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

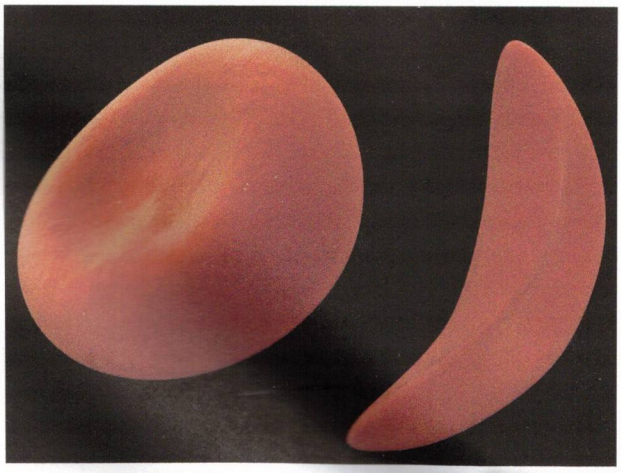

FIGURE 5.2 Normal red blood cells are easily distinguished from the distorted, "sickled" red blood cells. Sickled red blood cells carry less oxygen than do normal red blood cells, but they resist malarial parasites more successfully.

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