Many older people enjoy staying active and participating in sports.

Looking Ahead

1. How do environmental hazards, developmental processes, and genetic tendencies contribute to the aging process?
2. What is the difference between normal aging and pathological aging?
3. How does aging change a person's physical appearance and mental functioning?
4. How does aging affect a person's sensory organs?
5. What effects does aging have on the bones, joints, and muscles?
6. How does aging change a person's sexual capacity?
7. What effects does aging have on the heart and blood vessels?

Twice a week Henry Sypniewski runs the hills near his home in Orchard Park, New York. That's a pretty good training regimen for a 90-year-old runner, who was recently ranked first in the country in his age group. Henry has run more than 300 5k races, several half marathons, and one full marathon. He set a U.S. record by finishing, and now plans to run another marathon sometime next year. His remarkable fitness provides support for the argument that behavioral and social factors can reduce the risk of illness and death in old age and even reverse the aging process. At the other end of the spectrum are people like Reverend Scott, who at age 71 is disabled by severe rheumatoid arthritis. Unable to work or drive a car, he maneuvers around his house in a battery-powered wheelchair (Ball and Whittington, 1995). His condition would appear to support an opposing view, that aging is inevitably characterized by an increased likelihood of disease and dependence (Manton et al., 2008).

Most older people fit somewhere in the middle of the spectrum. They are neither running marathons nor wheelchair bound, and only a small fraction of their life before death entails illness and disability (Liang et al., 2008). Genetic, biological, and behavioral factors, as well as social factors such as socioeconomic status, gender, and race all

nfluence how long people remain free of disease and dysfunction and how successful they are in slowing down the aging process.

People differ not only in how they age but also in how they react to the changes taking place in their bodies. Some accept the changes gracefully. They view their wrinkles and gray hair as symbols of a life well lived. Others are devastated by the first gray hairs or the first tiny wrinkles that appear at the corners of their eyes. They may go to great lengths to hide these telltale signs of aging, by using creams and lotions that promise to provide a youthful appearance or by taking more aggressive measures, such as undergoing cosmetic surgery to eliminate sagging chins and bags under the eyes. People who are more concerned with physical health than with appearance may take megadoses of vitamins and herbs, convinced that such a regimen can slow or reverse the aging process. They are also likely to exercise regularly. Yet the proverbial fountain of youth remains elusive.

Many theories attempt to answer the question, Why do we age? This chapter describes the more commonly proposed theories of aging and examines the normal processes of biological aging in selected systems of the body. It also examines the difference between normal aging and pathology, for as each body system ages, some pathological conditions occur. In this chapter we also consider the causes of age-related illness and explore preventative measures for improving health and functioning in later life. Throughout the chapter we consider the relationship between biological aging and its social consequences.

Theories of Biological Aging

A century before the Pilgrims arrived in the New World, the Spanish explorer Juan Ponce de León landed on the shores of North America intent on finding "the river, whose water rejuvenated the aged" (Achenbaum, 1996:4). Although Ponce de León never found the fountain of youth, interest in increasing longevity remains. For centuries, philosophers and scientists have searched for a central mechanism that causes aging. The new explorers are armed not with ships and soldiers but with the tools of science. Like the explorers who preceded them, gerontologists are interested in understanding why people grow old and what can be done to reduce illness and disability in old age. The result of a better understanding of aging is a broader range of treatments and strategies for improving the quality of life of elderly people (Cristofalo, 1996).

Most scientists now agree that aging probably does not have a single cause. The aging process occurs in part because of environmental factors and in part because of some genetically programmed purposeful process in which vulnerability to the environment increases over time as the body advances through a natural developmental process from adulthood to death. In this section, we focus first on the environmental theories of aging and then turn to a discussion of the developmental and genetic theories of aging.

Environmental Theories of Aging

Wear and tear theory An early theory of aging, first proposed in 1882 by the German biologist August Wiesmann, is the **wear and tear theory.** According to this theory, the body is analogous to a machine, like an old car or truck, that simply wears out (Cristofalo, 1988).

The problem with the wear and tear theory is that it is difficult to test. Because we don't know what constitutes normal wear and tear, we can't predict the breakdown of various body systems (Hayflick, 1996). Another problem is that the idea of wear and tear implies that a more active organism should age more quickly. Yet the opposite is true in humans. Research clearly shows that low levels of physical activity are associated with an increased risk of death (Kaplan and Strawbridge, 1994). For these reasons, the wear and tear theory is now largely discredited.

Somatic mutation theory The **somatic mutation theory** proposes that harmful or deleterious mutations, that is, genes that are incorrectly copied, will accumulate with advancing age, leading to an increase in pathological changes in body

systems. Somatic mutation theory first became prominent after World War II when scientists noted the long-term damage caused to people who were exposed to radiation from bombs (Bengtson et al., 2005). It does not take exposure to something as dramatic as a bomb to cause genetic damage, however. Over a lifetime, a person's body is exposed to many external insults from air pollution, chemicals in food and water, and radiation. According to the somatic mutation theory of aging, these insults cause mutations (genetic damage) to somatic (body) cells.

The somatic mutation theory of aging may explain variations between body systems in the process of aging. As we learn more about how environmental stressors affect the body, we will be better able to explain differences between body systems in the rate of aging. As a general theory of aging, however, the somatic mutation theory fails to explain basic processes of normal change.

Developmental/Genetic Theories of Aging

The autoimmune theory The basic function of the immune system is surveillance. It is the body's army, constantly on alert, programmed before birth to recognize and destroy invaders. The invaders are foreign proteinlike materials called antigens, such as viruses, bacteria, or precancerous cells, that the immune system recognizes as nonself. The immune system creates antibodies to destroy antigens.

The **autoimmune theory of aging** is based on two scientific discoveries. The first is that protective immune reactions decline with age, as the body becomes less capable of producing sufficient quantities and kinds of antibodies (Bengtson et al., 2005). For example, one hypothesis proposes that rates of cancer are higher in older people because precancerous cells that are recognized and destroyed in younger individuals may slip past the immune system's surveillance mechanism in older individuals.

The second discovery that lends support to the immune system theory is that the aging immune system mistakenly produces antibodies against normal body proteins, leading to a loss of self-recognition. In other words, the immune system loses some of its ability to distinguish between self and nonself and instead attacks the proteins produced by the body as if they were invaders. Rheumatoid arthritis (discussed later in this chapter) is one example of what can happen when the immune system no longer recognizes self and begins to attack tissue in the joints of the body.

Although a decline in immune system functioning causes disease, there is no evidence to suggest that a less efficient immune system causes normal aging (Hayflick, 1996). Thus, the immune function theory suffers from the same limitation as the somatic mutation theory. It is unable to account for the mechanism of biological aging. Further research is necessary before we can confirm or disprove the immune system theory of aging.

Cross-linkage theory Our cells are composed mostly of protein. One of the most common proteins, found in tendons, ligaments, bone, cartilage, and skin, is collagen. Collagen is the glue that binds cells together by cross-links, which can be likened to the rungs of a ladder that connect the two side boards. In young people, the molecules that make up the collagen protein are held together by only a few cross-links. As we age, cross-links become more numerous, resulting in tissue that is stiffer and less flexible.

According to the **cross-linkage theory of aging,** the accumulation of cross-linked collagen is responsible for such changes as the loss of elasticity of the skin, hardening of the arteries of the circulatory system, and stiffness of joints throughout the body. Specifically, cross-linking of collagen is partly responsible for wrinkling and other age-related changes in skin, and cross-linking of proteins in the lens of the eye is believed to play a role in the formation of cataracts. Researchers also speculate that cross-linking of proteins in the walls of arteries accounts for some atherosclerosis (once called hardening of the arteries). Finally, cross-linking of the proteins in the filtering systems of the kidney is responsible for the decline in kidney function in older people (Mitteldorf, 2010). Although cross-linking is one of many biochemical changes

that occur over time, there is no reason to think it is the most important cause of aging.

Free radical theory One of the most popular theories of aging is the **free radical theory.** A molecule is a group of atoms that are chemically linked. Free radicals are unstable molecules that are produced when the body transforms food into chemical energy. This transformation occurs at the level of the individual cell. Free radicals also may be generated in the body through the influence of cigarette smoke, drugs, and radiation (Dietrich and Havrath, 2010). They are a by-product of normal cells.

When free radicals try to unite with other molecules that may be in the vicinity, they can damage the cell or cause cell mutation. According to this theory of aging, free radicals contribute to the aging process by forming age pigment and by producing cross-links. Thus, most changes associated with aging result from damage caused by free radicals (Bengtson et al., 2005). Free radicals have also been implicated in various cancers and in Alzheimer's disease (Hayflick, 1996).

The body has its own natural defense in the form of chemical inhibitors called antioxidants, which suppress the formation of free radicals and reduce the cellular damage they cause. Among the antioxidants that suppress free radicals are vitamins E and C and betacarotene (related to vitamin A). A recent study of mice engineered to produce high levels of an antioxidant enzyme lived 20 percent longer than normal and had less heart and other age-related diseases. Does that mean that you should rush to a health food store and stock up on antioxidant pills? The researcher who conducted the study says no, explaining that for now the evidence on the benefits of oral antioxidant pills is weak. A better strategy is to consume fruits and vegetables like broccoli that contain high amounts of antioxidants (Schriner et al., 2005). The question for humans is whether increasing the dietary intake of antioxidants can increase longevity.

The free radical theory combines an explanation of developmental change with environmental factors. Although it is useful for understanding why some individuals are at greater risk of certain diseases than others and for describing part of the aging process, it is not, in itself, a general theory of biological aging.

Genetic control theory In Chapter 4, we defined the life span as the greatest number of years a member of a species has been known to live. In humans that appears to be about 120 years. The distinction of being the oldest verified person in history belongs to a French woman named Jeanne Calment, who died in 1997. At the time of her death, she was 122 years and 164 days old.

Was Jeanne Calment biologically programmed for such exceptional longevity? No one knows for sure, but the variation in life span among different species does suggest that life span may be programmed into the genes. Studies of human twins also support the idea of genetic programming. Identical twins, who share the same genetic makeup, have similar life spans and tend to die of similar causes. Fraternal twins, who are no more alike in their genetic makeup than any other siblings, do not (Goldstein, 1971; Goldstein et al., 1989).

Where might the genetic control for aging reside? The **genetic control theory of aging** proposes that it is programmed into each cell of our bodies. Fascinating experiments using cell cultures support this idea. In these experiments, cells are taken from human embryos as well as from people of various ages and grown in cultures in a laboratory. The cells from an embryo will divide approximately 50 times before dying, but similar cells from an adult will divide only 20 times. Despite such evidence supporting the theory that the genetic information in our cells provides a blueprint for the entire aging process, other factors also seem to be at work. Many complex changes that precede cell death cannot be explained solely by genetics (Cristofalo, 1996).

Although genes influence life expectancy and the tendency toward certain diseases, genes do not determine whether an individual gets a specific disease or how long an individual lives. Many people with a genetic susceptibility to a specific disease never get it. For example, there is a tendency for Alzheimer's disease to be hereditary, but many people who have a close relative with Alzheimer's

do not succumb to this illness. Further, evidence suggests that engaging in challenging mental activity and physical exercise can delay the onset of Alzheimer's and lessen its severity. Similarly, susceptibility to breast cancer is hereditary, but many women whose mothers had breast cancer do not get it (Ryff and Singer, 2005).

The search for an explanation for biological aging has long preoccupied scientists, perhaps because humans wish to discover the secret to a long life. Recently scientists have discovered some genetic evidence that helps explain why some people live longer than others. In some cultures people are genetically isolated, either because they live in a relatively closed community or because they share a common culture and high rates of intermarriage. Often people in these communities have exceptional longevity. In studying this phenomenon, scientists have found gene mutations that seem to prevent the diseases that most often shorten life. One example is the Ashkenazi Jews, who are the descendants of central European Jews and have high rates of intermarriage. Many live well into their 90s or even 100s, like Irving Kahn who at the age of 106 worked five hours a day in his family's New York investment and brokerage firm. Ashkenazi Jews have genetic mutations that seem to provide protection against high blood pressure and Alzheimer's. Another group is the Old Order Amish, who live as an isolated community in Lancaster, Pennsylvania. They have a mutation that lessens fat in the blood. Male Japanese Americans carry a gene mutation that reduces the risk of cancer and heart disease. Although these gene mutations do have a protective effect, genes alone cannot explain all the variations in longevity. There is also evidence that environmental influences such as diet, exercise, and education can modify gene activity in a manner that increases or reduces longevity (Hall, 2013).

Neuroendocrine theory The **neuroendocrine theory** proposes that a functional loss in neurons and their associated hormones is central to the aging process. As we age, the body produces lower levels of hormones that are vital for well-being. For example, a decline in human growth hormone results in changes in body composition. Lean body mass shrinks and there is an increase in adipose (fatty) tissue. This loss of lean body mass leads to atrophy in skin, skeletal muscle and bone. The decline of human growth hormone can also lead to elevated cholesterol levels (Park et al., 2011).

The Aging Body

Biological aging refers to the structural and functional changes that occur in an organism over time. It is a period in the life history of an organism that begins at maturity when development is complete and lasts for the rest of the life span (Cristofalo,

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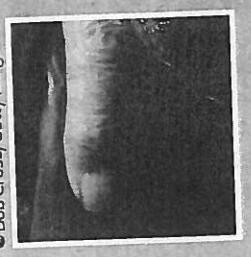

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Diversity in the Aging Experience

Life Expectancy and Health Behaviors among Mormons

Although it has long been known that Mormons who are active members of the Church of Jesus Christ of Latter-day Saints (LDS) have longer life expectancy than the general population, even researchers have been surprised at how much longer Church members live. Among men life expectancy for this group is 84 and for women, 86. That is more than five years longer for women and seven years longer for men than the national average.

Why do actively practicing LDS members live longer than everyone else? The main reason is that they lead a healthier lifestyle as advocated by their religion. Practices and beliefs that increase life expectancy include a strong family life, education and abstention from tobacco and alcohol. These data come from a 25-year-long survey that followed actively practicing LDS members in California since 1979 and shows that those who don't smoke, attend church weekly, have at least 12 years of education and are married have the lowest total death rates and the longest life expectancies ever documented. The researchers concluded that the healthy characteristics of the Mormon lifestyle are associated with substantially reduced death rates and increased life expectancy (Enstrom and Breslow, 2008).

What Do You Think?

1. Do you lead a healthy lifestyle?
2. What can you do to improve your longevity?

1996). The normal processes of biological aging rarely lead to death by themselves but rather involve a period of decline that results in increasing frailty. Frailty consists of three markers: the loss of a sense of invincibility, the loss of possibility for a subsequent life change, and the loss of ability to do things that are crucial for one's care (Hadler, 2011). The aging-related diseases such as cancer, diabetes, heart disease, and Alzheimer's disease do increase the risk of death.

A good example of increasing vulnerability is the reaction of the aging body to a fall. An 18-year-old boy who slips and falls on ice will react quickly, putting out his hand to break the fall. He might fracture his wrist and have to wear a cast for six weeks but then will resume his life as if nothing had happened. An 85-year-old woman who takes a similar fall has a good chance of fracturing a hip. She would probably spend time in a hospital and then more time in a convalescent home. Often, a broken hip will mean the end of independent living forever. In the worst cases it can mean death, for if people are inactive for an extended period, their lungs fill with fluid. Death from pneumonia may follow.

Often medical interventions can slow the progression of age-related diseases by blunting their symptoms and restoring functioning. Then people may be able to return to work, participate in sports,

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Aging Around the World

International Variations in Active Life Expectancy

Although nearly everyone wishes to live as long as possible, most people believe that active life expectancy is more important than just living more years. Average life expectancy varies dramatically across nations, so it is not surprising that active life expectancy is also influenced by where you live. Active life expectancy is highest in Switzerland, where men can expect to live 79 years free of disability and women 76 years. It is much lower for both men and women in Australia and Canada. The U.S. is somewhere in the middle. In most countries, women have lower active life expectancy than men.

Making comparisons across nations is still difficult, because active life expectancy is not an exact concept. It is measured by an individual's ability to complete **activities of daily living (ADLs)** such as bathing, eating, getting in and out of bed, and toileting. There are no standard measures of how well people perform these functions, but researchers are working to develop some. Some of the causes of variation across nations include rates of poverty, quality of medical care, crowded housing, nutrition, and amount of smoking (Gjonca and Marmot, 2005).

What Do You Think?

1. What is the active life expectancy of people in your own family? How does it compare with the average active life expectancy in the nation where you live?
2. Can you think of any reasons for the differences in active life expectancy?

and enjoy simple pleasures like attending the theater or working in the garden. Sometimes, however, medical interventions fail. When chronic conditions like arthritis, heart disease, or osteoporosis make it difficult for people to go about their daily activities, we say that they are disabled (Crimmins et al., 2009).

Although disability is a distressing outcome of some aging processes, most people spend very few of their years disabled. The measure of the number of years a person can expect to live without a disability is called **active life expectancy.** Men have an active life expectancy of 60 years, which is 84 percent of their whole life expectancy. Women can expect 58 years of disability-free life, which constitutes 82 percent of their lifetime (Kinsella and Gist, 1998). Women have fewer years of active life expectancy because they are more likely than men to live past 85, when the risk of becoming disabled from chronic ailments increases rapidly and they are more prone to disabling conditions like arthritis and osteoporosis. The good news is that the gender gap has declined among younger cohorts of women (Liang et al., 2008). Overall, active life expectancy has improved for both men and women. People are living longer and with better health (Manton et al., 2008). There is considerable evidence that shows that lifestyle habits contribute to longer life expectancy. The "Diversity in the Aging

Experience" discusses the reasons why Mormons live longer than the rest of the population.

Race and ethnicity are also related to active life expectancy, because minorities are less likely to have health insurance, more likely to be employed at jobs where there is greater risk of injury, and more likely to engage in behaviors like smoking that increase the likelihood of disability. We discuss this topic in more detail in Chapter 11. A 20-year-old white, non-Hispanic male can expect 14.5 percent of his predicted future years to be inactive due to disability. By contrast, an African American 20-year-old will have 18.6 percent of those years inactive, and a Native American, 24.8 percent (Hayward et al., 1996). Thus, active life expectancy varies by gender, ethnicity, and race. Finally, active life expectancy varies from one nation to the next. See "Aging Around the World" for a comparison of the active life expectancies in several Pacific, European, and North American nations.

Some of the bodily changes that occur with age decrease active life expectancy; others have few or no health consequences. Let's take a closer look at some of these changes.

Aging of the Exterior Body: Skin and Hair

Wrinkles and sagging skin The skin serves as the body's first line of defense. It protects against water loss, regulates heating and cooling, and contains receptors that monitor pain and pressure. One of the most obvious signs of aging is the change in skin texture that we know as wrinkling. Some wrinkling is related to use. Common facial expressions such as smiling and frowning hasten the appearance of wrinkles at the corners of the eyes (crow's-feet), forehead, and mouth (Donofrio, 2003). Most wrinkling, however, is caused by biological change that occurs as we age because the deeper layers of the skin lose their elasticity. As elasticity is lost, wrinkles appear in the smooth skin around the eyes and at the corners of the mouth; the chin sags. Hard areas of salt deposits further reduce the flexibility of the deeper skin layers.

The natural process of skin aging has no health consequences. No one dies of skin failure, although many people try to hide the aging process through the use of creams, surgical face-lifts, and collagen implants. The attempt to retain a youthful appearance that is so pervasive in our society reflects negative stereotypes and attitudes about aging (Kornadt and Rothermund, 2011). It also reflects age discrimination in the workplace, which pushes people to maintain a youthful appearance.

Hair Another common sign of aging is gray hair. In the hair roots, cells called melanocytes produce chemical proteins that determine the coloring of hair and skin. With age, the melanocytes weaken and their pigment-producing mechanism begins to cease functioning. The graying of hair is caused by a decrease in the number of active pigment-producing cells. When the melanocytes stop working completely, the hair turns white. There is great variation in how rapidly graying occurs. Some people may turn gray in their 40s; others still have their natural hair color at 70. Factors that affect premature graying include family history, smoking, and obesity (Shin et al., 2014).

Hair loss may also accompany aging, especially in men. It occurs through the interaction of genes with the male hormone testosterone. Men who have a genetic predisposition for baldness may show the classic signs of male pattern baldness as young as 20. In these men, testosterone acts with the genes to promote baldness. Men who lack this genetic predisposition may still have a full head of hair in their 80s.

Hair also grows as we age, but it seems to grow in all the wrong places. In men, the hair of the scalp grows more slowly, but hair in the nostrils, ears, and eyebrows grows more rapidly. In women, hair growth may occur above the upper lip, as a result of the decrease in the hormone estrogen that accompanies menopause. Although excess hair growth has no effect on health, many people find it unattractive and become self-conscious about their appearance. Fortunately, electrolysis is a simple procedure that eliminates unwanted hair.

Skin discoloration The superficial layers of the skin also show signs of aging in the form of darkened spots and other skin changes. **Lentigo** is the discoloration or spotting that commonly appears