

SECTION 1

BASICS OF GERIATRIC CARE

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1 BIOLOGY OF AGING

***Aging:** A process of gradual and spontaneous change, resulting in maturation through childhood, puberty, and young adulthood and then decline through middle and late age.*

***Senescence:** The process by which the capacity for cell division, growth, and function is lost over time, ultimately leading to an incompatibility with life; ie, the process of senescence terminates in death.*

Although aging has both the positive component of development and the negative component of decline, senescence refers only to the degenerative processes that ultimately make continued life impossible. Not all of the changes that occur with age—even those that occur in late life—are deleterious (eg, gray hair, baldness), and some may even be desirable (eg, increased wisdom and experience). The age-related increase in insulin levels and body fat that occurs in late life may be beneficial when available nutrition is limited. In contrast, the memory impairment that occurs with age is considered senescence. Senescence has no positive features.

Differentiating between normal aging and successful aging is useful. **Normal aging** refers to the common complex of diseases and impairments that characterize many of the elderly. However, persons age very differently: some acquire diseases and impairments, and others seem to escape specific diseases altogether and are said to have died of old age. The latter may maintain an active healthy life until death.

Successful (healthy) aging refers to a process by which deleterious effects are minimized, preserving function until senescence makes continued life impossible. Persons who age successfully avoid experiencing many of the unwanted features of aging. For example, they may avoid near-total tooth loss, which used to be (and is in some societies) usual and universal among the elderly. The elderly may be able to avoid the complications of vascular disease, even while the circulatory sys-

tem continues to age, by controlling blood glucose levels and body fat percentage.

The concept of successful aging is that aging is not necessarily accompanied by debilitating disease and disability. Although the percentage of persons > 65 and the proportion of the elderly > 85 have both increased in the USA, the percentage of elderly persons residing in nursing homes has decreased (to 5.2%). Similarly, the percentage of persons aged 75 to 84 who report disabilities has decreased (to < 30%), as has the percentage of persons with debilitating disease. Although there may be alternative explanations for these changes in health status, one viable explanation is an increase in the proportion of persons who are aging successfully.

Disease vs. aging: In both aging and senescence, many physiologic functions decline, but normal decline is not usually considered the same as disease. The distinction between normal decline and disease is often but not always clear and may be due only to statistical distribution. Glucose intolerance is considered normal aging, but diabetes is considered a disease, although a very common one. The incidence and prevalence of type II diabetes increase with age, so that among persons > 75 years of age, > 10% have diabetes. Cognitive decline is nearly universal with advanced age and is considered normal aging; however, cognitive decline consistent with dementia, although common in late life, is considered a disease. Alzheimer's disease is a pathologic process distinct from normal aging, a conclusion supported by analysis of brain tissue at autopsy.

LONGEVITY

The average life span of Americans has been increasing dramatically since the industrial revolution. However, most of the gains resulted from decreasing childhood mortality. The maximum life span, generally determined to be about 125 years for women and somewhat shorter for men, has changed little in recorded history, although some experts suggest that it may be slowly increasing.

Several factors influence longevity. One is heredity. Heredity primarily influences whether an individual will contract a disease. Inheriting a propensity to hypercholesterolemia is likely to result in a short life, whereas inheriting genes that protect against heart disease and cancer helps ensure a long life. Medical treatment contributes to increased survival after diseases are contracted, especially when diseases (eg, infectious diseases, cancer) are curable. Another important influence on longevity is lifestyle; avoiding smoking, maintaining a healthy weight and diet, and exercising appropriately help people avoid disease. Exposure to environmental toxins can shorten life span even among people with the most robust genetic makeup.

CELLULAR AND MOLECULAR AGING

Cells lose their ability to divide over time unless they become cancerous. This limit to cellular replicative capacity (Hayflick's limit or phenomenon) can be demonstrated in fibroblasts removed from the umbilical cord of newborns and cultured *in vitro*. The fibroblasts divide only until they are dense enough to contact each other—a phenomenon called contact inhibition. If diluted, the fibroblasts divide again until maximum density is reached. This process can be repeated; however, after about 50 divisions, the fibroblasts stop dividing regardless of their density. Hayflick's limit is thought to reflect *in vivo* processes; fibroblasts removed from elderly persons tend to divide fewer times. Studies have shown that the loss of replicative capacity does not depend on the total amount of time cells are cultured (chronologic age) but on the number of divisions (biologic age).

When cells divide so many times that they cannot divide again, they enlarge and exist for some time before gradually dying. Such cells differ in morphology and function from young cells that are still dividing and from young cells whose division has been arrested by experimental manipulation.

One biologic mechanism for Hayflick's limit is now understood. Telomeres are stretches of DNA at the end of chromosomes that serve as handles by which chromosomes are moved during the telophase of meiosis. Telomeres are irreversibly shortened each time a cell divides. When the telomeres become too short, the cell can no longer divide.

In transformed (eg, cancerous) cells, the enzyme telomerase lengthens telomeres after telophase. The telomeres of transformed cells do not shorten after each division, and thus the cells become immortal, dividing far beyond Hayflick's limit. Normal postmitotic cells (except for fetal and germ cells) express telomerase in very small amounts, and their telomeres become shorter after each cell division.

The relevance of Hayflick's limit to senescence of the whole organism is unclear. Although some cells (eg, intestinal epithelial cells, skin fibroblasts) divide more or less continuously throughout life, they are unlikely to approach the limit of 50 divisions. Even if they did, the cells most likely to cause functional failure during senescence are probably those that divide very little (immune and endocrine cells) or not at all (neurons and muscle cells). Furthermore, senescence in metazoans composed entirely of postmitotic cells is just as predictable and robust as that in metazoans containing mitotic cells.

Mechanisms other than telomerase shortening may be involved in senescence. For example, messenger RNA (mRNA) transferred from senescent cells into young cells stops cell division in the young cells. The mRNA acts as a gerontogene (a gene mutation that increases life span), whose function may resemble that of a tumor suppressor gene (eg, *p53*). Mutations in *p53* lead to uncontrolled cell division, cancer,

and often death of the organism. Mutations in gerontogenes extend the number of divisions in cells.

Necrosis and apoptosis: Cell death may occur by necrosis or apoptosis. Necrosis is due to physical or chemical insults (eg, metabolic inhibition, ischemia) that overwhelm normal cellular processes and make the cell nonviable. In necrosis, loss of ion gradients across the cell membrane leads to an influx of calcium and other ions, which triggers proteolysis and rupture of organelle membranes. Necrosis is a purely entropic phenomenon due to loss of the cell's ability to transform external energy.

In contrast, apoptosis is a highly regulated, orderly process by which a cell essentially commits suicide; usually, the stimulus for apoptosis is a physiologic signal or a very mild insult. A defining feature of apoptosis is the fragmentation of the cell's DNA, produced by a regulated activation of deoxyribonuclease. However, several other biochemical processes that also lead to cell death are simultaneously induced. Apoptosis is essential for normal development and remodeling.

Apoptosis has been implicated in several age-related diseases, including Alzheimer's disease. Whether age-related cell death is due primarily to necrosis or to apoptosis affects whether aging is considered the result of entropic processes (if due primarily to necrosis) or of relatively simpler, more regulated processes (if due primarily to apoptosis).

THEORIES OF AGING

There are more theories of aging than facts. Aging clearly occurs at different rates for different species, and even within a species, aging occurs at different rates among different individuals. The only reasonable conclusion is that aging must be genetically controlled, at least to some extent. Both within and between species, lifestyle and exposures may alter the aging process.

Most gerontologists view senescence as a collection of degenerative entropic processes related only by the fact that they occur over time. Some theories of aging address what controls these processes and why the controls exist as they do. Other theories of aging address the issues of whether senescence is more programmed than random entropy, thus offering some advantage for a species. For example, senescence may have evolved because without it, a species would accumulate ill-adapted older members. These members would compete with potentially better adapted younger members, slowing the rate at which adaptive mutations are introduced.

Loose cannon theory: This theory posits that an entropy-producing agent—free radicals or glucose—slowly disrupts cellular macro-

molecular constituents. Theoretically, free radicals, generated during oxidative phosphorylation, can variously modify macromolecules, primarily through oxidation. Considerable evidence suggests that oxidative damage increases with age. For example, in older organisms, specific amino acids in specific proteins tend to be oxidized residues, leading to decreases in the specific activity of these proteins. Additionally, specific oxidized derivatives of nucleotides from DNA increase in frequency. Experimentally induced simultaneous overexpression of superoxide dismutase and catalase (enzymes that attenuate free-radical damage) increases the life span of fruit flies by about 30%.

Glucose is thought to promote senescence mainly through nonenzymatic attachment to proteins and nucleic acids, through the same process that produces glycated hemoglobin. Glycated protein levels increase with age. Otherwise, there is little direct evidence that glycation has a major role in senescence. However, because dietary restriction increases maximum life span and also reduces blood glucose and the rate of glycation, interest in glycation's role in senescence continues.

Rate of living theory: This theory posits that smaller mammals tend to have high metabolic rates and thus tend to die at an earlier age than larger mammals. Thus, this theory is related to the idea that free radicals and other metabolic by-products play a role in senescence. However, studies of metabolic rates have shown wide variation in the correlation between size and longevity, undermining the credibility of this theory.

Weak link theory: This theory posits that a specific physiologic system—usually the neuroendocrine or immune system—is particularly vulnerable (presumably to entropic processes) during senescence. Failure of the weak system accelerates dysfunction of the whole organism. Failure of the neuroendocrine system would be expected to produce profound impairments in homeostatic systems, including loss of reproductive function and metabolic regulation, which occur with age. Failure of the immune system would be expected to produce an increased susceptibility to infection and a decreased ability to reject tumor cells. However, there is little evidence that failure of either system directly contributes to age-related diseases or to mortality (in contrast, for example, to the direct contribution of a compromised immune system to mortality in patients with AIDS). Furthermore, even if this theory explains some manifestations of aging in higher organisms, it does not explain aging in lower organisms, and little is known about the primary mechanism behind such weakness.

Error catastrophe theory: This theory posits that errors in DNA transcription or RNA translation eventually lead to genetic errors that promote senescence. Although data suggest that older organisms have

altered proteins reflective of such genetic changes, this theory does little to explain most observed age-related changes.

Master clock theory: This theory is one of the oldest theories of aging and no longer has high credibility; it states that aging is under direct genetic control. Teleologically, it suggests that the rate of aging within each species has developed for the good of each species. Individual variation develops because of maladaptation, exposure, and lifestyle. In the wild, such maladapted individuals tend to die out and the well-adapted ones persist, altering longevity in the best interest of the species.

Exactly what controls the rate of aging is unknown. It could be a gene that controls telomere shortening or some other process of cell division. Or it could be genetic control of another cellular process not involved in division, such as DNA repair, thus resulting in apoptosis.

DISEASES OF ACCELERATED AGING

Progeroid syndromes: In progeroid syndromes, which are rare, children exhibit several features similar to those normally observed in the elderly. These include baldness, osteoporosis, and dry, wrinkled skin. However, progeroid syndromes also include features that differ from normal senescent changes, such as lack of gonadal activity and unusually short stature. Thus, progeria is not exactly a model of accelerated aging.

Werner's syndrome produces sclerodermal skin changes and baldness, which make affected children appear elderly, producing an immediate sense of premature aging. Other features include premature cataracts, muscular atrophy, glucose intolerance, a high incidence of cancers (some of which are rare in unaffected persons), and early death due to atherosclerosis. However, the central nervous system is largely spared. The gene involved in Werner's syndrome codes for a DNA helicase, an enzyme that unwinds DNA to allow replication and possibly transcription. This discovery has led to speculation that during normal senescence, many age-related impairments may result from impairments in the helicase mechanism, although this hypothesis has not been studied systematically.

Wiedemann-Rautenstrauch syndrome and **Hutchinson-Gilford syndrome** also produce premature scleroderma, baldness, and other senile pathologies in children. The genetic basis of these syndromes remains undetermined.

Down syndrome: More common than the progeroid syndromes, Down syndrome also produces pathologies typical of senescence, including glucose intolerance, vascular disease, a high incidence of cancers, hair loss, degenerative bone disease, and premature death. In contrast to Werner's syndrome, Down syndrome greatly impairs the central nervous system, usually producing retardation and accelerating the accretion of neuritic

plaques and neurofibrillary tangles characteristic of Alzheimer's disease. Because Down syndrome results from duplication of all or a small part of chromosome 21, its cause was originally thought to be duplication of the gene for β -amyloid. Mutations of this gene, located on chromosome 21, have been implicated in Alzheimer's disease. However, although the duplicated region that produces Down syndrome is near the β -amyloid gene, the best evidence suggests that Down syndrome may occur without duplication of the β -amyloid gene. The specific gene or genes involved in Down syndrome remain undetermined.

2 DEMOGRAPHICS

The statistical characteristics of human populations.

As the geriatric population grows in the USA and worldwide, demographics is an important tool in the development of policies on aging.

U.S. DEMOGRAPHICS

POPULATION CHARACTERISTICS

Between 1900 and 1990, the total U.S. population increased three-fold, while the population of persons ≥ 65 years increased tenfold. In 1990, more than 31 million Americans were ≥ 65 , nearly twice as many as in 1960 (see TABLE 2-1). This number is estimated to reach almost 35 million by 2000, > 53 million by 2020, and > 75 million by 2040.

TABLE 2-1. ACTUAL AND PROJECTED GROWTH OF THE ELDERLY POPULATION*

Year	Total Population (all ages)	≥ 65 Years		≥ 85 Years	
		Number	% of Total	Number	% of ≥ 65
1960	179,323	16,560	9.2	929	5.6
1980	226,546	25,550	11.3	2,240	8.8
1990	248,710	31,079	12.5	3,021	9.7
2000	274,634	34,709	12.6	4,259	12.3
2020	322,742	53,220	16.5	6,460	12.1
2040	369,980	75,233	20.6	13,552	18.0

*Numbers in thousands.

From Day JC: "Population projections of the United States, by age, sex, race, and Hispanic origin: 1995 to 2050." *Current Population Reports Series P25*, No. 1130. Washington, DC, U.S. Government Printing Office, 1996.