CHAPTER

2

Biological Aging, Health, and Longevity

Why do we age, and what can we do to ensure a longer, healthier life? Most Americans over age 65 years would be pleased to live to be 100 years old, if they could be assured that they had good health and sufficient financial resources (Kiger, 2018). In this chapter, we will discuss theories regarding biological aging and factors that influence the quantity and quality of our later years.

As mentioned in Chapter 1, there are many different kinds of aging. In this chapter, we will focus on **biological aging**, the changes in the body's structure and function over time. Although these changes may include growth or decline, when we speak of biological aging in mid to late adulthood, most of those changes are declines. However, the location, rate, and timing of those declines are often the result of nature, nurture, and their interaction. Thinking back to Baltes's (1987) principles of life span development, you may recall that as we age, biology becomes *less* important in our development, whereas culture or nurture becomes more salient. This is true, in part, because biological forces and sociocultural influences have been acting on an aging individual since before birth. Remember, however, that Baltes was primarily addressing functional and psychological aging. In this chapter, we will highlight some of the mechanisms of biological aging, their relation to health and longevity, and some newer research that suggests that we might be able to alter the course of aging and health for many people.

Why Do We Age? Theories of Biological Aging

Early research with cardiac cells from chickens led Leonard Hayflick to state that cells are not immortal, but have a limited number of replications. For humans, Hayflick (1965) demonstrated that normal human cells divide and replicate 40 to 60 times, then begin the process of dying. These 50 or so replications are known as the **Hayflick Limit**. But why do our cells stop dividing and replicating after about 50 such divisions? As a science, we do not have a single, definitive answer to that question. We do, however, have useful theories to help us understand the phenomenon of biological aging.

People often misuse the term "theory" in everyday language, suggesting that a guess or suspicion is the same as a theory. For example, imagine that you are making plans to go see a movie with a group of your friends. One of these friends is disorganized and rarely makes schedule notes. When the day of your movie arrives, your disorganized friend is late. When you are asked why the friend is late, you might be tempted to say, "Well, I have a theory about that." In science, however, theories are more than mere hunches or educated guesses. They are useful tools that enable us to make sense of a large body of information and that guide future scientific explorations. To be properly termed a theory, the idea must meet specific criteria. To be viewed as a theory, the topic being studied must entail specific definitions. There must be an awareness of the limitations of the theory and the domains to which it can be applied. It should help us to see additional relationships among domains. Theories also lead to testable predictions. Additional characteristics help determine whether a theory is good. Among those qualities of a good theory is **parsimony**, the preference for simple explanations rather than overly complicated or convoluted ones. A good theory offers **heuristic value** or fecundity. Heuristic value is defined as usefulness for inspiring or producing additional insights. A good theory is also **generalizable**, in that it can be used to explain a wide range of findings (Wacker, 1998).

Over the past centuries, many theories have been proposed to explain why we age. As shown in Table 2.1, although some scholars prefer to group theories by whether the theory focuses on a specific organ system or at the cellular level, non-biologists generally divide theories of biological aging into two main groups: **genetic-programming theories** and **variable-rate** or **error** theories of aging (Hayflick, 1985; Jin, 2010). There are several programmed theories of aging, and each has some strong support. It is likely that as we continue to learn more about aging, some of these theories will be combined. Others may be discarded in favor of better or more parsimonious theories. For the purposes of this chapter, however, we will present each as a unique theory.

TABLE 2.1 Theories of Biological Aging

Genetic-Programming Theories	Description/Hallmark
Immunological	The MHC is the site of biological aging
Neuroendocrine Theory	Hormones are the "master clock"
Programmed senescence	Telomeres are the "master clock"
Error/Variable Rate Theories	
Wear-and-tear	Biological resources cannot repair sustained damage
Oxygen-free radicals	Free radicals cause damage within the body, which builds up over time
Autoimmune failures	Immune system falsely recognizes healthy tissue as pathogenic

Sources: Adapted from Hayflick, 1985; Jin, 2010; Weinert & Timiras, 2003.

Genetic programming theories rest on the idea that there is a biological limit to the integrity of our bodies—it is built into our genetics. Different theories, listed in Table 2.1, propose different sites or mechanisms for the onset of aging. The **immunological** theory attributes biological aging to decreases in the efficiency of our immune system and its production of antibodies. The master genetic control for the immune system is known as the Main Histocompatibility Complex (MHC), and systems associated with it and the thalamus are the sites of decline (Hayflick, 1985). Critics of this approach note that declines in immune system functioning are not universal, but all humans do age. Critics of the immunological approach point out that the immune system comprises cells, and these cells are aging. Thus, the mechanism of age-related decline may be more general, at the cellular level, rather than at the level of the immunological system. Finally, critics note that much of the immune system is regulated by hormones, and aging may relate to declines in hormones, rather than the immune system, per se (Hayflick, 1985).

The **neuroendocrine theory** focuses on the roles of hormones in the aging process and recognizes the importance of the **hypothalamic-pituitary-adrenal (HPA) axis** as a "master clock." The HPA is involved with coordinating communication within and across body systems, coordinating physiological responses to external stimuli, and maintaining a homeostatic balance of resources across reproductive and repair functions in the body. Thus, changes in the HPA axis have profound effects on the entire body in terms of homeostasis and resilience (Weinert & Timiras, 2003). Human menopause, the cessation of menstruation and ovulation in adult females, is often cited as evidence for the neuroendocrine theory of aging. As a consequence of menopause, women experience a reduction in both estrogen and progesterone, which are associated with decreases in immune function (Ghosh, Rodriguez-Garcia, & Wira, 2014).

A third preprogrammed theory links the rate and timing of aging to **programmed senescence**, which is under the control of telomeres (Anderson, Le Couteur, & de Cabo, 2018). **Telomeres** are the protective ends on chromosomes that prevent loss of genetic information during cell division and replication. As the cell divides and replicates, a small piece of the telomere is cut or lost. In addition to age, other factors can shorten the length of a telomere, including poor nutrition, high levels of stress, obesity, cigarette smoking, and physical inactivity (Shammas, 2011). The length of telomeres and the rate at which they shorten are important **biomarkers** of aging. Shorter telomeres are associated with increased risks of cancers in the lung, bladder, renal cells, digestive system, and head (Shammas, 2011).

In addition to these preprogrammed theories, there are several variable rate or error theories of aging. Variable-rate or error theories share the common assumption that aging occurs because of damage to the organism over time. Among the earliest error theory was that of **Wear-and-Tear** (Jin, 2010). The wear-and-tear approach states that our bodies age as a function of use and lack of proper repair or maintenance. This approach further suggests that if we had unlimited biological resources, we could continually renew and repair our bodies. But, as Baltes's Principles of Life Span Development state, we experience a decrease in biological resources, and more resources are needed for maintenance in late life, leaving fewer resources available for repair. The classic example of wear-and-tear concerns an automobile. With unlimited effort and resources, a person could maintain their

favorite vehicle indefinitely. But, as a car ages, its systems require more frequent and often more expensive attention. At some point, the owner may decide that the costs of repairing the vehicle exceed the wisdom of doing so.

Perhaps the most popular error theory today is the **free radical theory** of aging. During normal metabolic processes, an unpaired oxygen molecule is released in the body. This free radical sets off a chain reaction of damage throughout the body, as it searches for an available electron with which to pair. The free radical can cause damage to collagen and DNA, and it can cause a buildup of waste materials that weaken the cell walls (Hayflick, 1985). We have long known that antioxidants, like those found in certain foods, can help limit the free radical damage. Newer research is demonstrating that an increase in dietary antioxidants results in increased life expectancy among mice, rats, and fruit flies (Kennedy et al., 2014). Hekimi, Lapointe, and Wen (2011) argue that although free radicals are strongly linked to the rate of aging, their role in aging may be broader than solely a cause of oxidative damage. They suggest that free radicals may also play a role in mediating the body's response to stress and age-dependent damage. Thus, there is much still to be learned about the role of free radicals and aging.

A third error theory is the **autoimmune** approach. As the immune system declines, the body fails to appropriately recognize pathogens, and it falsely identifies healthy tissue as infectious. Thus, due to these errors, certain chronic health conditions occur, such as rheumatoid arthritis and diabetes (Hayflick, 1985). We will discuss these health conditions in more depth in the next sections.

Sensory Aging

Recall that a good theory must account for a broad range of findings in different domains. Thus, it is necessary to understand how aging looks in different systems of the body. Now that you have some understanding of the different kinds of theories related to biological aging, we turn our attention to the broad domain of **sensory aging**. Sensory aging refers to age-associated changes in the structure and functioning of our sense organs, especially in terms of our ability to see (**vision**), hear (**audition**), smell, (**olfaction**) taste (**gustation**), and feel (**somesthesis**). The effects of biological aging can be seen in every system of the body, including sensory systems. The rate, location, severity, and timing of these changes depend on both genetics and environment, and their interaction (National Institute on Aging [NIA], 2016). It is important to note that there are robust gender and race differences in the ways in which sensory aging affects Americans.

Moreover, these sensory changes do not occur in isolation; rather, adults often experience multiple sensory declines across mid and late adulthood. Research from the National Social Life, Health, and Aging Project (NSHAP) shows that 94% of older Americans experience at least one sensory deficit, 38% experience two sensory deficits, 28% report having three or more sensory deficits. Not surprisingly, having multiple sensory deficits is more common with advanced age, especially in vision and hearing. Men report better corrected vision than do women, but men experience more frequent and more severe losses in hearing, smell, and taste. Older Hispanic adults report better gustatory senses but perform more poorly on tests of vision, touch, and olfaction. Finally, older African American adults score lower on all senses except audition (Pinto et al., 2014).

Age-Related Changes in Vison

Peak visual functioning occurs during late adolescence or emerging adulthood and remains stable through midlife. However, a number of age-related changes occur in the visual system that can contribute to impaired vision (Besdine, 2016). By 2050, it is anticipated that approximately 2 million people will be classified as legally blind, nearly 7 million will have a visual impairment (VI) equal to seeing at 20 feet what most people can see at 200 feet, and more than 16 million will have a VI due to uncorrected refractive errors associated with myopia, hypermetropia, astigmatism, and presbyopia (Varma et al., 2016). Many of these vision impairments are associated with age-normative changes, including (1) a thickening, yellowing, and increased opacity of the lens, resulting in less light being projected onto the retina; (2) a weakening of the ciliary muscles which alters the ability of the lens to focus; (3) a thickening of the aqueous humor, which limits metabolic support for the lens and cornea and possibly increases intraocular pressure associated with **glaucoma**; and (4) a decrease in the pupil's resting diameter, known as senile miosis, which results from weakening muscles and contributes to less light on the retina. These structural changes result in decreased visual abilities or functions, which have consequences for the person's behavior and functioning (Kline & Scialfa, 1997; Schieber, 2006). For instance, problems with eyesight represent the leading reason given by both older men and women to limit or avoid driving (Ragland, Satariano, & Macleod, 2005).

In addition to these structural changes, the visual system experiences functional changes, too. As we age, we require more light to perform daily activities like reading or using our cell phones. This increase in **absolute threshold**, the minimum level of stimulus energy or intensity required to see an object, increases with age (Kline & Scialfa, 1997). If you are concerned about the safety of an adult with possible VI, you can make safety-related changes to the environment, as described in Table 2.2.

Accommodation is the process whereby the eye adjusts its focus both near and far in order to gain clarity. With aging, there is a decrease in the ability of the eye to focus and/or refocus on objects at varying distances (Panek, 1997). Age-associated problems with visual accommodation is known as **presbyopia**, which is a decline in the eye's ability to focus on near objects and is due to a loss of elasticity of the lens. This is why many individuals in middle age need glasses for reading or for working with objects that are close to them.

Visual acuity is the ability to resolve detail. It is equated with the accuracy of distance vision compared with that of a hypothetical normal person, which is measured by means of a Snellen chart, consisting of a standardized series of letters, numbers, or symbols that must be read from a distance of 20 feet. The ability to read this chart is termed **static visual acuity**. If an individual with normal vision can read a designated letter on the Snellen chart at a distance of 20 feet, this is called 20/20 vision. A person who can distinguish at only 20 feet a letter that a person of normal vision can distinguish at 100 feet is said to have a visual acuity of 20/100.

Visual acuity tends to be relatively poor in young children, but improves in young adulthood, and shows a slight decline from the mid-20s to the 50s. Beyond this point, the rate of decline is accelerated. The average static visual acuity for adults aged 65 years and older is 20/70. Decreased visual acuity creates difficulties in reading, watching television, and reading labels on medicine bottles. Providing more ambient

TABLE 2.2 Environmental Interventions to Support Sensory Declines

Specific Sensory Issues	Behavioral Challenge	Environmental Solution
Visual System		
Senile miosis Higher absolute threshold	Less light is available to the retina, so an increase in light is needed	Replace light bulbs early and use higher intensity bulbs
Decreased adaptation	Eye responds more slowly to changes in lighting	Place lighting at the top and bottom of staircases to ensure a steady level of illumination
Glare from headlights of oncoming traffic	To minimize effects of glare, look to the right side of the road, not directly at the light; slow your own driving to match that of the distance that your headlights illuminate	New technologies enable more light for drivers without adding to the glare for oncoming vehicles
Auditory System		
Higher volume threshold	Difficulty hearing speech, especially in noisy areas	Speaker should face the person with hearing loss, speaker should speak slightly louder but without exaggeration
Hair cells break	High frequencies are lost (difficulty hearing women or children)	Speaker should enunciate clearly, speaker should use a slightly lower register
Gustation		
Fewer and smaller taste buds	Lower sensitivity to taste; food may not be appealing	Avoid using excess salt, but experiment with other spices
Less saliva is produced	Dry mouth	Sips of water may help
Olfaction		
The number of neurons in the olfactory bulb are stable, but there may be fewer synapses	Decreased sensitivity	Use safety devices, like smoke detectors; maintain good nutrition (link between gustation and olfaction)
Touch		
Less blood and fewer strong signals from spinal cord	Difficulty feeling temperature change and regulating own temperature	Rely on thermometers to help decide how to dress; decrease temperature on water heater; be vigilant about scratches/injuries

Sources: Adapted from Barry, 2019; Lutz et al., 2018; MedLine Plus, 2019.

light or making objects larger and more distinct (termed contrast sensitivity) are relatively easy environmental supports for persons with static visual acuity problems (Long & Crambert, 1990; Schieber, 2006).

There are also age decrements in **dynamic visual acuity** (Long & Crambert, 1990), which is the ability to accurately identify a moving target, such as a television message, a weather warning, or a street sign seen from a moving car. The more quickly the target is moving, the more older people are disadvantaged. The decrement with age in dynamic visual acuity appears to be related to changes in the thickness of the lens and the size of the pupil. Dynamic visual acuity is especially critical when driving. When searching for a specific street, many older drivers must be much closer to the street sign in order to read it relative to an emerging adult. By the time the older can read the sign, they may not have sufficient time to brake and signal before turning.

Regarding **color vision**, with increased age there is increased difficulty in discriminating among the blues, blue-greens, and violets—the low to middle range of the visible light spectrum—and much better successes in discriminating among the reds, oranges, and yellows, the upper middle to high range of the visible light spectrum (Kausler, 1991). Color vision deficits are more apparent when levels of illumination are low and when fine discriminations in shades of a particular color are being made (Fozard, 1990). The consequences of distortions in color vision can range from minor, such as choosing two different colored socks, to severe, such as an inability to differentiate medication tablets by color.

Adaptation refers to the sensitivity of the eye to adjust to changes in levels of illumination. **Dark adaptation** is increased sensitivity to light in a dark environment. For example, when you enter a dark movie theater, your pupils will expand to increase the amount of light entering your eyes (dark adaptation). This process takes about 30 seconds for adults, but older eyes may require more time to adjust. The reverse happens when you leave the theater, that is, your pupils will automatically contract to cut down the amount of light entering your eyes (light adaptation). This process requires a shorter time than does dark adaptation (Hayslip et al., 2011). Think for a moment about the eye's adaptation to changes in illumination. If you exit a dark theater and move into bright sunshine, you may feel blinded. If you continue to move in that space, it is possible that you will not be able to see changes in elevations, like from a sidewalk to a parking lot.

These issues with adaptation also play a role when dealing with **glare**. Glare is the relatively bright light that results in unpleasantness or discomfort and/or interferes with optimum vision. We experience glare when light rays are diffused via a change in the composition of the vitreous humor. An example of this process occurs during night driving when you view the headlights of oncoming autos. The negative effects of glare on performance increases with age from age 40 on. For middle-aged and older drivers, this temporary blindness resulting from glare can easily cause an accident. In a recent study with older drivers, Kimlin, Black, and Wood (2017) found that nighttime glare and a decreased sensitivity to detect motion were significantly associated with driving impairments, especially for the detection of a pedestrian.

Glare is not a problem only for older drivers. Bright sunlight and the headlights of oncoming traffic can create glare problems for all drivers. Newer automobiles

are using much brighter headlamps than before. Drivers are able to see much more of the road ahead of them, but the glare caused by these lights may exacerbate glare problems for other drivers. However, automobile manufacturers are also developing technologies that allow the driver to continue to benefit from increased illumination, while decreasing some of the glare for oncoming drivers (Barry, 2019).

Visual field is the total extent of physical space visible to an eye in a given position—the whole area you see when your head is in a fixed position. For emerging adults, visual field is typically 180 degrees, but by age 70, it decreases to approximately 140 degrees. The **peripheral field** is the outer area of your overall visual field and shrinks several degrees per decade after age 45 (Kline & Schieber, 1985; Schieber, 2006). The more your visual field is restricted, the more you must turn your head to see what you used to see out of the corner of your eye, with your peripheral vision. This decline is significant since a great deal of important information from the environment comes to us from the peripheral visual field. Karlene Ball and her associates have developed and validated a measure of the **useful field of view** (UFOV; Ball, Owsley, Sloane, Roenker, & Bruni, 1993; Edwards et al., 2006). The UFOV assesses the amount of information that one can obtain from a visual array. Evidence shows that the size of the UFOV accurately predicts which drivers have a history of automobile accidents. Older adults with an especially narrow UFOV were 6 times more likely to have had an automobile accident in the previous 5 years. We will return to the UFOV in Chapter 3, when we discuss applied cognitive aging and the interactions among sensory and biological aging with cognitive performance.

Vision-related disorders are not considered to be part of normal aging, but they may increase with advanced age. Four common disorders are of special significance:

Cataracts are a clouding of the lens of the eye, interfering with our ability to focus, producing halos around objects, and increasing problems with glare. Cataracts can be caused by a variety of environmental factors, such as smoking or eye injury, but there may also be a familial influence. The clouding begins around age 60 but may not cause noticeable vision difficulty until age 75 years. By age 75, most people will have cataracts. Treatment includes environmental changes, such as increased lighting, but the cataract can only be removed by surgery (MedLine Plus, 2019).

Glaucoma refers to a group of eye diseases that result in damage to the optic nerve. Pressure inside the eye increases, causing damage to the optic nerve and reducing peripheral vision and giving the experience of tunnel vision. Over time, even this tunnel vision may decrease until the person has no vision remaining. Glaucoma is the leading cause of blindness in the United States and is more common among African Americans over age 40 years, Mexican American adults over age 60 years, and those with a family history of glaucoma. Currently, there is no cure for glaucoma, but eyedrops and surgery may be helpful (MedLine Plus, 2019).

A third common eye disease is **Age-Related Macular Degeneration** (AMD). With age, some people experience a decreased blood supply to the macula, within the retina of the eye. The macula is responsible for sharp focus. With a reduced blood supply, the macula and the entire retina are damaged. AMD results in a loss of sharp, central-field vision, making reading and other detail-oriented tasks difficult. Although the causes of AMD are not yet certain, White adults, women, people with a family history of AMD, adults who eat a high-fat diet, and cigarette smokers are at a higher risk (MedLine Plus, 2019). See Box 2.1 for new research highlighting a new treatment for AMD.

By 2050, it is expected that more than 14 million Americans will have **diabetic retinopathy**. Diabetic retinopathy is caused when diabetes damages the blood vessels in the retina. This damage leads to blurry vision, floaters that appear as splotches obstructing the visual field, halos around lights, loss of central vision, and loss of color vision. Adults with type 1 or type 2 diabetes, especially if poorly controlled, are at high risk. Women who experienced gestational diabetes during pregnancy are at high risk as well. Surgery and medications may help (MedLine Plus, 2019).

BOX 2.1

NEW STEM CELL TREATMENTS FOR AMD

Age-Related Macular Degeneration is a leading cause of vision impairment among adults over age 50 years. The visual impairment affects one's ability to perform basic Activities of Daily Living (ADLs), such as feeding oneself, Instrumental Activities of Daily Living (IADLs), such as getting around town, and many leisure time activities. Thus, AMD has the potential to significantly reduce one's quality of life.

Researchers have attempted to develop treatments for AMD that involved human stem cells. Stem cells, as you likely know, are a bit like a blank slate, having the potential to become any specialized cell in the body. Two critical issues with human stem cell research have prevented advances in stem cell research with AMD. First, when a stem cell strain is donated from another person, the recipient's body may reject those cells. Second, early stem cell research has shown that these cells have a higher likelihood of becoming cancerous. But new research from the National Eye Institute at the NIH is showing promising results.

Researchers are testing the effectiveness of using a person's own stem cells to become retinal pigment epithelium (RPR) cells. These RPE cells could then be grown into a patch that can be introduced to the retina affected by AMD. Studies with rats and pigs are showing that these patches can be integrated into the eye and that the cells continue to develop into mature stem cells, which should be able to help keep the photoreceptors in the eye healthy. Of note, these new cells do not show any signs of becoming cancerous, as was noted in previous stem cell trials. The researchers are now planning to test the safety of this treatment in people.

Source: Sharma et al., 2019.

Age-Related Changes in Audition

You probably learned in an elementary school class about how the human auditory system works: Sound travels through the air in waves that are converted to electrical signals. As the waves travel through the ear canal, the eardrum vibrates. This vibration is carried by the three bones in the middle ear through the fluid in the cochlea of the inner ear. When the vibrations from the fluid reach the basilar membrane, hair cells begin to move, which stimulate the production of chemicals. These chemicals rush into the cells, creating an electrical signal which is carried by the auditory nerve to the brain. The brain then interprets that signal as sound (National Institute on Deafness and Other Communication Disorders [NIDCD], 2018). Aging interferes at each step of the process, from receiving incoming sound waves through the conversion of electrical signals into sound.

Several structural and functional changes in the auditory system occur with age. These contribute to hearing loss in older adults: an accumulation of fluid in the middle ear, thickening of fluid in the ear, atrophy and degeneration of hair cells in the cochlea, loss of auditory neurons, and wax buildup (NIDCD, 2018). Typical age-related changes in hearing, termed **presbycusis**, include an increase in the absolute threshold for detecting sound, such that sounds must be louder in order to be detected; difficulty distinguishing between certain sounds (e.g., "s" versus "th"); and a loss of ability to hear high-frequency sounds, such as speech by women or children (NIDCD, 2018). Presbycusis is bilateral (i.e., occurs in both ears) and progressive so that a person is often not aware of the gradual loss in hearing.

Globally, hearing loss affects about one third of older adults, with between 50% and 80% of adults aged 80 years and older having significant hearing loss (World Health Organization, 2013). Hearing loss is associated with both genetic and environmental factors, as well as their interaction. The ability to hear decreases quite dramatically across the life span, potentially compromising performance on a variety of daily tasks. Older adults may experience difficulty engaging in normal conversation, hearing the telephone, hearing verbal instructions regarding the use of medications, or participating fully in therapy and geriatric assessments (Lutz, Gallegos, & Edelstein, 2018). We know that men tend to experience hearing loss at much younger ages than do women: Most men notice some decrease in hearing around age 30 years, whereas women notice declines around age 50 years. Moreover, men's hearing loss progresses more quickly than it does for women (Sharashenidze, Schacht, & Kevanishvili, 2007). In addition to these gendered differences, environmental factors such as occupational noise, pharmaco-therapeutic agents, industrial chemicals, rapid changes in ambient pressure, and chronic health conditions, such as diabetes, ear infections, and cardiovascular disease, affect hearing (Strawbridge, Wallhagen, Shema, & Kaplan, 2000).

Age-Related Changes in Gustation

The most common sensory loss identified in the NSHAP study was a decrease in gustation, with 74% of adults reporting a decreased sense of taste (Pinto et al., 2014). Your mouth produces less saliva as you age. This can cause dry mouth, which can

BOX 2.2

GUSTATION

We are able to detect four basic taste qualities: sweet, salty, bitter, sour, and the controversial taste quality termed umami (Moject, Christ-Hazelhof, & Heidema, 2001). An example of umami is monosodium glutamate (MSG), which is described as salty with a greasy aftertaste. The receptors for taste are the taste buds, which are on the tongue, and the taste buds for each of the four basic taste qualities tend to be clustered on specific locations on the tongue. The taste buds become fully developed in early adolescence and remain relatively unchanged until the mid-40s, when signs of atrophy begin to appear. The specific age-related changes in the taste system include a gradual decrease in the number of taste buds, a loss of elasticity in the mouth and lips, a decrease in saliva, and fissuring of the tongue.

affect your sense of taste. **Taste buds**, the sensory receptors on the tongue, also change with age. Adults have about 10,000 taste buds, but we experience a decrease in the number and size of taste buds as we age. Although many adults are able to identify a range of tastes, their sensitivity to the five (or six) tastes (see Box 2.2) often declines after age 60 (Saxon, Etten, & Perkins, 2014). This decreased sensitivity to taste is termed **hypogeusia**.

To assess gustation and potential hypogeusia, the NSHAP researchers (Correia et al., 2016) used paper strips that were infused with sour, bitter, sweet, and salty tastes. These were placed on the tongue, and adults were asked to identify the taste. Only 26% were able to correctly identify all four tastes. Another 26% correctly identified three of the four tastes. Nearly half of the middle-aged and older adults, 48%, had poor gustatory ability, making between two and four errors when identifying the four tastes.

Deficits in taste sensitivity may reduce the pleasure and comfort from food and thus represent risk factors for nutritional deficiencies. This decreased sensitivity may also present challenges to adhering to specific dietary regimens. The age-related decline in taste sensitivity is more pronounced for men compared to women.

Age-Related Changes in Olfaction

Age-related decreases in our sense of smell, olfaction, are also known as **presbyosmia**. Age-related changes in olfaction, like changes in gustation, may result as a consequence of biological aging, disease states (such as Alzheimer and Parkinson disease), medications, surgical interventions, and environmental exposure (Pinto, Wroblewski, Kern, Schumm, & McClintock, 2015). Although the sense of olfaction and gustation are closely linked, most studies suggested that the sense of smell is even more impaired than the sense of taste. Using data from the Sniffin' Sticks, the NSHAP study shows that olfaction declines at a rate of about one error for every

20 years. Racial differences exist, with African Americans' olfaction declining more rapidly than Whites' sense of olfaction. Gender, too, plays a role. Olfaction in men declines more rapidly than for women. It is important to note that these differences persisted, even when prior or existing socioeconomic status, health conditions, cognition, mental health, alcohol use, and smoking were considered. Researchers are continuing to examine the role of olfaction in cognitive disorders, as discussed in Box 2.3.

BOX 2.3

OLFACTION, PEANUT BUTTER, AND DEMENTIA

The popular media were excited. The general public was excited. A simple Internet search of "peanut butter" and "dementia" brings up more than 1.2 million hits. Here we discuss the original study that sparked the media attention, the solid science that prompted them to conduct that study, and additional research that help to answer the question: Can a peanut butter sniff test help diagnose dementia?

It has long been known that persons with dementia, such as Alzheimer's Disease (AD), exhibit sensory declines along with the hallmark cognitive challenges. In fact, disruptions in the sense of smell, hyposmia, might be among the very earliest warning signs. Although most older adults experience decreased olfactory sensitivity, these declines are even greater among persons with dementia (Albers et al., 2015; Murphy, Gilmore, Seery, Salmon, & Lasker, 1990). The left olfactory structures are physically closer to the parts of the brain most affected by dementia. Thus, in addition to general decrease in olfaction, it might be true that persons with dementia show differences in olfaction between the left and right nostril. This finding, along with other facts about the way that dementia influences cognitive processing, led a group of researchers to examine olfaction among persons with dementia and other forms of cognitive impairment, with the goal of developing an inexpensive but useful test to evaluate the presence of dementia.

Specifically, Stamps, Bartoshuk, and Heilman (2013) examined olfaction among 18 adults with AD, 24 adults with Mild Cognitive Impairment (MCI), 26 adults with non-AD dementia, and 26 controls without dementia. The materials were simple: 14 grams of peanut butter within a one-ounce container. Adults closed their eyes and mouth and were asked to hold one of their nostrils closed from the side, using their index finger. The container of peanut butter was placed on a 30 cm ruler (about one foot long) and moved toward the person's nostril in increments of 1 cm. The adult indicated at what distance they could smell the peanut butter. This procedure was repeated for the opposite nostril. The reported results were uniformly consistent: Each person with dementia showed a higher threshold for olfaction, meaning that the peanut butter needed to be closer to the nose before it could be detected. More important, the researchers reported that all of the persons with dementia showed a clear discrepancy between the left and right nostril before they could detect the odor. The left-right difference for persons with AD was more than 12 cm. About a 5 cm difference was present between the left and right nostrils for persons with non-AD dementia. People with MCI differed between nostrils by less than 2 cm and controls showed no left-right difference! The authors suggested that the peanut butter sniff test could be a reliable and inexpensive test for dementia.

Dramatic research results often capture the attention of the general public. The scientific community also takes notice when reported results are dramatic. Other research teams began the work of attempting to replicate the Stamps et al. (2013) finding. For example, Doty et al. (2014) repeated the

Stamps et al. study using the exact procedures with 15 adults with AD. Even though these adults were more impaired than the adults in the Stamps et al. sample, none exhibited the left-right discrepancy for identifying peanut butter. To rule out the possibility that asking a person to close their own nostril contributed to these results, Doty and colleagues used medical tape, rather than the index finger, to close off the nostril. Again, no left-right discrepancy was observed. Doty and colleagues then recruited an additional 20 adults with dementia to examine the possibility of a left-right difference in detecting up to 20 odors. Again, no such discrepancy was observed.

Doty et al. (2014) consider several important implications of the failure of the Stamps et al. (2013) results to replicate. First, they improved the procedures in several ways—they included persons who were more impaired than those participating in the Stamps et al. study. If there really is a left-right difference, one might expect larger discrepancies among the most impaired. But that did not occur. Second, Doty et al. expanded to other odors—no discrepancy was observed.

The fear of dementia is especially high among the general public, and the idea of an inexpensive assessment is attractive. Although the peanut butter sniff test is not likely to change the way we diagnose or assess dementia, scientific labs across the world are actively identifying new diagnostic tests, new pharmacological and behavioral treatments, and new supports for individuals and families of persons with dementia.

Age-Related Changes in Somesthesis

Sensitivity to touch, vibration, temperature, kinesthesis, and pain are collectively known as **somesthesis** (Hayslip et al., 2011). Age-related changes in such sensitivity can be attributed to a decreased number of sensory receptors for each sense and vary by the part of the body involved. For example, sensitivity of the feet starts to decrease at an earlier age than does that of the forearm.

Regarding touch, sensitivity appears to remain relatively stable throughout midlife. However, we experience an increase in absolute threshold, resulting in decreased sensitivity to some forms of touch, around age 50 to 55 years (Whitbourne, 1985). An especially important aspect of age-related change in our sense of touch is **thermal perception**, our sensitivity to heat and cold. Older adults are less able to regulate their own body temperatures and less able to respond to changes in ambient temperature. Thus, older adults may be especially susceptible to frostbite, heatstroke, burns, and other conditions related to temperature fluctuations (Saxon et al., 2014).

Finally, somesthesis also involves the **vestibular system**, with includes our sensitivity to balance and movement, sometimes referred to as kinesthesis. Age-related changes in the vestibular system can result in dizziness, vertigo, and other balance problems. Moreover, changes in our sense of balance may result in injuries and falls. Injuries can occur when we move too quickly or sharply through our environment. You may have noticed that when you are especially stressed, you might bump into walls or turn too quickly.

For older adults, that kind of dysregulation is likely to result in injury (Saxon et al., 2014). Older adults who fall are especially likely to experience severe bruising and broken ankles and hips. Falling is the leading cause of traumatic brain injury in older adults, and falls represent the most common cause of accidental death in older adults worldwide (WHO, 2018).

Age is a primary factor associated with both injuries and deaths from falls, with very young children and adults over age 65 years at a high risk. With the aging of the Baby Boomers, it is no surprise that the number of deaths from falls among older adults has increased. Estimates suggest that among adults aged 65 years and older, deaths from falls have increased by 30% over the past decade. If this trend continues, it is estimated that by the year 2030, seven older adults per hour will die from a fall (CDC, 2019b)! But there are many preventative efforts that one can enact to reduce falls: regular medication checkups to avoid side effects like dizziness, removing trip hazards like throw rugs, installing hand rails in bathrooms, and maintaining adequate lighting. Good health habits, including eating nutritious foods and engaging in strength training, especially for the lower torso, can also help. Preventing falls is such an important public health issue that the Centers for Disease Control and Prevention (CDC) has developed a special initiative to help health care providers to screen, assess, and intervene for fall risks. This program, Stopping Elderly Accidents, Deaths, & Injuries (STEADI) Initiative, provides many informational resources for physicians and the general public.

Specific Chronic Health Conditions

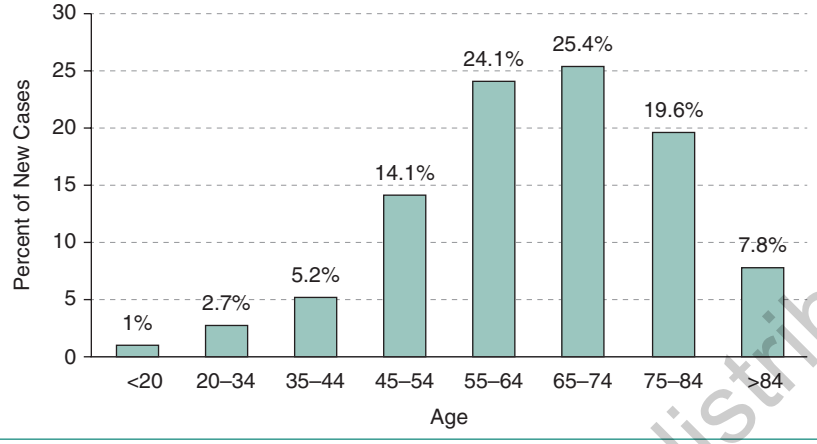
Most older adults live with one or more **chronic health conditions**, with about 77% having at least two. The most commonly experienced chronic health conditions in late life are heart disease, cancer, stroke, and diabetes. These four health conditions account for more than 60% of all deaths in the United States each year (National Council on Aging, 2018).

Heart disease is caused by the buildup of fatty plaques in the walls of the coronary arteries, a condition known as **atherosclerosis**. These fatty deposits impair blood flow and oxygen supply in the heart, which weakens the heart muscle. This blockage may also lead to chest pain (angina) or a heart attack (NIA, 2016).

Cancer refers to a collection of diseases in which the cells of the body fail to stop dividing and replicating (National Cancer Institute [NCI], 2015). In addition to specific environmental risk factors, advanced age is associated with an increase in cancer. Across all types of cancer, half occur in people older than age 66 years (NCI, 2015). Some cancers are more likely to be diagnosed at mid or late life, as shown by the median age of diagnosis. Figure 2.1, reprinted from the National Cancer Institute, shows the relation between age and site of new cancer diagnoses. The **median age** is the cut-point at which half of the diagnoses occur younger than and half occur at ages older than the stated age. Half of all diagnoses for breast cancer occur after age 61 years. For colorectal cancer, the median age of diagnosis is 61 years. Age 66 years is the median for prostate cancer diagnoses. Half of all lung cancer diagnoses occur after age 70 (NCI, 2015). For all cancers, regardless of age, good health habits and regular visits to health care providers can reduce risk and aid in early detection.

Stroke and **hypertension** are especially prevalent in the United States, with 90% of those over age 55 years being at risk. Gender differences are apparent, with 77% of women and 64% of men aged 75 years and older having hypertension (NIA, 2016). Hypertension is linked to both heart disease and stroke. Stroke occurs when the brain experiences a sudden lack of blood, through either blockage or ruptures. As

FIGURE 2.1 Percent of New Cancers by Age Group: All Cancer Sites



Source: "Age and Cancer Risk" was originally published by the National Cancer Institute, 2015.

the fourth leading cause of death, strokes lead to more long-term disabilities than any other chronic health condition in the United States (NIA, 2016).

Diabetes is a disease characterized by excessive levels of blood glucose. There are different types of diabetes, but in terms of aging, **type 2 diabetes (T2D)** and prediabetes are of special concern. More than 12 million adults aged 60 years and older have T2D, and another 57 million adults over age 20 years have prediabetes, increasing Americans' risks for heart disease, stroke, kidney disease, and diabetic retinopathy (National Institute of Diabetes and Digestive and Kidney Diseases [NIDDK], 2016). Lifestyle interventions involving maintaining a healthy body weight and engaging in regular physical activity are especially effective for reducing risk of developing T2D. Evidence shows that these health promotion behaviors can reduce T2D risk by more than 70% in adults over age 60 years (NIDDK, 2016).

Everyday Experience of Biological Aging

So far, we have discussed a variety of theories regarding why we age and effects of aging on various sensory systems, and we have discussed a few of the most common chronic health conditions at mid and late life. In the remaining sections of this chapter, we turn our attention to issues of longevity and the multiple causation mentioned by Baltes (1987) in his principles of life span development.

Life Expectancy and Life Span

How old is old? It seems like a simple question, but as Chapter 1 points out, the answer depends on historical period, social context, and individual perceptions. Because it defines upper limits, life span is an important contributor to our definitions of "younger" versus "older." **Life span** can be defined as the maximum age

TABLE 2.3 Life Span Across Species

Species	Maximum Life Span
Actinobacteria[1]	500,000 years
Glass sponges[2]	11,000 years
Humans[3]	125 years
Dog[3]	12 years
Horse[3]	25 years
House fly[3]	1 day

Sources: [1]Sample, 2010; [2]Max-Planck-Gesellschaft, 2012; [3]Shock, 1977.

that a member of a species can survive. Few members reach that limit, and different species have markedly different life spans (Hayflick, 2007). For example, humans are thought to have a maximum life span of around 125 years. The common house fly has a life span of about 5 weeks. For other variations in life span across species, see Table 2.3.

One factor that influences our view of "how old is old" is **life expectancy**. Life expectancy is defined as the average age a person can expect to live, based on environmental characteristics, lifestyle and behavioral choices, and genetic factors. Using empirical evidence, we can estimate an individual's life expectancy. There are a lot of life expectancy calculators available, but one of the best such calculators was developed by Dr. Thomas Perls.

As a physician, Perls approaches life expectancy using the same techniques that one uses to identify the precursors to disease, using a Patient Zero approach. Perls focused on **centenarians**, adults who reached their 100th birthday. By speaking with many people who were especially long-lived, Perls and his team were able to identify patterns in their family history and lifestyle habits (Andersen, Sebastiani, Dworkis, Feldman, & Perls, 2012).

You may have noticed that the Perls's life expectancy calculator used your current age to determine your estimated life expectancy. That is because life expectancy differs for different birth cohorts. In 1901, life expectancy at birth in the United States was 49 years; in 1950 it was around 68 years; in 2011, it was about 78 years; and in 2014, a baby born in the United States was expected to reach age 79 years. However, there are marked discrepancies in life expectancy within the United States. For example, an infant born on the Pine Ridge Indian Reservation in 2014 was expected to reach age 66 years. Infants born in Colorado could expect to live 86 years or more (Dwyer-Lindgren et al., 2017). Some of the lowest life expectancies in the United States can be found among residents of rural Appalachia, especially in eastern Kentucky, southwest West Virginia, parts of Alabama, and western Mississippi. These **place-based health disparities** are the result of multiple influences that challenge the economic, physical, and emotional well-being of adults (Allen & Roberto, 2016; Krout & Hash, 2015; Patrick, Nehrkorn-Bailey, Clark, & Marello, 2020).

TRY IT NOW

Take Dr. Perls's empirically based life expectancy calculator at https://www.livingto100.com/ to learn about the factors that influence your life expectancy. See Box 2.5 for a discussion of cohort differences in life expectancy at birth.

BOX 2.5

COHORT AND LIFE EXPECTANCY

Human life expectancy has shown a significant increase from the time of the ancient Greeks to the present. Life expectancy at birth in ancient Greece in 500 B.C. was 18 years, 25 years in ancient Rome (A.D. 100), and 35 years in thirteenth-century England. From 1775 to 1900 in the United States, the increase in life expectancy was from about 15 years to 35 years. The most significant increases in life expectancy in the United States occurred between 1900 and 1940; increases have been relatively minor since then. For example, for white males, during the 50-year period from 1900 to 1950, predicted life expectancy at birth increased by approximately 20 years (46.6 to 66.5 years). The increase in life expectancy since 1900 was due to improvements in housing, sanitation and hygiene (especially handwashing), antiseptics and antibiotics, public health laws, immunization for diseases, and nutrition. However, from 1950 to 1990, predicted life expectancy at birth increased by only about 5½ years (66.5 to 72 years). This relatively smaller increase relates to the primary causes of death among middle-aged and older adults. The primary causes of death for both men and women are chronic diseases, such as cardiovascular diseases and cancer, which, generally speaking, currently do not have a cure. These two diseases account for three fourths of all the deaths of older adults (Hayslip et al., 2011; Kastenbaum, 2006).

However, a new trend is emerging. For the first time, the current generation of children might expect to have shorter life expectancies than their parents. Three major contributors to this decrease in life expectancy are deaths from drug overdoses, chronic liver disease, and suicide (CDC, 2019a). These three diseases of despair are responsible for increasing numbers of deaths among those 25 to 64 years, especially in rural areas (Meit, Heffernan, Tanenbaum, & Hoffmann, 2017).

Racial and ethnic differences are also present in life expectancy at birth. For example, African Americans have an average life expectancy at birth equal to 74.6 years. Native Americans have a mean life expectancy of 76.9 years. White Americans have an average life expectancy at birth of 78.9 years. In contrast, Latinx Americans have a mean of 82.8 years, and Asian Americans have a mean life expectancy at birth of 86.5 years (SimplyInsurance.com, 2019).

In addition to birth cohort and geographic location, gender differences exist for life expectancy. But these gendered differences in life expectancy do not occur in isolation. That is, gender interacts with a host of other factors, including education and race. People who finish college generally live longer than those who do not

TABLE 2.4 Changes in U.S. Life Expectancy at Birth

	All Races			White Adults			Black/African Americans			Hispanic Adults		
Birth Year	**Both**	**Men**	**Women**	**Both**	**Men**	**Women**	**Both**	**Men**	**Women**	**Both**	**Men**	**Women**
1900	47.3	46.3	48.5	47.6	46.6	48.7	33.0	32.5	33.5	--	--	--
1950	68.2	65.6	71.1	69.1	66.5	72.2	60.8	59.1	62.9	--	--	--
2000	76.8	74.1	79.3	77.3	74.7	79.9	71.8	68.2	75.1	--	--	--
With a consideration for Hispanic/Latinx ethnicity												
2012	78.8	76.4	81.2	78.9	76.5	81.2	75.1	71.9	78.1	81.9	79.3	84.3
2013	78.8	76.4	81.2	78.8	76.5	81.2	75.1	71.9	78.1	81.9	79.2	84.2
2014	78.9	76.5	81.3	78.8	76.5	81.2	75.3	72.2	78.2	82.1	79.4	84.5
2015	78.7	76.3	81.1	78.7	76.3	81.0	75.1	71.9	78.1	81.9	79.3	84.3
2016	78.7	76.2	81.1	78.6	76.2	81.0	74.9	71.6	78.0	81.8	79.1	84.3
2017	78.6	76.1	81.1	78.5	76.1	81.0	74.9	71.5	78.1	81.8	79.1	84.3

Sources: Adapted from Arias & Xu, 2019; SimplyInsurance, 2019; Woolf, S. H. & Schoomaker, H., 2019.

complete high school. Across educational levels, women outlive men. When we factor in race, White women tend to live longer than Black/African American women, at least until age 85. After age 85 years, Black women show a slight longevity advantage. In contrast, Hispanic adults born in the United States tend to outlive their White and African American peers. It is important to remember, however, that just as there are many ethnic and cultural variations within White or Black groups, Hispanic Americans are also a diverse group. For example, recent Hispanic immigrants to the United States may expect to obtain a significantly lower life expectancy than U.S.-born Hispanic adults (Olshansky et al., 2012). As you read through Table 2.4, identify the public health, political, and other social conditions that influence the changes in life expectancy across race, gender, and birth cohort.

From Theory to Application: Life Extension and Health Span

We have known for more than 80 years that **caloric restriction (CR)** increases life expectancy in short-lived animals (Anderson et al., 2018). Studies of whether CR increases life expectancy in primates have found that CR may be associated with a compression of disease but might not increase the number of years that one survives (Hayslip, Patrick, & Panek, 2011). For more discussion of this issue, see Box 2.6.

BOX 2.6

HEALTH SPAN

At the start of this chapter, we posed the question, "How old is old?" Your answer is important because it reflects your knowledge and attitudes about aging in general, and it influences how long you might want to live. An emerging construct, **health span**, may also influence your answer. Although there is no agreed-upon definition, or indeed even a scientifically sound way to measure it, health span can be defined as the period of time in which one lives (relatively) disease free and functions at optimal levels (Kaeberlein, 2018). Using this definition, the individuals over 100 years old studied by Perls and his team often showed great similarity between their life expectancy and their health span—they were living relatively healthy lives until the last few years (Andersen et al., 2012). Compression of disease may be an important component of health span.

Most Americans in the United States and Canada say that they do not want to live beyond age 80 or 85 years, and few have knowledge about current life extension science (Pew Research Center, 2013). In a recent survey, Donner et al. (2016) asked 1,000 adults about whether they would like to live to ages 85 years, 120 years, 150 years, or indefinitely. If conditions were such that an individual could be guaranteed mental and physical youthfulness and vitality, almost 80% responded that they would prefer to live to age 120 years or longer. More than half (53.1%) said that with sustained vitality, they would choose to live indefinitely. Adults who previously identified a preference for a lower life expectancy changed their responses to 120+ years when the option of sustained vitality was mentioned. Health span seems to matter to adults in North America. Not surprisingly, adults who were more scientifically minded were especially likely to prefer a long and vital life.

However, there are difficulties related to translating lab studies with rats, mice, worms, and flies to humans. Notably, human CR usually is not initiated until adulthood, whereas animal studies may begin much earlier in life, even before birth. Humans live 75+ years, much longer than rats (3 years) or flies (a few weeks). Human ethics procedures also include the right to decline invitations to participate in research, as well as the right to withdraw one's consent to participation. Animal models do not include those particular aspects. Finally, although there are many similarities between different animal species and humans, there are differences as well.

Possible benefits of CR for humans first came to the public's attention more than 3 decades ago, with a 1983 book, *Maximum Lifespan*, and other writings by noted researcher and physician Roy Walford. Walford was part of the Biosphere Project and was able to gather evidence that CR, reduced caloric intake accompanied by high nutritional value, could result in improved health among humans (Walford, 1986). Even though Walford was a recognized expert, his small sample size observations included humans who wanted to try CR; thus, they might have differed in many dimensions from other adults, including being science-minded and having high self-control.

More modern research in CR is following clinical trial protocols. In these protocols, people are randomly assigned to different conditions, and they are evaluated many times over longer periods, using a variety of methods and assessments. Ravussin et al. (2015) reported results from the first 2 years of human trials with CR. This study follows more than 200 normal-weight adults, aged 21 to 50 years, who were randomly assigned to one of two conditions: a control group who ate their normal caloric intake across the 2-year period, referred to as the ad litum group, and the Caloric Restriction (CR) group, who consumed 25% fewer calories than their preferred intake, with careful attention to the nutrition intake of that reduced calorie diet. In the first 2 years, adherence was impressive, with 82% of those in the CR group and 95% who were in the Control group completing the study. Adults in the CR group lost and maintained a weight loss around 10%, equivalent to about 16 pounds, most of which was fat. No such changes were observed for the Control group. Of course, it is not surprising that adults who eat less than they prefer to eat will lose weight. Other health changes were observed, however. At year 1, but not year 2, those in the CR group had lower resting metabolic rates and lower body temperatures than the Control group. Although the study continues, it is among the best-designed studies to demonstrate that CR is feasible in typical adults.

Other researchers argue that additional factors contribute to the benefits of CR beyond simply reducing caloric intake while maintaining high nutrient density. These researchers focus on *when* one ingests calories. For example, Di Francesco, Germanio, Bernier, and de Cabo (2018) and Mattson (2014) lay out convincing evidence regarding the mechanism whereby CR influences health and argues that the benefits can be increased through **Intermittent Fasting (IF)**. IF, in which one restricts the number of hours per day or even the number of days per week in which one eats, increases health span by decreasing our risks of inflammatory-related diseases, such as Alzheimer's, Parkinson's, and cardiovascular diseases. Reducing the preferred number of calories while maintaining high nutrient density is associated with a host of benefits that increase the quantity (life expectancy) and quality

(health span) of life. These benefits are not merely due to the effects of reduced caloric intake on obesity, cardiovascular disease, diabetes, and hypertension, however (Di Francesco et al., 2018). Specific systems of the body are healthier when adults engage in CR and IF. Benefits are seen in the blood, liver, intestine, brain, cardiovascular system, pancreas, and a decrease in adipose tissue.

Benefits related to energy expenditure also contribute to the improved health and functioning. Specifically, Di Francesco and colleagues (2018) highlight systemic benefits of IF that include the following:

1. Improved tissue repair and reduced damage from oxidative stress (free radical damage)
2. Improved function due to decreased inflammation
3. Improved metabolic homeostasis via improved protein synthesis
4. Healthier mitochondria

Integrating Across Topics

Many of the early explanations of biological aging did not address the group differences we observe in life expectancy or health span. Early sociological theories (see Table 2.5) examined the social context of health and longevity, noting that Black Americans experienced the onset of age-related diseases about 10 years earlier than their White peers.

TABLE 2.5 Early Theories Regarding Race Differences in Longevity and Racial Health Disparities

Theory	Summary
Multiple Jeopardy	Being a member of a racial or ethnic minority (and lower socioeconomic status; and female) and old combine in an additive or multiplicative way to create health disparities
Age-as-Leveler	The effects of aging are so profound that race differences are evened out
Persistent Inequality	The racial differences in health are observed in earliest childhood and simply continue into old age
Age-as-Survival	Older minorities may be healthier than older Whites because only the healthiest minorities survive to late life and because older minorities have gained coping strategies to deal with a lifetime of discrimination
Cumulative Dis/Advantage	As stated in Chapter 1, the idea that a lifetime of stresses and discrimination result in health challenges that persist and decrease one's reserve capacity

Sources: Adapted from Dowd & Bengtson, 1978; Ferraro & Farmer, 1996.

Thus, it was not surprising that Black adults had shorter life expectancies relative to White adults. Take another look at Table 2.4. Although in the United States, Black life expectancy has increased dramatically in the past 60 years, there is still room for progress. Drawing across different disciplines and integrating the social and applied sciences' focus on social and environmental resources and biology's focus on geroscience, we have a better understanding of the how **socioeconomic position (SEP)** factors influence health across the life span. For example, Glymour, Ertel, and Berkman (2009) pose five models describing how the **timing of risk exposure** may influence development:

1. The immediate effect model looks to identify immediate causes for effects, with the assumption that once a risk factor is removed, symptoms or functioning should change. Scientists can test this model by using designs in which a person serves as her own statistical control. For example, if we were interested in studying how caffeine leads to an increase in blood pressure, we could test blood pressure before, during, and after ingestion of caffeine. We assume that once the caffeine was out of person's system, their blood pressure would return to its baseline measure.
2. A social trajectory model suggests that exposure to certain factors creates a persistent pathway of exposure to other risk factors. For example, Glymour and colleagues discuss the influence of lower educational attainment. Lower education levels are associated with lower paying jobs, which in turn affect the foods one eats, where one lives, and the general environmental conditions, such as pollution and crowding. Thus, the effects on health under a social trajectory model may persist across the life span or may be able to be disrupted at some point.
3. Cumulative models adhere to the idea that risk exposure at each stage of development may cause a cascading of effects, because not only are social trajectories altered, but the risks directly alter the physiological hardiness of the person. Thus, early exposure may weaken a person's **reserve capacity** such that they are less able to deal with future illnesses or diseases.
4. Sensitive Period models suggest that the effects of exposure to specific risks may be magnified or minimized depending on when a person experiences the exposure. For example, Ingber and Pohl (2016) present data with lab animals' exposure to methylmercury (MeHg) which highlight the need to distinguish effects based on the timing of exposure, dose of exposure (including doses for specific systems), differences in effects across species, and the effects' specific mechanisms of action (i.e., genetic mutations, chromosome damage, chemical imbalances).
5. Physiological effects of trajectory models focus explicitly on how the *changes* in risk factors that are present in the environment lead to later disparities. These kinds of models examine how the magnitude and direction of risk exposure influences health over time. Thus, they focus on the severity of adverse events, not just their mere presence.

Summary and Conclusion

Biological aging is a rich and active area of geroscience. Good theories help us to understand the complexities of aging as we continue to discover the mechanisms by which aging influence the systems and cells of our bodies. A focus on the specific sensory changes and chronic health conditions experienced by older adults prompts us to focus on solutions to these issues. Moreover, good theory and data are helping us to understand differences in racial, ethnic, and place-based health disparities that may help to even out differences in life expectancy.

TERMS

Absolute threshold 21
Accommodation 21
Adaptation 23
Age-Related Macular Degeneration (AMD) 25
Atherosclerosis 30
Audition 20
Autoimmune theory 20
Biological aging 17
Biomarkers 19
Caloric restriction (CR) 35
Cancer 30
Cataracts 24
Centenarians 32
Chronic health conditions 30
Color vision 23
Dark adaptation 23
Diabetic retinopathy 25
Dynamic visual acuity 23
Error theories 18
Free radical theory 20
Generalizable 18
Genetic-programming theories 18
Glare 23
Glaucoma 21
Gustation 20
Hayflick Limit 17
Health span 35
Heart disease 30
Heuristic value 18
Hypertension 30
Hypogeusia 27
Hypothalamic-pituitary-adrenal (HPA) axis 19
Immunological theory 19
Intermittent fasting (IF) 36
Life expectancy 32
Life span 31
Median age 30
Neuroendocrine theory 19
Olfaction 20
Parsimony/parsimonious 18
Peripheral field 24
Place-based health disparities 32
Presbycusis 26
Presbyopia 21
Presbyosmia 27
Programmed senescence 19
Reserve capacity 38
Sensory aging 20
Socioeconomic position (SEP) 38
Somesthesis 20
Static visual acuity 21
Stroke 30
Taste buds 27
Telomeres 19
Thermal perception 29
Timing of risk exposure 38
Type 2 diabetes (T2D) 31
Useful Field of View (UFOV) 24
Variable-rate theories 18
Vestibular system 29
Vision 20
Visual field 24
Wear-and-tear 19