*When a Bavarian artist did this woodcarving of people seeking rejuvenation in a fountain of youth some 460 years ago, lives were short. Not today. As of 1980, the average newborn American can expect to live 70 years if male, almost 78 if female. Those who reach the age of 65 have an even greater average life expectancy: close to 79 years for men and 83 for women.*

# The Elderly in America

In "Old Age," an essay that he wrote in 1870, Ralph Waldo Emerson, 67, lamented that "America is a country of young men, and too full of work hitherto for leisure and tranquillity."

Emerson thought that Americans ignored the "particular benefits" of age, especially the value of experience. For youth, he said, "every object glitters and attracts," and life is apt to be "a heap of beginnings" with little result. The elderly are different: "Age sets its house in order, and finishes its works, which to every artist is a supreme pleasure."

In many ways, the extent of that pleasure is a measure of civilization. In poor, primitive tribes, older people who can no longer provide for themselves may be treated harshly. Eastern cultures, and those that nurtured the Judeo-Christian tradition, tend to venerate age. The Arabic term *sheikh* (leader) originally meant "elder." The Old Testament declared that "a hoary head is a crown of glory" and awarded patriarchs including Methuselah long lives exceeding 900 years. In ancient Greece, where most did not reach even their thirtieth year, the Spartans were ruled by a *gerousia*, a council of elders over 60. At first, the Roman Senate was made up of retired magistrates.

In modern times, the role of elders as anchors of society and family, for better or worse, has been a recurrent theme of the literature of many nations—Leo Tolstoi's *The Death of Ivan Ilyich* (1886), Thomas Mann's *Buddenbrooks* (1901), many of Victor Hugo's novels, where sage elders, such as Jean Valjean in *Les Misérables* (1862), find an "unspeakable dawn in happy old age."

The reality of such portraits is now being tested as never before: Thanks to improvements in public health, medicine, and hygiene, long life has become a mass phenomenon. For most of history, that was rare. In 17th-century France, for example, half of those who survived birth died before reaching the age of 20. As recently as 1900 in America, the average life expectancy was only 47 years. But today it is more than half again as long.

This considerable advance has largely taken place during the past 60 years—that is, mostly during the lives of today's

depression-born elderly. Since 1900, the ranks of the aged in America have grown from 3,080,000, or four percent of the U.S. population, to 27,384,000, or 12 percent.

Their numbers will double again as the Baby Boomers born in the 1950s start to retire. The graying of the nation is, of course, fraught with social and political implications. Even now, for example, programs supporting the aged absorb 27 percent of the federal budget, about the same as the Pentagon. Meanwhile, gerontologists are narrowing down the exact causes of aging—more in hopes of further extending *active* life, than of extending the life span per se.

Here, Albert Rosenfeld summarizes the latest research on the aging process, and Timothy M. James surveys the culture of the elderly; historian W. Andrew Achenbaum traces the rise of the U.S. government's commitment to the aged.

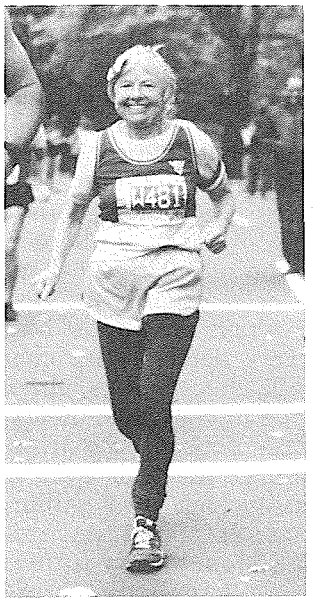
# STRETCHING THE SPAN

*by Albert Rosenfeld*

Can we start senescence—the aging process—on its way to obsolescence?

At any previous moment in scientific history, that would have been a ridiculous question. Yet the virtual abolition of old age as we have always known it has become the goal of a number of working gerontologists—specialists in the study not just of geriatrics, the infirmities of the human elderly, but of the aging process itself, in all species.

The possibility of extending our years of useful vigor still does not attract much serious public discussion. We have learned to scoff at all the failed attempts, from before Juan Ponce de León's 16th-century quest for the Fountain of Youth in Florida to the purveyors of "monkey glands" and other frauds in modern times. And for all the present American interest in fitness, many adults remain persuaded that "devoting one's life to keeping well is one of the most tedious of ailments," as the 17th-century French essayist La Rochefoucauld put it.



*People just do not run down as they used to. When Madge Sharples, a 65-year-old competitor in the 1981 New York Marathon, was born, the average American newborn could be expected to live only to about age 52.*

The gerontologists' goal—to permit the individual "to die young as late as possible"—is apt to be dismissed as just another tiresome sign of what social critic Christopher Lasch has called our "culture of narcissism."

Nonetheless, we are approaching a more detailed understanding of senescence. Scientists have long established that there is nothing immutable about the life span of a given species. This was proven during the 1930s, when Clive McCay of Cornell showed that rats would live a third longer if their diet were kept balanced but held to near-starvation levels. Today, gerontologists believe that progress toward interfering with the aging process can and is being made.

While nothing like an anti-aging vaccine is in prospect, the mechanism of aging is becoming clearer, thanks to the rapid advance since the 1930s of gerontology and its associated sciences—biochemistry, cell biology, molecular genetics, immunology, endocrinology, and the neurosciences.

The effects of aging are familiar: the progressive loss of hair and teeth, the wrinkling and shrinking, the stoop and shuffle, the fading of hearing and sight. Inside, the lungs' maximum capacity declines (by 40 percent at age 80), and the heart pumps less efficiently as accumulated cholesterol and other debris gather on artery walls. The defenses against infection and stress

begin to wither away, connective tissue stiffens, the sex urge becomes less insistent, and the memory less reliable.

The standard charts illustrating this decline usually begin at age 30, the presumed peak of health and vigor. But most gerontologists now agree that, physically, the peak years end during the early twenties; some argue that they end at puberty. The chief debate now centers on *how* aging takes place.

Even gerontologists used to joke that there were as many theories of aging as there were people seriously studying it. Given the number and diversity of "events" that occur in the body's cells, organs, and systems, the directions that investigation can take are almost limitless. In an aging organism, virtually every deteriorative change can prompt other types, in a "cascade effect." Thus, almost any type of aging change can be parlayed into a whole theory of senescence.

Most of the theories involve (*a*) a process of "wear and tear" on the human machine over time, or (*b*) the idea that we have "clocks" ticking away within us that are *genetically* programmed to dictate the manner and rate at which we age and die.

But if one theory turns out to be right, the others need not be wrong. A unified view of aging is slowly emerging that may encompass just about all of the theories, each explaining part of the process.

There are several wear-and-tear theories:

■ *"Garbage" accumulation.* As cells age, they have a harder time disposing of their wastes. Some of this "garbage" is a fatty substance called lipofuscin, which accumulates especially in those cells that in adulthood no longer divide, such as brain and muscle cells. Eventually, lipofuscin may take up as much as 20 percent of a cell's available space. Think of the cell's working molecules as waiters in a nightclub trying to get across a dance floor that grows increasingly crowded: Service would get slower and finally might come to a standstill. Most gerontologists now view this phenomenon as a result, rather than a cause, of aging.

■ *Cross-linkage.* The body has many large molecules that perform indispensable functions. The so-called blueprints of life, the genes, are made up of molecules of the nucleic acid DNA

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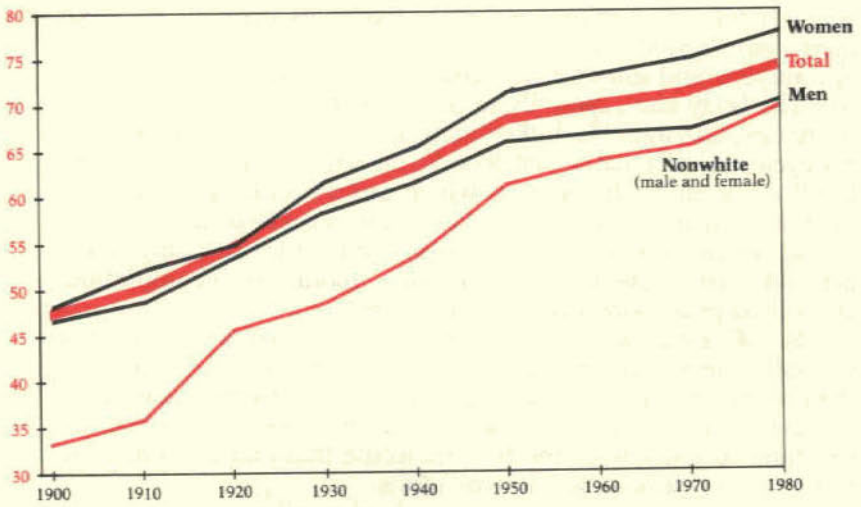
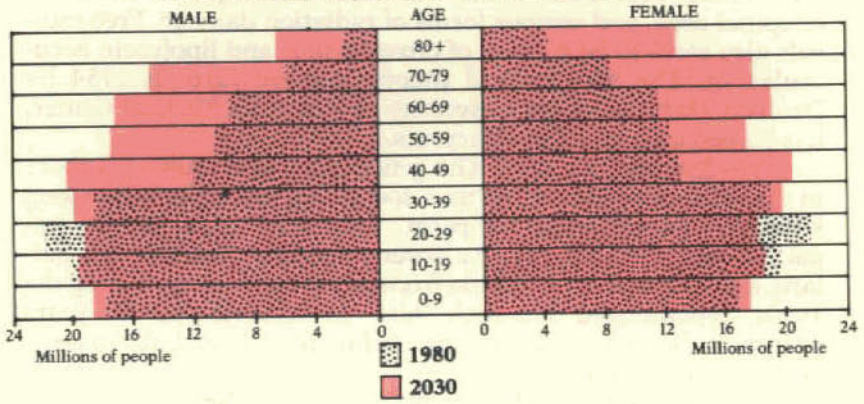
*Albert Rosenfeld, 64, is consultant on future programs for the March of Dimes and an adjunct professor at the University of Texas Medical Branch in Galveston. Born in Philadelphia, he received his B.A. degree from New Mexico State University in 1950. A former science editor of* Life *and the* Saturday Review, *he is currently preparing a new edition of his 1976 book on aging research,* Prolongevity.

## RISING LIFE EXPECTANCY IN THE U.S.




## A GRAYING POPULATION: 1980 AND 2030




*America's 55-percent rise in average life expectancy since 1900—a rise of 26 years—nearly equals the gain achieved in all the previous 3,000 years. During the 1960s and 1970s, the 65-plus age group grew more than twice as fast as the whole population; the 75-plus category is the fastest rising of all. Partly as a result, the U.S. median age, just 23 in 1900, has reached 31 and may surpass 33 by 1990. The female edge in longevity, 7.5 years in 1970, has stopped growing, perhaps because more women are holding jobs.*

Sources: National Center for Health Statistics, U.S. Department of Health and Human Services; Bureau of the Census.

(deoxyribonucleic acid); other sizable molecules are proteins, including hormones and enzymes, and those that make up cartilage, tendons, and other connective tissue. As the cells go about their complicated business, these molecules keep bumping into one another and sometimes become attached—cross-linked.

The body can repair these mistakes, but its ability to do so decreases with age. The linked molecules can stop vital biochemical cycles in cells, cause bottlenecks on critical molecular assembly lines (such as the ones on which amino acids are made into proteins), stiffen connective tissue, and create other forms of havoc. While this process doubtless contributes to aging, most gerontologists now believe that, like lipofuscin accumulation, cross-linkage is more a consequence than a cause.

■ *Free radicals.* In the course of normal oxidation—part of virtually every cellular process—small, highly charged pieces of the interacting molecules are often left over as by-products. They are called free radicals. Because each of these has an electron yearning to unite with the first molecule that comes along, free radicals can cause molecular collisions.

Such collisions are heavily involved in all sorts of injury to cells, including the damage to the heart muscle that continues after a heart attack and to nervous tissue after injury to the brain or spinal cord, and various forms of radiation damage. Free radicals also seem to be a cause of cross-linkage and lipofuscin accumulation. The free radical theory, put forward in 1954 by Denham Harman of the University of Nebraska Medical Center, has gained a great many adherents.

■ *Somatic mutation.* The genes in somatic cells—all those in the adult organism other than sperm and egg cells—were long known to be vulnerable to "point" mutations caused by the impact of, say, a cosmic ray or a potent chemical. The late Leo Szilard, as he turned his attention from physics to biology during the 1950s, hypothesized that such "hits" accumulate over the years to impair the genes, causing the decline of cells and, ultimately, of the whole organism.

This theory has had its strong advocates. Nevertheless, the genes, once we began to understand them at all, turned out to be much more complex than originally thought. They coil and "supercoil" into complex configurations; the irreversible unraveling of these structures is suspected to be instrumental to the aging process. Also, DNA is now known to have a self-repair capacity. The decline of this capacity, as observed in living cells, constitutes another theory of aging.

■ *Error catastrophes.* Once any organism, including a human, is full-grown, one of the main functions of the DNA in its

cells appears to be directing the manufacture of new proteins to be used for renewing the cell's own substance or for export as, say, hormones. In carrying out this "protein synthesis," the DNA's instructions are copied by another nucleic acid that carries the message into the main body of the cell; there, with the help of enzymes, the appropriate amino acids are strung together to form the desired protein.

The fact that cells that keep dividing must continue to copy and recopy their genetic instructions suggested a Xerox model of aging to Alex Comfort, who is a respected gerontologist as well as the author of the popular *Joy of Sex* books: After many copies, a genetic message gradually fades. Even in cells that no longer divide, errors occur as the cell renews itself, and mistakes are made in the activities that depend on it. The cell can usually rectify such mistakes. But if it does not, and a crucial molecule is impaired, some important job does not get done. That, as Leslie Orgel of the Salk Institute for Biological Studies has noted, can lead to further errors—and ultimately to what he calls an "error catastrophe" that can result in the cell's death. The error catastrophe theory does not now have many adherents, but it has spurred much fruitful thinking.

■ *The decline of immunity.* Roy Walford of the University of California, Los Angeles (UCLA), argues that an important cause of aging is a breakdown of the immune system. For instance, in early adulthood the thymus (the gland in the upper chest whose hormones stimulate the white blood cells needed to fight infection and cancer) has already begun to shrink. As life goes on, the immune system loses some of its ability to recognize and attack bacteria and other invaders, as well as incipient cancer cells. The immune cells may also begin to attack the body's own healthy cells, leading to autoimmune diseases such as rheumatoid arthritis and certain kidney ailments.

As for the "clock" theories, they assume that aging is genetically programmed, that a built-in "timer" exists. But where?

■ *The cellular clock.* One school of scientists holds that there is a timer in each cell. During the 1960s, Leonard Hayflick, now at the University of Florida, demonstrated convincingly that cells have a finite life span. In laboratory experiments, for instance, he was able to prove that one type of cell will divide into two only about 50 times before quitting. Even if these cells are frozen after 30 divisions, stored away, and then thawed, they will still divide only about another 20 times. Hayflick has also shown that the controls for this "aging under glass," as he calls it, are in the gene-bearing nucleus of each cell.

■ *The brain clock.* Other researchers hold that the aging

### THE ETERNAL DREAM

*Homo sapiens* has never been content with just being one of the longest lived mammalian species. Recorded history abounds in charlatans and dreamers who sought or claimed to have found the secret of extreme longevity. Before he died in 1971, for example, Swiss physician Paul Niehans made a career of administering "cellular therapy," i.e., injecting humans' ailing organs with cells taken from young lambs. The technique was effective only in drawing big fees from a hopeful clientele that included Bernard Baruch, Gloria Swanson, and even Pope Pius XII.

The chief cause of this century's great gains in average life expectancy (besides the development of vaccines and "wonder drugs") has been mundane improvements in public health—better sanitation and cleaner water supplies, for instance. Now gone from the list of the leading causes of death are those communicable diseases that can strike at any age, such as influenza, pneumonia and tuberculosis. Typhoid and polio have been virtually eliminated in America; smallpox and bubonic plague now occur chiefly in poor nations. Today's top killers are those ailments that typically appear later in life: heart disease, stroke, cancer, and Alzheimer's disease, the "mystery" brain disorder afflicting nearly 20 percent of the three to four million Americans over 65 who are mentally impaired.

timer is in the area at the base of the primitive brain housing the hypothalamus as well as the pituitary, the gland that controls the release of hormones. W. Donner Denckla, formerly of the Roche Institute and Harvard, believes that the pituitary begins at puberty to release a hormone, or a family of them, that causes the body to decline at a programmed rate. This "aging hormone"—which has not yet been isolated and thus proven conclusively to exist—hinders the cell's ability to take in thyroxine, the hormone produced by the thyroid gland. Thyroxine controls the metabolic rate in the body's key cardiovascular and immune systems, whose failure is involved in the diseases that kill most older persons.

Denckla's theory is backed by experiments with thousands of rats. He has found, for instance, that injections of the extract of ground-up pituitaries cause young rats to age prematurely. Older rats that have had their pituitaries removed and been given thy-

Who lives the longest? In general, primitive people age fast and die young. Still, the societies with the most notable longevity are those of poor mountain dwellers—the Vilcabambans of Ecuador, the Hunzas on the Chinese-Pakistani border, and the Abkhasians of the Soviet Union. While their claims of ages of 120 years or more are unverified, these tribes do include many centenarians. Researchers have not been able to determine whether something in these peoples' gene pools promotes longevity. What they are known to have in common is a slow-paced rural life, regular exercise from negotiating steep paths, and a lean and virtually meat-free diet. The Hunzas consume fewer than 2,000 calories a day, versus about 3,300 for the average American.

The longevity record among Americans is generally awarded to Charlie Smith, a black who arrived from Africa as a slave in 1854 and died in Florida in 1979, allegedly at the age of 137. Where firm documentation exists, however, the longest American lifespans appear to have been 111 or 112 years. The 1980 census found more than 30,000 American centenarians, two-thirds of them women.

What do those who reach advanced years have in common? Studies show that, to a degree, longevity is inherited; or, rather, that long-livers come from families with no history of early heart attacks or other life-shortening ailments. Beyond that, generalizations are hard to make. Insurance company records show that people who are rich and successful tend to live longer than others, though prosperity itself may or may not lead to longevity. Research on centenarians shows that they are people who are relaxed, able to shrug off life's vicissitudes, and notably cheerful in disposition. The apparent moral: He who *worries* too much about his health probably will not live to an extreme old age.

roxine injections (along with critical steroid hormones) have shown "young" characteristics in several areas, including fur growth, and in their cardiovascular and immune systems.

Could Hayflick and Denckla both be right? They could be, and I think they may well be.

Doubters may ask: If a brain clock controls aging, how could it affect cells like those that Hayflick has experimented with in the laboratory? A likely answer is that there is a cellular clock as well, though perhaps mainly intended as a fail-safe back-up mechanism.

Another question: Since almost any one of the wear-and-tear theories can account for nearly everything that happens in aging, why postulate a genetic clock?

There are a number of answers. All creatures seem to have evolved a "species-specific" life span. A shrew will live, say, a year and a half, while a Galápagos tortoise will go on for a cen-

tury and a half or more. If aging was just a matter of random wear and tear, would we not expect to see, now and then, a shrew that is 150 years old? Or a Galápagos tortoise that dies of old age at one and a half? But we never do.

Or take cells in tissue culture. Normal cells have a finite life span; they age and die at roughly the "Hayflick limit." But cancer cells are immortal; they do not age. If cancer cells are exposed to the same conditions as normal cells, how is it that the normal ones age and the abnormal ones do not? The apparent answer is deeply ironic: Cells do have an aging clock, but cancer somehow stops it.

Gerontologists do not want to stretch out, Tithonus-like, the years of senility. They want to increase the *vigor* of the later years. Some would also like to retard, stop, or even, in some respects, reverse the aging process (as Denckla seems to have done in his rats).

### Trying to Beat the Clock

It is now common in the laboratory to retard aging and to extend both the life expectancy and the life span of animals, as McCay did in the 1930s. The lives of fruit flies, fish, and other cold-blooded creatures have been extended by keeping them in a cooler-than-usual environment. At the National Institute of Aging's Gerontological Research Center in Baltimore, Charles Barrows has combined this technique and the McCay low-calorie stratagem. Working with rotifers—tiny pond-dwellers with a normal life of 18 days—he added another 18 days up front by restricting their calories. Then he added 18 more days to their mature period by cooling the water they lived in. Result: a tripled life span.

Seeking other life-extending techniques, investigators have used various antioxidants (to fight the damage caused mainly by free radicals), immune-system boosters and suppressors, and temperature-lowering drugs. Old and young rats have been joined surgically tail-to-shoulder so that they share a common circulatory system; the older rats age more slowly. Skin cells of old mice that have been transplanted to young ones easily outlive their original host. Evidently, the cells acquire *something* that keeps them vigorous.

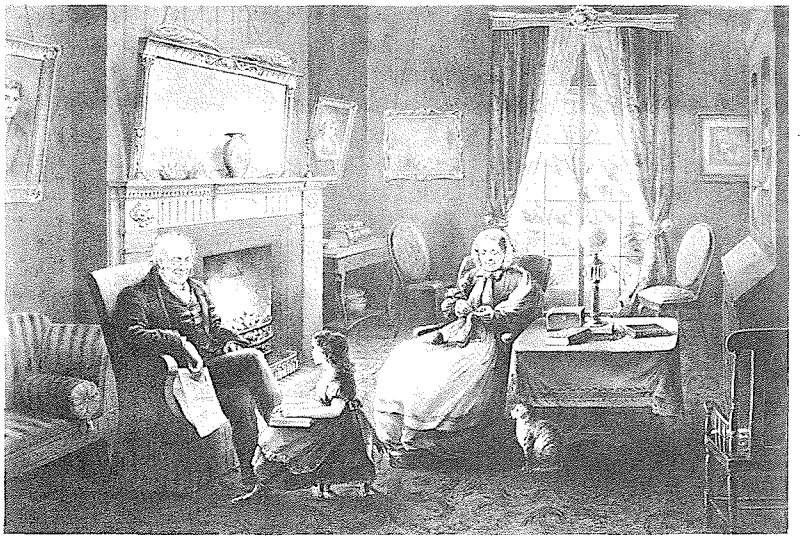
Can aging in *humans* be slowed, if not stopped entirely?

The hormonal brain clock, if it exists, could be counteracted by inhibiting the hormone—not an easy task, but by no means impossible. And if the cellular clock is found, it could be adjusted through genetic-engineering techniques.

A number of possible antidotes to wear and tear in humans are being studied. Vitamins C and E, glutathione, beta-carotene, and selenium are among the antioxidants that may curb free-radical damage—and offer protection against cancer as well. The thymic hormones offer promise as immune-system boosters. A steroid called DHEA (dehydroepiandrosterone), abundant in the bloodstream, may have potential as an aging-inhibitor and as an antiobesity, antidiabetes, and anticancer drug.

Meanwhile, researchers have hopes of producing lipofuscin scavengers, drugs that delay mental decline, and enzymes to replace those that diminish with age. More can be done with diet: UCLA's Walford is working on adapting McCay's low-calorie technique for use with adult animals, including humans.

At present, most medical research is aimed at specific diseases. The juvenile ailments, such as childhood cancer and juvenile arthritis, are believed to be largely genetic in origin, but most adult disorders simply come with age. As it happens, many of the techniques that retard aging in laboratory tests, such as calorie restriction, also seem to retard cancer and other degenerative diseases of adulthood. Surely the simplest way to deal with those diseases would be to deal with aging itself. After a heart by-pass, for instance, the hardening-of-the-arteries process that prompted the surgery goes right on as be-



*This 19th-century Currier and Ives ideal of a sedentary* Old Age *is rejected by gerontologists. They seek to increase the vigor of late life.*

fore. To go on pouring money into by-pass operations, or kidney dialysis, or nursing homes instead of trying to alter the aging process makes as much sense as it would have made, say, for the March of Dimes to have thrown all its resources into buying iron lungs instead of helping Jonas Salk and Albert Sabin develop their polio vaccines.

Many argue that even if we can extend vigorous life, we should not. Consider the personal, social, ethical, and political problems that would arise. If people were to live longer and in ever greater numbers, continuing to consume and perhaps reproduce, what—they ask—would happen to Social Security and insurance premiums? Would the global shortage of resources and our problems with pollution worsen? Would we have to put age limits on parenthood or consider the Huxleyan notion of requiring a license for parenthood? What would happen to creativity and progress without the continued influx of new ideas—and opportunities for young people to put them into action? Could *longer* lives lead to gerontocracy? To conflict between younger and older generations?

We must indeed give our most serious consideration to such possibilities. But we should also consider the consequences of *not* doing anything about aging. Though some scientists speak of actually extending the life span, that prospect is not yet with us. What society does face is the probability that the pattern of the final stage of life can be changed from decrepitude and dependency to something much healthier. Is such an outcome really to be deplored? In obeisance to what ethical doctrines should we condemn the elderly, now and forever, to continue to suffer the ravages of senescence? And condemn our societies to continue to bear the resulting burdens? Will we really choose to supplement the genes' tyranny with our own?