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).