of the 3 billion chemical base pairs that make up human DNA
- to store this information in databases
- to improve tools for data analysis
- to transfer related technologies to the private sector
- to address the ethical, legal, and social issues that may arise from the project (http://www.ornl.gov/sci/techresources/Human_Genome/home.shtml)

As anthropologist Nadia Abu El-Haj (2007) has shown, some molecular biologists quickly mobilized the information produced by the HGP to attempt to develop forms of medical treatment based on the identification of genes associated with particular diseases. Some formed private biomedical research companies that promised to help create a future of *personalized medicine*: therapies based on knowledge of individuals' genomes that were precisely tailored to a particular individual's degree of genetic risk for a particular disease.

In recent years the cost of sequencing individual genomes has been dropping; Tattersall and DeSalle predict that "with the $1000 genome on the horizon, we will soon have the ultimate tool for individualized medicine" (2011, 184). However, the cost has been high enough that many researchers have used genetic data from other members of populations to which individuals belong as a surrogate, or stand-in, for an individual's particular genome. For example, if your mother's brother suffers from a particular disease with a genetic component, researchers may conclude that you and other biological relatives have an increased risk for that disease. That is, your biological family becomes a surrogate, or stand-in, for genetic risk factors that potentially are faced by individual family members. As Abu El-Haj explains, some biomedical researchers in the United States use "racial" groups as surrogates for individuals who consider themselves members of such groups. The thinking is that if a genetic disease marker shows up in the genomes of some people said to be members of a particular "race," then this may be an indication that other people classified in the same "race" might also be at risk for the disease.

Does this pragmatic use of race in medical research mean that the researchers are committed to the doctrines associated with scientific racism? Abu El-Haj (2007, 284) says no, for two reasons. First, the old race concept focused on the classification of *phenotypes*, whereas the new race concept classifies *genotypes*. The transition from a phenotypic to a genotypic view of race came about, she says, as a consequence of changing historical understandings of sickle-cell disease in the United States. In the first part of the twentieth century, sickle-cell anemia was identified as a disease of "black" people—of African Americans. But later, as we will shortly discuss, research in population genetics traced its cause to molecular genes: the presence of an abnormal "sickling" hemoglobin allele at a particular locus on a chromosome. "At the meeting point between these two definitions of the disease . . . the commitment to race as a molecular attribute took form," leading over time to "the correlation of disease risk and racial difference" (Abu El-Haj 2007, 287).

Second, nineteenth-century race science aimed to discover how many races existed and to assign all individuals to their "true race." The commercial technologies used by biomedical researchers regularly distinguish human populations in terms of the continents from which their ancestors presumably came. But all these technologies assume that everyone has a mixed ancestry of some kind; the goal is to measure how much of which ancestry markers are present in each population, thereby determining the degree of risk that members of that population face for genetic diseases associated with particular ancestries. As Abu El-Haj (2007) says, ancestry markers "are not used to discover one's 'true' race. . . . Instead, ancestry markers are used, for example, to understand the Puerto Rican population's risk for asthma" (288). That is, if genome analysis determined that some ancestral population contributed genes to contemporary Puerto Rican populations that enhanced their risk for developing asthma, this information would be crucial in devising personalized drugs precisely keyed to individuals with different risks for asthma.

Third, Abu El-Haj (and others) have pointed out that many African Americans view medical research and drug trials in which they are involved to be nothing less than a form of long-overdue biomedical justice. Anthropologist John Hartigan recently reviewed studies showing that, starting in the 1980s, the U.S. government began to respond to pressure from racial minorities protesting the fact that most medical research focused on white males only. The exclusion of groups like African Americans in such research, however, was the result of

"reforms in the 1970s to counter researchers' excessive reliance on 'vulnerable populations' such as women and prisoners" (Hartigan 2013, 9–10). One notorious example was African Americans' past participation in the Tuskegee Study of Untreated Syphilis in the Negro Male, conducted between 1932 and 1972. According to the website for the Centers for Disease Control and Prevention (http://www.cdc.gov/tuskegee/timeline.htm), a review panel set up in 1972 found that participants in this study

> had agreed freely to be examined and treated. However, there was no evidence that researchers had informed them of the study or its real purpose. In fact, the men had been misled and had not been given all the facts required to provide informed consent.
>
> The men were never given adequate treatment for their disease. Even when penicillin became the drug of choice for syphilis in 1947, researchers did not offer it to the subjects. The advisory panel found nothing to show that subjects were ever given the choice of quitting the study, even when this new, highly effective treatment became widely used.

All these matters come together in the contentious and much-analyzed example of BiDil, a medication designed to treat African Americans suffering from heart disease. On its website (which has since been taken down), NitroMed, the original manufacturer of BiDil, described this drug as "a fixed-dose combination medicine consisting of isosorbide dinitrate and hydralazine hydrochloride. It is approved by the FDA for the treatment of heart failure in self-identified African American patients when added to standard heart failure medicines" (http://www.bidil.com/pnt/questions.php#1). FDA approval, the site reported, was based on the results of the African-American Heart Failure Trail (or A-HeFT), which "studied 10,050 self-identified African American patients with heart failure: It is the largest number of African American patients ever studied in a major heart failure trial. . . . A-HeFT was started on May 29, 2001, and the study was halted early in July 2004 due to a significant survival benefit seen with BiDil as compared to standard therapy alone" (http://www.bidil.com/pnt/questions.php#2).

The original BiDil website also listed a series of "common questions" people ask about BiDil, including the following: "What about claims that BiDil is a 'race drug'?" The site's answer included the following excerpt from a 2007 article by the FDA doctors who approved the drug:

> Only African American patients were studied in A-HeFT, so the FDA approval for BiDil is for "self-identified African American patients with heart failure" only. There is insufficient clinical trial data to draw any conclusions about the effects of BiDil in other populations. . . .
>
> Not understanding the reasons for the difference in treatment effect by race did not justify withholding the treatment from those who could benefit from it. . . . Race or ethnicity is clearly a highly imperfect description of the genomic and other physiological characteristics that cause people to differ, but it can be a useful proxy for those characteristics until the pathophysiological bases for observed racial differences are better understood. (http://www.bidil.com/pnt/questions.php#9)

As these excerpts show, neither NitroMed nor the FDA endorsed nineteenth-century American racial categories. They emphasized that the drug trial showing the effectiveness of BiDil involved only "self-identified" African American subjects, which the FDA agrees is a "highly imperfect" but "useful proxy" for whatever factors are responsible for the observed "racial differences." However, BiDil quickly became the center of a controversy that ended in commercial failure for NitroMed in 2008. In 2011 BiDil was purchased by Arbor Pharmaceuticals, which was still marketing the drug in March 2014 (http://bidil.com/coupon/).

Ann Pollock, who provides a detailed analysis of the BiDil controversy, points out that none of those involved disputed BiDil's efficacy: it worked. Rather, the challenge was to bring the drug to market in a way that would simultaneously address the needs of different stakeholders with an interest in African American heart failure. That is, the FDA, NitroMed, and the Association of Black Cardiologists (ABC) (who carried out the original A-HeFT trials) shared "an interest in health disparities, the deluge of data around African American responses to ACE inhibitors, and the increasing capacity of African American cardiologists to do clinical trials" (Pollock 2012, 162). In Pollock's view,

> In the lead-up to BiDil, there was alignment of interests by NitroMed and ABC, but they were not necessarily seeing BiDil as a solution to the same problem. For NitroMed, the principal problem was how to get approval for the drug combination in a way that would be profitable. . . . For ABC, the problem was and is more diffuse: how to get the funding to run trials and thus participate in the production of evidence-based medicine, and how to find solutions for black morbidity and mortality from heart failure. (2012, 162–63)
>
> The current situation is perplexing, to say the least: such notions as race and "genetics" and "biology" are still with us, but their meanings appear to have changed, producing consequences that seem to be both positive and negative. Some observers suspect that this kind of research will only give the older racial classifications a new lease on life (see In Their Own Words, page 153). John Hartigan (2013) argues,

> however, that although biomedical research of this kind "seems to affirm that 'biological differences' are a more powerful explanation for health disparities than are social factors," the situation is better understood as "the outcome of various ways in which people struggle to contend with the significance of race in multiple social and biological registers simultaneously, often in contradictory manners." (10)

One way to disentangle these matters may be to follow the suggestion of anthropologist Clarence Gravlee and examine more closely a widespread tendency, found among medical researchers and ordinary citizens alike, to equate genetics with biology in discussions of race and disease. Gravlee rightly points out that everyone agrees that race cannot be defined in terms of genetics, as we saw previously. And anthropologists and other social scientists are also well aware of the sociocultural and historical factors in the United States and elsewhere that have created the conditions of racism with which African Americans and other nonwhite groups must contend. However, "the claim that race is not biology unwittingly perpetuates genetic determinism because it tacitly reduces biology to genetics. The more we appreciate the complexity of human biology beyond the genome, the sooner we can explain how race *becomes* biology through the embodiment of social inequality" (Gravlee 2013, 22).

Gravlee (2013) reminds us that many discussions of possible links between race and genetics use "the concept of biology and genetics interchangeably, often pitting these concepts against socioeconomic factors. . . . The implication is that the mere observation of biological differences is sufficient evidence of a genetic one" (30). Instead, he argues, we need to stop using biology as a synonym for genetics and "to pay as much attention to the meaning of biology as we have paid to the meaning of race" (32). Since the deciphering of the human genome, scientists are increasingly learning that many factors other than genes contribute to disease. At the same time, biological theorists have begun to pay closer attention to the factors that affect the health of developing organisms throughout their life course. As we saw earlier, renewed attention is being paid to phenotypic plasticity, a phenomenon, Gravlee reminds us, that Boas was insightfully investigating a century ago.

These considerations have led Gravlee to develop a model of the phenotype that pays attention to a hierarchy of causal influences that shape it over time. As a developing organism encounters these influences (which may have individual, cultural, or historical sources), the organism's responses become *embodied* in the organism's physiology in ways that shape the biological functioning of individual human bodies. "Most relevant," Gravlee (2013) writes, "is the evidence that racism at multiple levels of analysis has direct and indirect effects on health" (33). If we argue that "race is not biology," however, and equate biology with genetics, we blind ourselves "to the biological consequences of race and racism," leaving ourselves "without a constructive framework for explaining biological differences between racially defined groups" (Gravlee 2013, 34).

Gravlee's (2013) approach brings together what anthropologists have learned about "race": first, race does not line up with patterns of genetic variation in human populations; second, race is a sociocultural and historical construct that shapes the circumstances of people's lives; and third, awareness of the consequences for health of living under racist conditions constitutes "a mandate for ethnographic research on the social reality of race and racism . . . to identify . . . the experiences and exposures that shape the emergence and persistence of racial inequalities in health" (41).

Gravlee and his colleagues used this approach to carry out research in Puerto Rico, attempting to explain why darker skin pigmentation was associated with higher blood pressure. They discovered that skin color had two dimensions that needed to be distinguished: "the phenotype of skin pigmentation and the cultural significance of skin color as a criterion of social status" (Gravlee 2013, 38). Measurement of skin pigmentation was carried out using the method of reflectance spectrometry, which reliably estimates the concentration of melanin in the skin. Measurement of the cultural relationship between skin color and social status required ethnographic methods. This "biocultural" (or "biosocial") approach revealed that Puerto Ricans with darker skins and higher socioeconomic status actually experienced higher blood pressure than other Puerto Ricans. This was interpreted as resulting from the fact that such individuals were likely to experience more intense racism as their social status increased, thereby producing increasingly frustrating social interactions that contributed to higher blood pressure (Gravlee 2013, 38). When Gravlee (2013) and his colleagues later included genetic-based estimates of African ancestry, they found that

> adding sociocultural data to the model revealed a statistically significant association between blood pressure and a particular candidate gene for hypertension—an association that was not evident in the analysis including only African ancestry and standard risk factors. This finding suggests that taking culture seriously may both clarify the biological consequences of social inequalities and empower future genetic association studies. (39)

Biocultural or biosocial approaches like that of Gravlee and his colleagues demonstrate, in the words of Greg Downey and Daniel Lende, how "social differences

can become biology because they shape the emerging nervous system" (2012, 31). As Downey and Lende explain, "the predominant reason that culture becomes embodied . . . is that neuroanatomy inherently makes experience material" (2012, 37). Ultimately, they conclude, "The material environment, both natural and artificial, provides structure and information to the growing organism while being incorporated with its inherited biological legacy" (2012, 44).

The Four Evolutionary Processes

What controls the patterns of gene frequencies that characterize a given population? As we have seen, **natural selection** among variant traits is responsible for evolutionary changes in organisms, and **mutation** is the ultimate (and constant) source of new variation. These two important evolutionary processes shape the histories of living organisms; however, they are not the only processes in the natural world that can alter gene frequencies.

Most genetic variation results from mixing already existing alleles into new combinations. This variation is the natural result of chromosomal recombination in sexually reproducing species. However, gene frequencies can be drastically altered if a given population experiences a sudden expansion resulting from the in-migration of outsiders from another population of the species, which is called **gene flow**. A population that is unaffected by mutation or gene flow can still undergo **genetic drift**—random changes in gene frequencies from one generation to the next. Genetic drift may have little effect on the gene frequencies of large, stable populations, but it can have a dramatic impact on populations that are suddenly reduced in size by disease or disaster (the *bottleneck effect*) or on small subgroups that establish themselves apart from a larger population (the *founder effect*). Both of these effects accidentally eliminate large numbers of alleles.

Therefore, modern evolutionists recognize four evolutionary processes: mutation, natural selection, gene flow, and genetic drift. Chance plays a role in each. The occurrence of a mutation is random, and there is no guarantee that a useful mutation will occur when it is needed; many mutations are neutral, neither helping nor harming the organisms in which they occur. Nor is there any way to predict the factors that make population migrations possible or to foresee the natural accidents that diminish populations. Unpredictable changes in the environment can modify the selection pressures on a given population, affecting its genetic makeup. Moreover, as we saw in Chapter 2, *niche construction*—the enduring consequences of efforts organisms make to modify the environments in which they live—can sometimes alter the selection pressures they, their descendants, and other neighboring organisms experience in those environments. As we shall see, control of fire and the invention of clothing made it possible for early humans to colonize cold environments that were inaccessible to earlier ancestors, who lacked these cultural skills. Niche construction of this kind buffers us from experiencing some selection pressures, but it simultaneously exposes us to others.

Today, many biologists and anthropologists agree that the most intense selection pressures our species faces come from disease organisms that target our immune systems and from human-made environmental threats, such as pollution and the ozone hole (Farmer 2003; Leslie and Little 2003). Evidence that microorganisms are a major predatory danger to humans comes from research on the connection between infectious diseases and polymorphic blood groups (i.e., blood groups that have two or more genetic variants within a population). Biological anthropologists James Mielke, Lyle Konigsberg, and John Relethford (2011) point out, for example, that the diseases human beings have suffered from have not always been the same. When our ancestors were living in small foraging bands, they were susceptible to chronic parasitic infections, such as pinworms, or diseases transmitted from animals. After the domestication of plants and animals, however, human diets changed, settled life in towns and cities increased, and sanitation worsened. Populations expanded, individuals had more frequent contact with one another, and the stage was set for the rise and spread of *endemic* diseases (i.e., diseases particular to a population) that could persist in a population without repeated introduction from elsewhere. As a result,

> the increase in endemic diseases started to apply selective pressures that were different from those exerted by chronic diseases. These diseases usually select individuals out of the population before they reach reproductive age. Differential mortality (natural selection) based on genetic variation in the blood types would be expected to influence genetic polymorphisms. Thus recurrent epidemics of diseases such as smallpox,

natural selection A two-step, mechanistic explanation of how descent with modification takes place: (1) every generation, variant individuals are generated within a species as a result of genetic mutation, and (2) those variant individuals best suited to the current environment survive and produce more offspring than other variants.

mutation The creation of a new allele for a gene when the portion of the DNA molecule to which it corresponds is suddenly altered.

gene flow The exchange of genes that occurs when a given population experiences a sudden expansion caused by in-migration of outsiders from another population of the species.

genetic drift Random changes in gene frequencies from one generation to the next caused by a sudden reduction in population size as a result of disaster, disease, or the out-migration of a small subgroup from a larger population.

IN THEIR OWN WORDS

DNA Tests Find Branches but Few Roots

The ambiguities surrounding the molecularization of race in biomedicine also show up in some genetics researchers' efforts to use DNA testing to trace "racial" ancestry. In 2007, journalist Ron Nixon reported in the New York Times *about the growth of private companies that will trace genetic ancestry for their clients, sometimes for a hefty fee.*

Henry Louis Gates, Jr., whose PBS special *African American Lives* explores the ancestry of famous African Americans using DNA testing, has done more than anyone to help popularize such tests and companies that offer them. But recently this Harvard professor has become one of the industry's critics.

Mr. Gates says his concerns date back to 2000, when a company told him his maternal ancestry could most likely be traced back to Egypt, probably to the Nubian ethnic group. Five years later, however, a test by a second company startled him. It concluded that his maternal ancestors were not Nubian or even African, but most likely European.

Why the completely different results? Mr. Gates said that the first company never told him he had multiple genetic matches, most of them in Europe. "They told me what they thought I wanted to hear," Mr. Gates said.

An estimated 460,000 people have taken genetic tests to determine their ancestry or to expand their known family trees, according to *Science* magazine. Census records, birth and death certificates, ship manifests, slave narratives, and other documents have become easier to find through the Internet, making the hunt for family history less daunting than in years past.

Yet for many, the paper or digital trail eventually ends. And for those who have reached that point, genetic DNA tests may help to provide the final piece of the puzzle.

The expectations and reasons for taking the test vary. For some, the test allows them to reconnect with African ancestors after centuries of slavery wiped out links between African Americans and their forebears. Others want to see if they have links to historical figures like Genghis Khan or Marie Antoinette. For still others, it's an attempt to fill gaps in family histories and find distant cousins they might not otherwise have known.

The demand has spawned an industry. Almost two dozen companies now offer such services, up from just two or three only 6 years ago. The field is so hot that private equity investors have moved in: Spectrum Equity Investors recently bought Ancestry.com, an online genealogical site, for about $300 million shortly after the site added genetic testing as a service.

But as the number of test takers and companies has grown, so has the number of scientists or scholars like Mr. Gates who have questioned assertions that companies make about their tests. One of the most controversial issues is the ability of the tests to determine the country or the ethnic group of origin for African Americans or Native Americans.

Mr. Gates, director of the W. E. B. Dubois Institute for African and African American Research at Harvard, said his experience and similar stories from others have prompted him to enter the field.

Mr. Gates recently teamed up with Family Tree DNA, a DNA testing and genealogy firm in Houston, to provide genetic testing and genealogy work for African Americans. The new venture is called AfricanDNA.

"What we hope to do is combine this with genealogical and other records to try to help people discover their roots," he said. "The limitations of current genetic DNA tests mean you can't rely on this alone to tell you anything. We hope to bring a little order to the field."

In an editorial in *Science* magazine in October [2007], a number of scientists and scholars said companies might not be fully explaining the limitations of genetic testing, or what results actually mean.

The authors said that limited information in the databases used to compare DNA results might lead people to draw the wrong conclusions or to misinterpret results. The tests trace only a few of a customer's ancestors and cannot tell exactly where ancestors might have lived, or the specific ethnic group to which they might have belonged. And the databases of many companies are not only small—they're also proprietary, making it hard to verify results.

"My concern is that the marketing is coming before the science," said Troy Duster, a professor of sociology at New York University who was an advisor on the Human Genome Project and an author of the *Science* editorial.

"People are making life-changing decisions based on these tests and may not be aware of the limitations," he added. "While I don't think any of the companies are deliberately misleading customers, they may have a financial incentive to tell people what they want to hear."

(continued on next page)

IN THEIR OWN WORDS

DNA Tests Find Branches but Few Roots *Continued*

Bennett Greenspan, founder and president of Family Tree DNA, said his company sometimes has to tell clients just the opposite. "We'll have people who may think that they have a certain type of ancestry and we'll tell them based on the test they are not," he said. "I can only tell them what the tests show, nothing more. And sometimes it's not what they want to hear."

Nixon explains that the tests can analyze either mitochondrial DNA, which is passed on only by females to their male and female offspring, or the Y chromosome, which is passed on only to males. He reviews the practices of several different companies and the mixed experiences of different customers. He then continues: Even some early proponents of DNA testing for ancestry have doubts about how useful the tests are.

Bert Ely, a geneticist at the University of South Carolina, was a cofounder of the African American DNA Roots Project in 2000, hoping to use DNA tests as a way to find connections between African Americans and ethnic groups in Africa.

"I originally thought that the mitochondrial DNA test might be a good way for African Americans to trace their country of origin," Mr. Ely said. "Now I'm coming to the opposite conclusion."

[Mr. Ely] matched the DNA sequences of 170 African Americans against those of 3,725 people living in Africa. He found that most African Americans had genetic similarities to numerous ethnic groups in Africa, making it impossible to match African Americans with a single ethnic group, as some companies assert they can do.

Mr. Ely also published a paper in which he tried to determine whether the country of origin of native Africans could be found by using mitochondrial DNA tests. Several of the Africans in the study matched multiple ethnic groups. For example, DNA results for a person from Ghana provided genetic matches with people in 20 African countries. . . .

It's not that the tests are wrong, scientists say. Most companies use the same statistical methods and, in some cases, the same labs to extract DNA from samples. But even the largest databases have only a few thousand records in them, and some areas and populations are sampled more than others. Most companies get data from information published in publicly available research papers; few collect samples themselves. Scientists emphasize that much of this data was gathered for other purposes and was never intended to be used for personal genealogical testing.

For their part, testing companies say they continually update their databases to get a larger number of samples.

As part of the reporting for this article, I [Mr. Nixon] decided to submit my own samples for a mitochondrial DNA test. *Roots* had left an impression on me. . . . Like most African Americans, I longed to know where I came from. Could tests tell me? . . .

At a 2007 reunion for descendants of slaves of James Madison, Dr. Bruce Jackson, director of the African American DNA Roots Project at the University of Massachusetts, collects a DNA sample from Dr. Gladys Marie Fry of Washington, DC.

IN THEIR OWN WORDS

Six weeks after I submitted the first samples, the results started to roll in. Every company told me that my mother's female ancestors were all African. But after that things got murky.

African Ancestry said my DNA was a match with that of the Mende and Kru people from Liberia. Family Tree DNA's database showed a match with one person who was Mende. But my DNA also matched that of several other groups, like the Songhai in Mali, and various ethnic groups in Mozambique and Angola. Other peoples cited were the Fula-Fula (also known as the Fulani), who live in eight African nations, and the Bambara, who are primarily in Mali.

Why so many? "We try to be brutally honest and give you everything the test results show," said Mr. Greenspan of Family Tree DNA. "If there are multiple matches, we're going to show you that."

Mr. Ely's African American DNA Roots Project, which examined DNA sequences that other companies provided to me, confirmed many matches from Family Tree DNA and African Ancestry, but added additional ethnic groups. DNA Tribes, whose test shows DNA results from a combination of genetic material from both parents, added even more ethnic matches.

I once thought that my ancestors, like those of most African Americans, would have come from West Africa. But some of the results showed links to regions that I had thought weren't engaged in the slave trade with the United States—like Mozambique. But then a search of the TransAtlantic Slave Trade database, which was compiled from slave ship records, showed that some Africans from Mozambique did indeed end up in the United States. So maybe the Mozambique results were possible.

The companies also offered technical support to understand the results, and I spent considerable time trying to make sense of them. I learned a lot about how they reached conclusions, but not much about where I or my ancestors ultimately came from.

"What this all means is that you can't take one of these tests and go off and say you're this and that," Mr. Gates said. "Somewhere down the road, the results could change and you might have another group of people who might also be your genetic cousins."

Sandra Jamison contributed reporting.

Source: *New York Times*, Sunday, November 25, 2007, BU 1,7.

> cholera, plague, and measles, which swept through continents, undoubtedly contributed to the shaping of the genetic landscape. (Mielke et al. 2011, 105–06)

Several evolutionary processes may affect a population at the same time. For example, a rare, helpful allele (say, one that increased resistance to a disease like malaria) might appear in a population through mutation. If malaria were an environmental threat to that population, we would expect natural selection to increase the frequency of this new allele. But suppose a natural disaster like an earthquake struck the population and many people died. If the new allele were still very rare, it might be completely lost if its few carriers were among those who perished (genetic drift). Alternatively, the frequency of a harmful new allele might increase in subsequent generations if its carriers survived such a disaster and if they introduced the new allele into a larger population through inbreeding (gene flow). Niche construction could also be implicated if, for example, gene flow were enabled or intensified as a result of persisting, environment-modifying activities of the populations exchanging genes.

Measuring the interaction among these evolutionary processes allows population geneticists to predict the probable effects of inbreeding and outbreeding on a population's gene pool. Inbreeding tends to increase the proportion of homozygous combinations of alleles already present in a population. If some of these alleles are harmful in a double dose, inbreeding increases the probability that a double dose will occur in future generations and thus decrease fitness. If helpful combinations of alleles occur in an inbreeding population, their proportions can increase in a similar way.

At the same time, inbreeding over several generations tends to reduce genetic variation. Natural selection on genes has a better chance of shaping organisms to changed environments if it has a wider range of genetic variation to act on. Perhaps for this reason, mating with individuals from outgroups is widely observed in the animal kingdom. Monkeys and apes, for example, regularly transfer into a new social group before they begin to reproduce (Figure 5.1). Human beings ordinarily do the same thing, except that our reproductive practices are shaped by culture; people in different societies draw the boundaries around in-groups and out-groups differently. In one society, the children of brothers and sisters may be considered members of the same family and, thus, off

FIGURE 5.1 Monkeys and apes regularly transfer into a new social group before they reproduce.

limits for marriage; in another, they may be considered members of different "families" and, thus, ideal marriage partners. However, cultural rules forbidding *incest*, or sexual relations with close kin, do not always succeed in preventing such relations from occurring.

Table 5.2 summarizes the effects of the four standard evolutionary processes on gene frequencies within and between populations.

Microevolution and Patterns of Human Variation

Gene Flow As we have seen, phenotypic variation in different human populations does not require different alleles for different populations; rather, the variation we find mostly involves differences in the proportions of the same sets of alleles common to the human species as a whole. Therefore, genetic relationships between interbreeding human groups are best understood in terms of gene flow between superficially distinct populations whose gene pools already overlap considerably. For example, we know that individuals from European and African populations have interbred considerably since Europeans brought the first Africans to the New World as slaves. Similar processes have mixed the genes of these and other in-migrating populations with the genes of indigenous American populations. These are examples of gene flow among populations of a single species that had experienced relative isolation in the past but that continued to exchange enough genes often enough with neighboring populations to prevent speciation.

Accidents of geography and history had allowed for relative isolation between these populations prior to the European voyages of exploration in the fifteenth century. From the fifteenth century on, similar chance factors brought them together. Moreover, the way in which reproductive isolation ended was powerfully shaped by the social and cultural forces that brought Europeans to the New World in the first place, structured their relationships with the indigenous peoples, and led them to enslave Africans. Similar cultural forces continue to affect the degree to which different human populations in the Americas remain reproductively isolated or exchange genes with other populations.

TABLE 5.2 Effects of the Four Evolutionary Processes on Variation within and between Populations

EVOLUTIONARY PROCESS	VARIATION WITHIN POPULATIONS	VARIATION BETWEEN POPULATIONS
Mutation	Increases	Increases
Gene flow	Increases	Decreases
Genetic drift	Decreases	Increases
Natural selection	Increases *or* decreases	Increases *or* decreases

Genetic Drift One kind of genetic drift, the founder effect, occurs when a small subgroup of a larger population becomes isolated for some reason, taking with it unrepresentative proportions of the alleles from the larger population's gene pool. One of numerous examples of genetic drift that have occurred in human history began early in the nineteenth century when British soldiers occupied the island of Tristan da Cunha in the Atlantic Ocean. Eventually, the soldiers withdrew, leaving only a single married couple who were later joined by a few other settlers. Throughout the nineteenth century, the population of Tristan da Cunha never grew much beyond 100 individuals. This tiny population was later reduced even more, once in the late 1850s by the out-migration of 70 inhabitants and again in 1885 by the drowning of all but 4 adult males (only one of whom contributed genes to the next generation). Over the twentieth century, the population grew to as many as 270 people, all of whom owe an enormous proportion of their genes to a very few individuals. It was calculated that nearly a third of those living on the island in 1961 had genes contributed by just 2 members of the original founding population (Roberts 1968; Underwood 1979).

Mutation and Natural Selection Mutation is responsible for variant alleles that may be present at a single locus. Some of these mutant alleles are mobilized during development to help produce specific physical traits. When a trait proves helpful, evolutionary theory predicts that the frequency of the alleles involved in its production will be increased by natural selection. Perhaps the most famous instance of microevolution of such a trait by means of natural selection concerns a variant of hemoglobin, one of the proteins in red blood cells.

In many human populations, only one allele—hemoglobin A (*HbA*)—is present. In other populations, however, mutant forms of hemoglobin A may also be present. One such mutant allele, known as *HbS*, alters the structure of red blood cells, distorting them into a characteristic sickle shape and reducing their ability to carry oxygen (Figure 5.2). When individuals inherit the *HbS* allele from both parents, they develop sickle-cell anemia. About 85% of those with the *HbS*/*HbS* genotype do not survive to adulthood and, hence, do not reproduce. Although many people in the United States think that sickle-cell anemia affects only people with ancestors who came from Africa, in fact many people in India, Saudi Arabia, and Mediterranean countries such as Turkey, Greece, and Italy also suffer from the disease.

Because the *HbS* allele seems to be harmful, we would expect it to be eliminated through natural selection. But in some populations of the world, it has a frequency of up to 20% in the gene pool. Why should that be? Geneticists might have concluded that this high

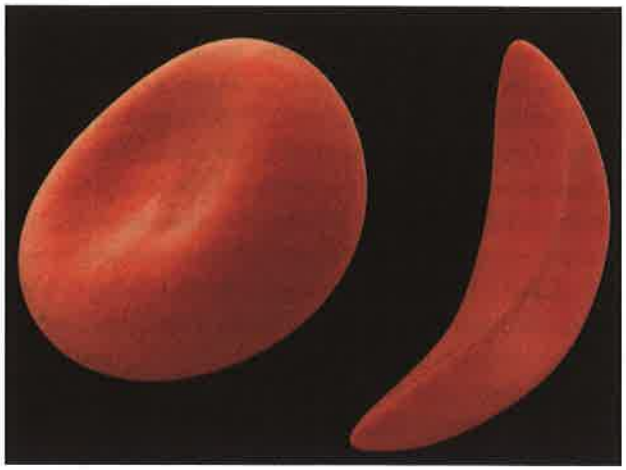

FIGURE 5.2 Normal red blood cells are easily distinguished from the distorted, "sickled" red blood cells. Sickled red blood cells carry less oxygen than do normal red blood cells, but they resist malarial parasites more successfully.

frequency was the result of genetic drift if it were not for the fact that the areas with a high frequency of *HbS* are also areas where the mosquito-borne malaria parasite is common. There is, in fact, a connection. People exposed to malaria have a better chance of resisting the parasite if their hemoglobin genotype is *HbA*/*HbS* rather than the normal *HbA*/*HbA*. This is an example of what geneticists call a "balanced polymorphism," in which the heterozygous genotype is fitter than either of the homozygous genotypes. In Mendelian terms, we would say that the *HbA* and *HbS* alleles are codominant, with the result that a single *HbS* allele changes the structure of red blood cells enough to inhibit malarial parasites but not enough to cause sickle-cell anemia.

The rise of malarial infection in human beings appears to have begun only a few thousand years ago (Livingstone 1958). Before that time, the people who lived where malaria is now found gathered and hunted wild foods for a living. This way of life kept forests intact, leaving few open areas where water could collect and malaria-carrying mosquitoes could breed in large numbers. As these inhabitants began to cultivate plants for food, however, they needed to clear large tracts of forest for their fields, creating large open spaces where rainwater could collect in stagnant pools, providing ideal breeding conditions for mosquitoes. And as the population of cultivators grew, so grew the number of hosts for the malaria parasite.

If the *HbS* allele first appeared in the populations of gatherers and hunters, it probably had a low frequency. But once cultivation began, land was cleared, water accumulated in open spaces, and the number of malaria-infested mosquitoes increased, selection pressures changed. At that point, individuals with the *HbA*/*HbS* genotype were fitter because they had a greater probability

of surviving and reproducing than individuals with *HbA/HbA* or *HbS/HbS*. As a result, the frequency of *HbS* increased in the population, despite the fact that in a double dose it was generally lethal. This example also illustrates the way niche construction can reshape the selection pressures that a population experiences. In this case, a switch from one pattern of human food getting to another created new niches for humans, mosquitoes, and malaria parasites, simultaneously reshaping the selection pressures experienced by all three populations (Odling-Smee et al. 2003). Indeed, niche construction may also be implicated in discussions of gene flow and genetic drift since in both cases activities undertaken by particular human populations may alter their respective niches in persistent ways, thereby altering the selection pressures that each population subsequently experiences.

Adaptation and Human Variation

One of the breakthroughs of modern genetics was the discovery of *gene interaction*. That is, a single gene may contribute to the production of more than one phenotypic feature (*pleiotropy*), and many genes regularly combine forces (*polygeny*), helping to produce a single phenotypic feature. Pleiotropy and polygeny help explain how it is that genes, which are discrete, could influence phenotypic traits such as body size or skin color, which show continuous gradations. Traits that are the product of multiple genes offer multiple and varied opportunities for natural selection to shape phenotypic traits in ways that are adaptive for the organisms in which they are found.

In discussions of gene action, biologists commonly distinguish between genes of major effect and polygenes of intermediate or minor effect. A *gene of major effect* is a gene at one locus whose expression has a critical effect on the phenotype. The *HbS* allele that produces the sickling trait in red blood cells is an example of a gene of major effect. But phenotypic traits that depend on one or a few genes of major effect are rare. The evolution of a phenotypic trait may begin with selection on genes of major effect, but the products of such genes may be pleiotropic, producing adaptive as well as harmful consequences for the organism. Further selection on multiple *polygenes of intermediate or minor effect* that also affect the trait, however, may modify or eliminate those harmful consequences (West-Eberhard 2003, 101–04). Finally, because gene expression does not take place in an environmental vacuum, many phenotypic traits in organisms are even more finely tuned for their adaptive functions by inputs from environmental factors such as nutrients, temperature, humidity, altitude, or day length. Human phenotypic traits such as body size or skin color, for example, are the outcome of complex interactions among multiple gene products and environmental influences throughout the life cycle.

Many students of human genetics have devoted attention to the way natural selection may mold complex human phenotypic traits, better adapting human populations to their specific environments. More recently, developmental biologists have been able to show how the responsiveness of organisms to their environments also contributes to the abilities of those organisms to adapt to their environments. A fertilized human egg (or zygote) has its own phenotype, and the zygote's phenotype can respond to environmental influences—such as those encountered in a woman's uterus—*even before its own genes are active*. This responsiveness is called **phenotypic plasticity**: "the ability of an organism to react to an environmental input with a change in form, state, movement, or rate of activity" (West-Eberhard 2003, 35). Because all living organisms exhibit phenotypic plasticity, it is *incorrect* to assume that genes "direct" the development of organisms or "determine" the production of phenotypic traits. Indeed, much of the "action" that goes into producing adult organisms with distinctive phenotypes goes on during development (Figure 5.3).

It is important to stress that acknowledging the phenotypic plasticity of organisms has nothing to do with Lamarckian ideas of use and disuse and the inheritance of acquired characteristics, neither of which is accepted by modern evolutionary biologists. As West-Eberhard (2003) points out,

> There is no hint of direct (Lamarckian) influence of environment on genome in this scheme—it is entirely consistent with conventional genetics and inheritance. By the view adopted here, evolutionary change depends upon the genetic component of phenotypic variation screened by selection, whether phenotypic variants are genetically or environmentally induced. It is the genetic *variation* in a response (to mutation or environment) that produces a response to selection and cross-generational, cumulative change in the gene pool. . . . (29)

Some of the most exciting work in evolutionary biology today involves linking new understandings about developmental influences on phenotypes with understandings of traditional evolutionary processes like mutation, gene flow, genetic drift, and natural selection (Oyama et al. 2001; Gould 2002; West-Eberhard 2003).

As we saw earlier, **adaptation** as a *process* refers to the mutual shaping of organisms and their environments.

phenotypic plasticity Physiological flexibility that allows organisms to respond to environmental stresses, such as temperature changes.

adaptation (1) The mutual shaping of organisms and their environments. (2) The shaping of useful features of an organism by natural selection for the function they now perform (see Chapter 2).

FIGURE 5.3 Changes in environment can have major effects on phenotype. Generational differences in height are often connected with changes in diet.

However, the term *adaptation* can also be used to refer to the *phenotypic traits* that are the outcome of adaptive processes. As Zaneta Thayer and Amy Non explain,

> Humans must adapt to multiple timescales of evolutionary change. . . . Very stable environmental trends can be accommodated through natural selection, the slowest mechanism of genetic change. Immediate, minute-to-minute fluctuations in the environment, such as changes in temperature, are accommodated via homeostatic processes, including changes in blood flow. At a more intermediate level on the timescale of months to years, organisms adapt to environmental conditions via developmental plasticity. (2015, 727–28)

The sickling trait in hemoglobin described in the previous section is a classic example of a genetic adaptation produced by natural selection, in response to environmental conditions that stabilized in regions where tropical forests were cleared for farming several thousand years ago, creating expanded breeding grounds for mosquitos carrying the malaria parasites and thereby increasing human exposure to the parasites. In this case, the form of the hemoglobin molecule is the phenotypic product of a single-locus gene of major effect. Most human phenotypic traits, however, are the product of pleiotropy, polygeny, and inputs from the environment.

The shivering response in humans illustrates adaptation to the brief timescale of minute-to-minute fluctuations in the environment, a response in human beings sometimes called "short-term **acclimatization**." Human beings are warm-blooded organisms who need to maintain a constant internal body temperature to function properly. When the surrounding temperature drops, however, and threatens to cool our internal organs below this threshold temperature (roughly 98.6° Fahrenheit), this temperature drop triggers a twitching response in the muscles that surround our vital organs as a way of generating heat. If we are able to increase our body temperature above the threshold—by going indoors, putting on clothes, or moving closer to the fire—the shivering stops.

Other forms of acclimatization take shape over more intermediate timescales. Such adaptations emerge over the course of many months or years, as human phenotypic plasticity is shaped by inputs from the particular environments within which individuals develop. That is, physiological or morphological changes resulting from developmental plasticity are not a consequence of genetic variation. Put another way, "developmental plasticity allows one genotype to give rise to multiple phenotypes in response to variation in the environment in which an organism develops" (Thayer and Non 2015, 728). For example, some environments in which human populations live, such as the highlands of the Andes Mountains in South America, are characterized by *hypoxia*; that is, less oxygen is available to breathe than at lower altitudes. Studies have shown that people who grow up in high altitudes adapt to lower oxygen levels by developing greater chest dimensions and lung capacities than do people living at low attitudes. These changes—sometimes called "developmental acclimatization"—are a consequence of human phenotypic plasticity and occur when the human body is challenged by a low level of oxygen in the environment. Studies have shown that individuals who were not born in such an environment increased in chest dimensions and lung capacity the longer they lived in such an environment and the younger they were when they moved there (Greska 1990).

One kind of biological mechanism that seems to allow environmental stresses to mold phenotypic plasticity are called *epigenetic marks*. Epigenetic marks are "chemical modifications to DNA that are associated with changes in the way genes are expressed or turned on, and are essential for normal development in mammals" (Thayer and Non 2015, 725). One kind of

acclimatization A change in the way the body functions in response to physical stress.

epigenetic mark is called *DNA methylation*. Methylation chemically modifies a portion of the DNA molecule in a way that reduces the level of gene product produced by a particular gene, "and its influence is dependent on the genetic context within which the methylation occurs" (2015, 723). Unlike DNA itself, methylation and other epigenetic marks "are sensitive to environmental exposures throughout growth and development, [and] they represent a prime candidate mechanism underlying developmental plasticity" (2015, 728). It is important to emphasize, however, that epigenetic modifications are fully compatible with the Darwinian foundations of contemporary evolutionary theory. This is because epigenetic marks "can only occur in interaction with the underlying genetic variation that is available" (2015, 725).

Skin Color Skin color is a highly visible, complex, continuous phenotypic trait in human populations. Variation in skin color seems to be the product of a few genes of major effect, additional polygenes of intermediate or minor effect, and input from the environment. As Nina Jablonski (2004) writes, "determination of the relative roles of variant genes and varying environments has proven extremely challenging" (613), and it is not clear how many alleles are involved or whether identical genes are responsible for the dark skin of apparently unrelated human populations (Marks 1995, 167–68). Biological anthropologists agree that skin color is adaptive and related to the degree of ultraviolet radiation (UVR) that human populations have experienced in particular regions of the globe.

It is important to emphasize that "similar skin colors have evolved independently in human populations inhabiting similar environments," making skin color "useless as a marker for membership in a unique group or 'race'" (Jablonski 2004, 615). Indeed, some of the most striking features of human skin are clearly consequences of developmental and phenotypic plasticity: variations in skin thickness are a function of age and history of sun exposure; the outer layers of the skin in darkly pigmented or heavily tanned people have more, and more compact, cell layers, making the skin more effective as a barrier to sun damage. The overall intensity of skin color is thus determined by a combination of morphological, physiological, environmental, and developmental factors. When the intricate articulation of these factors is destabilized, the outcome can be anomalous skin conditions such as *albinism* (an absence of pigmentation), abnormally intense pigmentation, or a patchy spotting of light and dark skin (Jablonski 2004, 590).

Human skin color exhibits clinal variation, with average pigmentation growing gradually lighter in populations that live closer to the poles (Figure 5.4). The pigments in human skin (melanins) protect the skin against sunburn by absorbing and scattering UVR and by protecting DNA from damage that can lead to cancer (Jablonski 2004, 590). Of course, as humans we risk sun damage to the skin because we do not grow fur coats, like our closest primate relatives. Dark fur coats can actually protect primates from tropical heat by absorbing short-wave radiation (UVA) near the surface of the coat and reflecting much long-wave radiation (UVB) away before it reaches

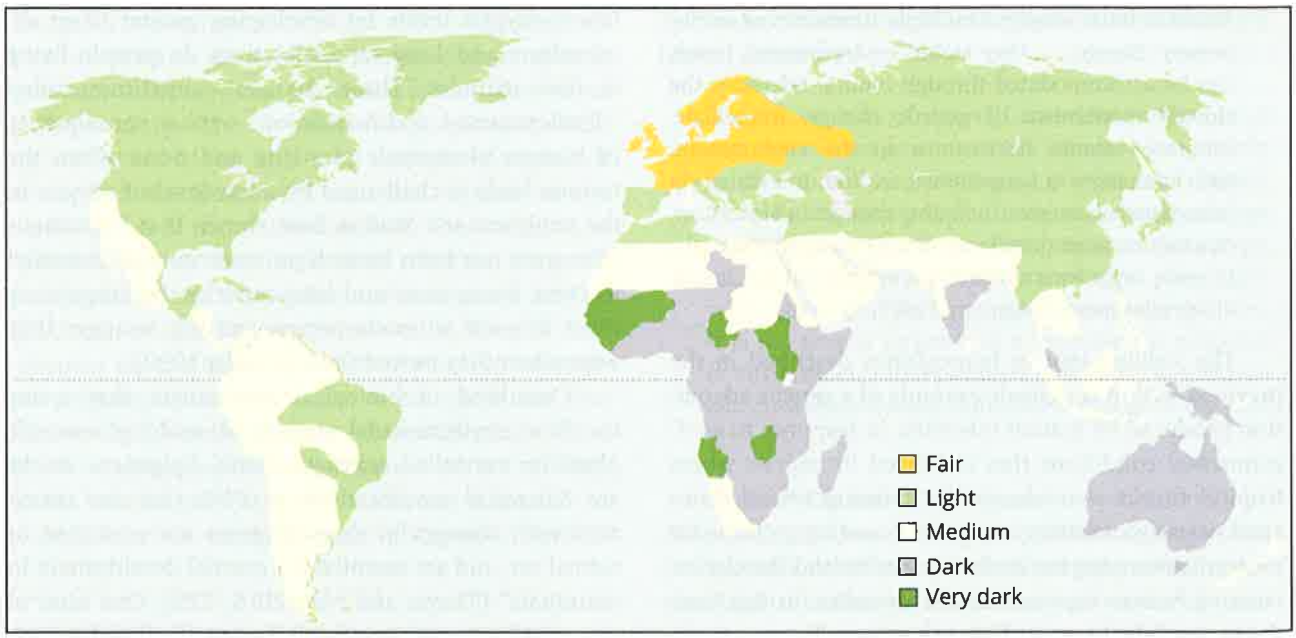

FIGURE 5.4 When the unexposed skin of indigenous peoples is measured and mapped according to the degree of pigmentation, skin shades tend to grow progressively lighter the farther one moves from the equator.

the skin. These advantages of fur, however, are reduced if the fur is wet with sweat, which can happen if the temperature rises or the organism's activity level increases. Under these conditions, "thermal sweating as a method of cooling becomes more important" and it is "greatly facilitated by the loss of body hair" (Jablonski 2004, 599). It is now hypothesized that the last common ancestor of humans and chimpanzees probably had light skin covered with dark hair, like other Old World primates. However, the loss of hair created new selection pressures in favor of increasingly darker skin, such that by 1.2 million years ago (mya), early members of the genus *Homo* would have had darkly pigmented skin (Rogers et al. 2004). In addition, contemporary human populations all seem to show sexual dimorphism in skin color, "with females being consistently lighter than males in all populations studied" (Jablonski and Chaplin 2000; Jablonski 2004, 601).

Exposure of human skin to solar radiation has complex and contradictory consequences. Too much sunlight produces sunburn, and UVB destroys a B vitamin, folic acid, which is a crucial factor in healthy cell division. At the same time, solar radiation also has positive consequences: UVA stimulates the synthesis of vitamin D in human skin. Vitamin D is crucial for healthy bone development and other cellular processes. According to Jablonski and Chaplin (2000), these selective pressures have produced two opposing clines of skin pigmentation. The first cline grades from dark skin at the equator to light skin at the poles and is an adaptive protection against sun damage. The second cline grades from light pigmentation at the poles to dark pigmentation at the equator and is an adaptive response favoring vitamin D production. In the middle of these two clines, they argue, natural selection favored populations with enhanced phenotypic plasticity who could tan more easily during hot, sunny seasons but easily lose their tans in seasons when temperature and sunlight levels decreased.

Jablonski (2004) concludes that "the longer wavelengths of UVR . . . have been the most important agents of natural selection in connection with the evolution of skin pigmentation" (604). At the same time, because people have always migrated, different populations vary in the numbers of generations exposed to the selective pressures of any single regime of solar radiation. Human cultural practices (wearing clothes, using sun block, staying indoors) have shaped the levels of pigmentation and levels of vitamin D production in particular individuals or populations. Gene flow following the interbreeding of human populations with different selective histories would further complicate the relationship between the skin colors of their offspring and selection pressures imposed by local levels of solar radiation.

Many of these factors may explain why the skin colors of the native people of South America are lighter than those of native populations in Asia or Europe who live at similar latitudes. Most anthropologists estimate these populations migrated from the Old World perhaps 10,000–15,000 years ago, which means they have had far less time to experience the selective pressures associated with local solar radiation levels anywhere on the continent. In addition, these migrants were modern humans with many cultural adaptations to help them modify the negative effects of solar radiation, including both protective clothing and a vitamin D–rich diet. Obtaining vitamin D from food rather than sunlight has thus altered selection pressures that otherwise would have favored lighter skin. Thus, the darker skin pigmentation of circumpolar peoples may be the consequence of selection pressures for darker skin as a protection against solar radiation reflected from snow and ice (Jablonski 2004, 612).

Intelligence Intelligence may be the most striking attribute of human beings. However, attempts to define and measure "intelligence" have a long history of controversy. Is intelligence a single, general, unitary "thing" that people have more or less of? If not, what attributes and skills ought to count? Psychologist Howard Gardner (2000) points out that "Every society features its ideal human being" (1). In his view, "the intelligent person" in modern Western societies has been exemplified by individuals who could do well at formal schooling and succeed in commerce. It is perhaps not surprising, then, that tests developed in Western societies purporting to measure individuals' intelligence quotient (IQ) traditionally have equated high scores on verbal and mathematical reasoning with high intelligence.

But these are not the only areas in which humans display differing levels of ability or skill. Gardner, for example, has long argued that in addition to linguistic and logicomathematical intelligence, human beings possess different types of intelligence, including bodily–kinesthetic intelligence (displayed by exceptional athletes and dancers), interpersonal or intrapersonal intelligence (displayed by individuals with exceptional understanding of social relations or their own psyches), musical intelligence, spatial intelligence, and naturalist intelligence (which attunes us to plants and animals in the world around us). In Gardner's view, these types of intelligence can probably be enhanced in all individuals, given the right kind of environmental support. Indeed, even linguistic intelligence and logicomathematical intelligence require the proper environmental support—long-term training and practice in rich cultural settings—to produce the highest levels of achievement.

Because the definition of *intelligence* is so controversial and because not all forms of intelligence are equally

rewarded in the United States, great controversy results when attempts to measure intelligence are applied not only to individuals but also to entire social groups, defined on the basis of gender, class, or "race." The former president of Harvard University was subjected to strong criticism when he acknowledged that fewer women than men become scientists and suggested, in the face of massive evidence to the contrary, that perhaps this meant that women simply had less "intrinsic aptitude" for science and engineering than men (http://www.harvard.edu/president/speeches/summers_2005/nber.php). Controversies have been as great or greater when ideas about intelligence have been linked to ideas about race. In the United States, for example, people tend to assign each other to "races" on the basis of phenotypic criteria like skin color. As we have seen, such "races" are then often regarded as different natural kinds, each sharing its own biological essence. From this assumption, it is a short step to conclude that differences between races must include differences in intelligence. Some scientists have devised IQ tests that they claim can measure intelligence, the results of such testing repeatedly showing that the average IQ score for African Americans is below that of European Americans, which is below that of Asian Americans.

Do IQ scores show that racial differences in intelligence are clear-cut and genetically determined? They do not. First, the idea that races are natural kinds assumes that racial boundaries are clear and that traits essential to racial identity (e.g., skin color) are discrete and nonoverlapping. However, as we noted above, skin color is a continuously varying phenotypic trait, both among members of the so-called racial groups and the boundaries of those groups. Particular shades of skin color cannot be assigned exclusively to particular socially defined races, nor can they be used to infer any other so-called racial attribute, such as intelligence or athletic ability.

Second, it is far from clear that there is a single, accurately measurable substance called "intelligence" that some people have more of than others. Performing well on paper-and-pencil tests tells us nothing about problem-solving skills and creativity, which might equally deserve to be called "intelligence." Third, even if intelligence is such a measurable substance, we do not know that IQ tests actually measure it. People can score badly on an IQ test for many reasons that have nothing to do with intelligence: they may be hungry or ill or anxious, for example. When different social groups within a society consistently score differently as groups, however, we may suspect that the test itself is to blame. Arguing that IQ tests measure cultural knowledge, not intelligence, many critics contend that the vocabulary items used on most IQ tests reflect experiences typical of European American middle-class culture. People from different cultural backgrounds do poorly on the test because their experiences have not provided them with the knowledge being tested.

Many studies have shown that how an individual will do on an IQ test is more accurately predicted by social class and educational background than by "race." When African Americans and European Americans are matched in terms of these factors, the differences in average IQ scores disappear (Molnar 1992). Similarly, African American children adopted by middle-class European American parents scored an average of twelve points higher on IQ tests than did African American children who remained in the lower-income communities from which the adoptees had come (Woodward 1992). Studies like these demonstrate repeatedly that IQ scores are not phenotypic traits uniquely determined by genes but that they are powerfully affected by a range of environmental factors over the course of the human life cycle. Or as Greg Downey and Daniel Lende put it, "humans' capacity for thought and meaning making emerges equally from social and individual sources, built of public symbol, evolutionary endowment, social scaffolding, and private neurological achievements" (2012, 23–24).

Phenotype, Environment, and Culture

In recent years, many evolutionary biologists and biological anthropologists have recognized that trying to attribute every phenotypic trait of an organism to adaptation is problematic. Sometimes an adaptive explanation seems transparently obvious, as with body shape in fish and whales or wing shape in bats and birds, which equips these animals for efficient movement through water and air. Other times, adaptive explanations are less obvious, or even contrived. As we saw in Chapter 2, the wings of contemporary insects are better understood as an exaptation, when appendages that evolved as an adaptation to one set of selective pressures began at some point to serve an entirely different function.

In other words, the trait an organism possesses today may not be the direct result of adaptation but, instead, may be the byproduct of some other feature that was being shaped by natural selection. It may also be the consequence of random effects. Jonathan Marks (1995) has observed, for example, that anthropologists have tried, without notable success, to offer adaptive explanations for the large, protruding brow ridges found in populations of human ancestors. He suggests that brow ridges might well have appeared "for no reason at all—simply as a passive consequence of growing a fairly large face attached to a skull of a small frontal region" (Marks 1995, 190).

We must also remember that phenotypes are shaped by environment as well as by genes. For example, some have argued that slow growth in height, weight, and body composition and delayed onset of adolescence among

Guatemalan Mayan children constitute a genetic adaptation to a harsh natural environment. However, by comparing measurements of these traits in populations of Mayans who migrated to the United States with those in Guatemala, Barry Bogin was able to disprove these claims because "the United States–living Maya are significantly taller, heavier and carry more fat and muscle mass than Mayan children in Guatemala" (Bogin 1995, 65). Similarly, other biological anthropologists working in the Andean highlands have refuted the hypothesis that hypoxia is responsible for poor growth among some indigenous populations (Leonard et al. 1990; de Meer et al. 1993). They point out that the genetic explanation fails to consider the effects on growth of poverty and political marginalization.

At the beginning of the twenty-first century, it has become fashionable for many writers, particularly in the popular media, to treat genes as the ultimate explanation for all features of the human phenotype. Given the great achievements by molecular biology that followed the discovery of the structure of the DNA molecule, this enthusiasm is perhaps understandable. But discussions of human adaptive patterns that invoke natural selection on genetic variation alone are extremely unsatisfactory. For one thing, they mischaracterize the role genes play in living organisms. Speaking as if there were a separate gene "for" each identifiable phenotypic trait ignores pleiotropy and polygeny, as well as phenotypic plasticity. It also ignores the contribution of the other classic evolutionary processes of genetic drift and gene flow, as well as the influences of historical and cultural factors on human development (as in the case of the Mayan migrants). Researchers in the Human Genome Project originally expected that, given our phenotypic complexity, the human genome would contain at least 100,000 genes; today, we know that the actual number is more like 20,000, only twice as many as the roundworm *Caenorhabditis elegans*, one of the simplest organisms that exists (http://www.genome.gov/11007952). Clearly, the number of genes possessed by an organism is not coupled in any straightforward way to its phenotypic complexity.

The gene-centered approach gained considerable influence in anthropology after 1975 because of the widespread theoretical impact of a school of evolutionary thought called "sociobiology." Sociobiology attracted some anthropologists who proposed explanations of human adaptations based on sociobiological principles. Other anthropologists have been highly critical of sociobiology. However, after four decades, some proposals emerging from this debate have come a long way toward meeting the objections of sociobiology's original critics.

It is important to understand that much of this research is based on **formal models**. These models are "formal" because scientists use the tools of formal logic or mathematics to find answers to particular questions about the evolution of human behavior. For example, evolutionary psychologists typically assume that the psychological abilities possessed by modern human beings are adaptations that were shaped by specific environmental challenges early in our species' evolutionary history. They employ formal psychological tests on contemporary human subjects to demonstrate the presence of these abilities and then use logical deduction to "reverse engineer" from these contemporary abilities back to the hypothetical selective pressures that would have shaped these abilities. By contrast, scientists who study gene-culture coevolution, cultural group selection, or niche construction use mathematical formulas to predict outcomes of particular kinds of human interactions under different hypothesized conditions. Computers allow them to simulate, for example, what happens when certain behavioral patterns are repeated for many generations. The researchers then examine the reports of ethnographers or other social scientists to see if any of the outcomes produced by their mathematical calculations match the actual behavior patterns found in real human societies.

No beginning anthropology textbook can offer an in-depth introduction to formal modeling of human biological and cultural evolutionary processes (Table 5.3). But students should be aware of this dynamic and contentious field of research, in which anthropologists, biologists, ecologists, psychologists, and other scientists collaborate. Students should also be aware that many anthropologists—cultural anthropologists in particular—are highly critical of formal models, especially formal models of cultural evolution. They point out that formal modeling cannot work unless actual human interactions, which are messy and complex, are tidied up and simplified so that they can be represented by variables in mathematical equations. Reverse engineering has also been criticized for being overly reliant on logical deduction, rather than empirical evidence, in the generation of hypotheses about the human past. Critics argue that these approaches produce nothing more than cartoon versions of everyday life that often reveal systematic Western ethnocentric bias.

In our view, the perspective with the most promise is that of niche construction, which articulates in unusually clear language a point of view many anthropologists and others have held for a very long time. And they are not the only ones. As ecologist Richard Levins and biologist Richard Lewontin pointed out in 1985,

> [using] cultural mechanisms to control our own temperature has made it possible for our species to survive

formal models Mathematical formulas to predict outcomes of particular kinds of human interactions under different hypothesized conditions.

TABLE 5.3 Formal Models in the Study of Human Biological and Cultural Evolution

THEORETICAL PERSPECTIVE	KEY FEATURES
Sociobiology	• Defined by E. O. Wilson (1980), one of its founders, as "the systematic study of the biological basis of all social behavior" (322). Originally focused on explaining the evolution of *altruism*—the willingness to give up benefits for oneself to help someone else. Sociobiologists argued that altruism makes sense if we pay attention not to individuals but to the genes they carry. • Organisms share the most genes with their close relatives; therefore, sociobiologists hypothesize, natural selection will preserve altruistic behaviors if the altruists sacrifice themselves for close kin, a concept known as *kin selection*. Some anthropologists adopted the sociobiological approach to human societies, whereas others viewed sociobiology as a pernicious perspective that threatened to resurrect nineteenth-century racism.
Behavioral ecology	• A school of thought based on sociobiological reasoning that accepts the importance of natural selection on human adaptations, but rejects sociobiology's genetic determinism. Behavioral ecologists accept the view that human adaptations depend on cultural learning rather than on genetic control, but they insist that the cultural behavior human beings develop is closely circumscribed by the selection pressures imposed on us by the ecological features of the environments in which human populations have lived (see Cheverud 2004; Sussman and Garber 2004).
Evolutionary psychology	• Like earlier sociobiologists, evolutionary psychologists insist that human adaptations are phenotypes under close genetic control. Unlike earlier sociobiologists, however, evolutionary psychologists do not invoke natural selection on genes to explain human behavior patterns as adaptations to present-day conditions. Rather, they argue that natural selection on human genes was most significant millions of years ago, in the environment in which our ancestors lived when they were first evolving away from the other African apes (called the "environment of evolutionary adaptedness," or EEA). • Evolutionary psychologists argue that natural selection in the EEA produced a human brain consisting of a set of sealed-off "mental modules," each of which was designed by natural selection to solve a different adaptive problem (see Barkow et al. 1992).
Gene–culture coevolution	• An analysis of the origin and significance of culture in human evolution that is critical of standard sociobiological accounts. The version developed by Robert Boyd and Peter Richerson (1985) argues that human behavior is shaped by two inheritance systems, one genetic and one cultural. Cultural traits are passed on by learning, not via the chromosomes, but since these traits vary, are passed on from individual to individual, and confer differential fitness on those who use them, they can undergo natural selection (76). • The two inheritance systems are interconnected: human biological evolution creates the possibility for cultural creativity and learning, whereas human cultural traditions created the environment that allows human biological processes to continue, even as culture creates selection pressures of its own that shape human biological evolution. This is why the process is called gene-culture *coevolution* (see also Cavalli-Sforza and Feldman 1981, Durham, 1991).
Cultural group selection	• Sociobiologists argue that group selection cannot occur as the outcome of natural selection operating on genes unless group members are biological kin who share genes (see *kin selection*, above). If group members do not share genes, the good of the individual and the good of the group no longer coincide; this means that individuals who sacrificed themselves for other group members would take their "group selection" genes with them to the grave. • But if behaviors are shaped by cultural inheritance rather than genetic inheritance (as in gene–culture coevolution) this argument may not hold. When the forces of cultural learning are powerful enough, the fitness of an individual may come to depend on the behaviors of other individuals in a local group. This is known as cultural group selection. Once the forces of cultural transmission take hold, it is usually easier and cheaper to behave the way the group dictates than it is to strike out on one's own (D. S. Wilson 2002, Richerson and Boyd 2005).
Niche construction	• Odling-Smee, Laland, and Feldman (2003) argue that human evolution depends not just on our genetic heritage and our cultural heritage but also on an additional heritage of modified selection pressures that we pass on to our descendants in the form of a constructed niche. They use the concept of "artifact" to represent these environmental modifications: artifacts include birds' nests and rodents' burrows as well as human artifacts like clothing and furnaces. Odling-Smee et al. argue that their "triple-inheritance" theory offers a more satisfactory explanation of the evolutionary histories of organisms than do accounts focusing on genes and culture alone.

> in almost all climates, but it has also created new kinds of vulnerability. Our body temperature now depends on the price of clothing or fuel, whether we control our own furnaces or have them set by landlords, whether we work indoors or outdoors or leave places with stressful temperature regimes. . . . Thus our temperature regime is not a simple consequence of thermal needs but rather a consequence of social and economic conditions. (259)

Can We Predict the Future of Human Evolution?

Current arguments among evolutionary biologists illustrate their varied attempts to grasp the meaning of evolution. How we classify the natural world matters not only to scientists, who want to be sure their classifications match what they find when they go to nature, but also to nonscientists. How we make sense of evolution is important because people of all societies see a connection between the way they make sense of the natural world and the way they make sense of their own lives. Many people believe that human morality is, or ought to be, based on what is natural. For such people, evolutionary interpretations of nature can be threatening even if they portray a natural world that is orderly. If nature's order is dog-eat-dog and if human morality must be based on nature's order, then survival at any cost must be morally correct because it is "natural." This is clearly why many people found the more extreme claims of human sociobiology so repugnant. For those who want to root compassion and generosity in human nature, sociobiology offers a portrait of human nature in which such behavior has little or no value.

But perhaps the uncontrolled and uncontrollable pursuit of food and sex is no more natural in our species than sharing, compassion, and nonviolent resolution of differences. As we saw in Chapter 3, many primatologists have evidence to show that, most of the time, most apes and monkeys do not live by the "law of the jungle." The law of the jungle is not a law after all.

Human beings, like all living organisms, are subject to evolutionary processes. Like other organisms, our species shares a gene pool whose different combinations, together with environmental input over the course of a lifetime, produce a range of different human phenotypes that develop over their lifetimes, incorporating a certain range of adaptive responses. But we are not like other organisms in all respects, and this is what makes the study of human nature, human society, and the human past necessary. To adapt to our environments—to make a living and replace ourselves—we have options that do not exist for other organisms: cultural adaptations that are passed on by learning, even when there is no biological reproduction (see Figure 5.5).

The rich heritage of human culture is the source of much wisdom to guide us in our moral dealings with one another. The more we learn about biology, however, the more we realize that neither genotypes nor phenotypes nor environmental pressures provide obvious answers to our questions about how to live. If anything, "nature" offers us mixed messages about what is, or is not, likely to promote survival and reproduction. And in any case, with the development of culture, for good or for ill, human beings have long been concerned not only with survival and reproduction but also with what it takes to lead a meaningful life. Physical life and a meaningful life usually, but not always, go together. This paradox has been part of the human condition for millennia and is likely to remain with us long after our contemporary scientific debates have become history.

FIGURE 5.5 An individual may have high cultural fitness and no genetic fitness at all. Here, a religious teacher who is celibate (thereby reducing her genetic fitness to zero) passes cultural knowledge to a new generation of other people's offspring.

Chapter Summary

1. The neo-Darwinian evolutionary synthesis of the 1930s and 1940s combined Darwinian natural selection with Mendelian ideas about heredity. Neo-Darwinians studied populations of reproductively isolated species, concentrating on the population's gene pool, estimating the frequency of occurrence of different alleles of a particular gene, and predicting how those gene frequencies might be affected by different selection pressures.
2. Human population genetics has shown that different human populations from all over the world share basically the same range of genotypic variation, no matter how different they may appear phenotypically, reinforcing the position that the concept of "race" is biologically meaningless.
3. Natural selection, mutation, gene flow, and genetic drift are four evolutionary processes that can affect change in gene frequencies in a population over time. Sometimes one evolutionary process may work to increase the frequency of a particular allele while a different process is working to decrease its frequency. Inbreeding over several generations can be harmful because it decreases genetic variation and increases the probability that any alleles for deleterious traits will be inherited in a double dose, one from each parent.
4. Natural selection seems to have molded many complex human phenotypic traits, better adapting human populations to their environments. Anthropologists have studied how variation in traits such as skin color appear to have been shaped by natural selection. Anthropologists have also shown how variations in IQ test scores reflect variations in social class and educational background rather than "race."
5. Many evolutionary biologists and biological anthropologists recognize that trying to attribute every phenotypic trait of an organism to adaptation is problematic. Some traits may not be the result of adaptation but the byproduct of some other feature that was shaped by natural selection—or even the consequence of random effects.
6. Gene-centered explanations of human evolution gained considerable influence in anthropology after 1975 because of the widespread theoretical impact of a school of evolutionary thought called "sociobiology." Sociobiologists have used formal mathematical models borrowed from population genetics and game theory to back up some of their claims. However, critics have also used formal models to test sociobiological principles.
7. Anthropologists have been involved in the development of formal models critical of sociobiological models. The most influential critical models include those of gene–culture coevolution, cultural group selection, and niche construction. Anthropologists still face the challenge of deciding how to situate such critical formal models within broader anthropological discussions of human culture and history.

For Review

1. Distinguish between microevolution and macroevolution.
2. How is a species defined in your text?
3. Explain what a cline is and why it is important.
4. Explain what is meant by the "molecularization of race."
5. What are the four evolutionary processes discussed in the text?
6. Describe how natural selection explains why a high proportion of the sickling allele is maintained in certain human populations and not others.
7. What is phenotypic plasticity, and why is it important?
8. Explain the difference between short-term acclimatization and developmental acclimatization.
9. Summarize the discussion of skin color in the text.
10. Why do anthropologists and many other scholars insist that IQ is not determined by genes alone?
11. Explain why natural selection on genetic variation alone is not sufficient to explain the range of human adaptive patterns revealed by archaeology, ethnography, and history.
12. What are formal models?

Key Terms

acclimatization 159
adaptation 158
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gene flow 152
gene frequency 147
gene pool 147
genetic drift 152
macroevolution 144
microevolution 144
mutation 152
natural selection 152
phenotypic plasticity 158
polymorphous 147
population genetics 147
species 144

Suggested Readings

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Marks, Jonathan. 2011. *The alternative introduction to biological anthropology.* New York and Oxford: Oxford University Press. *An up-to-date introduction to the subfield, raising critical issues in the field that are often sidestepped in introductory textbooks. Especially strong on the value of the anthropology of science for biological and cultural anthropology.*

Mielke, James H., Lyle W. Konigsberg, and John H. Relethford. 2011. *Human biological variation,* 2nd ed. New York: Oxford University Press. *Provides a thorough and contemporary view of our biological diversity. Integrates real-world examples on interesting topics, including genetic testing, lactose intolerance, dyslexia, IQ, and homosexuality.*

Relethford, John H. 2013. *The human species: An introduction to biological anthropology,* 9th ed. New York: McGraw–Hill. *A fine introduction to modern biological anthropology, with up-to-date reviews of current research on human variation as well as chapters on primatology and human evolution. Comes with a related web page.*

Robins, A. H. 1991. *Biological perspectives on human pigmentation.* Cambridge: Cambridge University Press. *A concise survey of what is known about the biological factors responsible for human pigmentation as well as the possible evolutionary significance of variation in pigmentation in different human populations.*

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Stinson, Sara, Barry Bogin, and Dennis O'Rourke, eds. 2012. *Human biology: An evolutionary and biocultural perspective,* 2nd ed. Malden, MA: Wiley Blackwell. *The essays in this collection cover a range of topics of interest to contemporary biological anthropologists, including genetic variation, human adaptability, human biology and health, the human life course, and the dynamics of human populations.*