

The Aging of the Human Species

Our species has modified the evolutionary forces that have always limited life expectancy. Policymakers must consequently prepare to meet the needs of a population that will soon be much older

by S. Jay Olshansky, Bruce A. Carnes and Christine K. Cassel

For the first time, humanity as a whole is growing older. The demographic aging of the population began early in this century with improvements in the survival of infants, children and women of childbearing age. It will end near the middle of the next century when the age composition of the population stabilizes and the practical limits to human longevity are approached. No other species has ever exerted such control over the evolutionary selection pressures acting on it—or has had to face the resulting consequences.

Already the impact of the demographic transformation is making itself felt. In 1900 there were 10 million to 17

million people aged 65 or older, constituting less than 1 percent of the total population. By 1992 there were 342 million people in that age group, making up 6.2 percent of the population. By 2050 the number of people 65 years or older will expand to at least 2.5 billion people—about one fifth of the world's projected population. Barring catastrophes that raise death rates or huge inflations in birth rates, the human population will achieve a unique age composition in less than 100 years.

Demographers, medical scientists and other workers have anticipated the general aging of the human species for several decades, yet their attention has been focused almost exclusively on the concurrent problem of explosive population growth. We believe, however, that population aging will soon replace growth as the most important phenomenon from a policy standpoint. In a more aged population, the patterns of disease and disability are radically different. Many economic and social institutions that were conceived to meet the needs of a young population will not survive without major rethinking. Attitudes toward aging and the aged will have to be modified to address the demands of a much larger and more diverse older population.

Age structure is a characteristic of populations that reflects the historical trends in birth and death rates. Until recently, the shape of the human age structure was fairly constant.

Before the mid-19th century the an-

nual death rates for humans fluctuated but remained high, between 30 and more than 50 deaths per 1,000 individuals. Those elevated, unstable rates were primarily caused by infectious and parasitic diseases. The toll from disease among the young was especially high. Often almost one third of the children born in any year died before their first birthday; in some subgroups, half died. Because childbirth was very hazardous, mortality among pregnant women was also high. Only a small segment of the population ever lived long enough to face the physiological decrements and diseases that accompany old age.

The only reason *Homo sapiens* survived such terrible early attrition was that the number of births more than compensated for the deaths. It was common for women to give birth to seven or more children in a lifetime. The higher birth rates were part of a successful survival pattern that reflected an array of favorable evolutionary adaptations made by humans.

Together the evolutionary constraints and adaptations produced a long-term average growth rate for the human species that, at least before the mid-19th century, hovered just above zero. The age structure of the population had the shape of a pyramid in which a large number of young children made up the broad base. At the apex were the few people who lived past their reproductive adulthood. The mean age of the population was low.

Clearly, much has changed since then.

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ELDERLY PEOPLE OF TOMORROW are only children today. For the first time, much of the population is living into advanced old ages. That demographic change carries potential risks. Re-

forms in social policy and further biological research may determine whether the additional years of life available to the population will be healthy and prosperous ones.

During the 20th century, the disparity between high birth rates and low death rates led to population growth rates that approached 2 to 3 percent and a population doubling time of only about 25 years. In the U.S. today, people aged 65 and older make up 12.5 percent of the population; by 2050 they will constitute 20 to 25 percent. This change is the result of declining mortality during the early and middle years. It was initially brought forth by improvements in sanitation and was later assisted by other public health measures and medical interventions. Collectively, they asserted control over the death rates from infectious and parasitic diseases and from maternal mortality.

The series of steps by which a population ages has been the subject of considerable research. Indeed, the patterns of this demographic transformation and the speed with which they occur

are central to understanding the social problems now on the horizon.

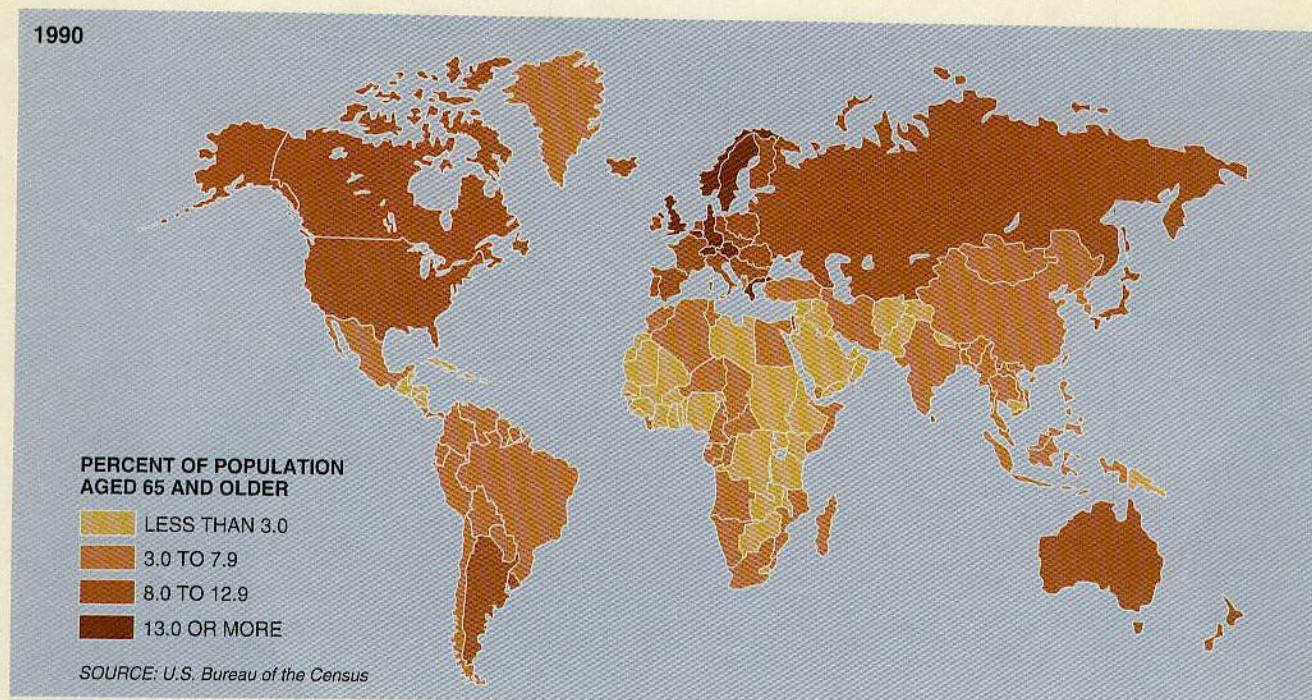
Initially, declines in infant, child and maternal death rates make the population younger by expanding the base of the age pyramid. Yet that improvement in survival, along with social and economic development, leads to a drop in birth rates and the beginning of population aging. Fewer births produce a narrowing of the pyramid's base and a relative increase in the number of people who are older.

As risk of death from infectious and parasitic diseases diminishes, the degenerative diseases associated with aging, such as heart disease, stroke and cancer, become much more important. Whereas infectious and parasitic diseases usually occur in cyclic epidemics, the age-related diseases are stable and chronic throughout an ex-

tended life. Consequently, the annual death rates fall from high, unstable levels to low, steady ones of eight to 10 persons per 1,000. Abdel R. Omran, when at the University of North Carolina at Chapel Hill, was the first to describe this change as an "epidemiologic transition." The rate of change and underlying causes of the transition differ among subgroups of the population.

In the final stage of the epidemiologic transition, mortality at advanced ages decreases as medical and public health measures postpone the age at which degenerative diseases tend to kill. For example, heart disease, stroke and cancer remain the primary causes of death, but healthier ways of life and therapeutic interventions permit people with those diseases to live longer. Disease onset and progression can also be delayed.

Once the birth and death rates in a population have been in equilibrium at



AGING OF THE WORLD POPULATION will become much more apparent during the 21st century. The trend is already pronounced in the industrialized countries. Within just a few decades, much of the population in the developing world will

low levels for one average life span—approximately 85 to 100 years—the age structure becomes almost permanently rectilinear: differences in the number of persons at various ages almost disappear. Thereafter, more than 90 percent of the people born in any year will live past the age of 65. About two thirds of the population could survive past 85, after which death rates would remain high and the surviving population will die rapidly. Such age structures have been observed in laboratory mice and other animals raised in controlled environments.

A crucial feature of the rectilinear age structure is its stability. If birth rates increase and temporarily widen its base, its rectilinear shape will gradually reassert itself because nearly all the members of the large birth generation will survive to older ages. Conversely, if the birth rate falls, the aging of the population will temporarily accelerate because the young become proportionally less numerous. The rectilinear age structure persists as long as early and middle-age mortality remain low.

The trend toward stable, low death rates has already been observed for a substantial segment of the world's population. Nevertheless, no nation has yet achieved a truly rectilinear age structure. Countries such as Sweden and Switzerland are much further along in the demographic transformation to population equilibrium than are other developed nations.

In the developed nations, two major phenomena have had a particularly noteworthy influence on the transformation of the age structure. The first is the post-World War II baby boom, the rise in birth rates that occurred during the middle of the century. Although 100 years is usually enough time for an age structure to become stable, the high birth rates of the baby boom postponed the aging of the population by widening the base of the age structure again. As the baby boomers grow older, however, the average age of the population will increase much faster. The stabilization process will probably take about 150 years for the developed nations, in which rectilinear age structures should become common by 2050.

The second factor that influenced population aging in developed nations was the unexpected decline in old-age mortality that began in the late 1960s. Few scientists had anticipated that death rates from vascular disease could substantially be reduced at older ages. A fall in old-age mortality accelerates population aging by raising the age at which death becomes more frequent and the age structure begins to narrow. Death has become an event that occurs almost exclusively at older ages for some populations.

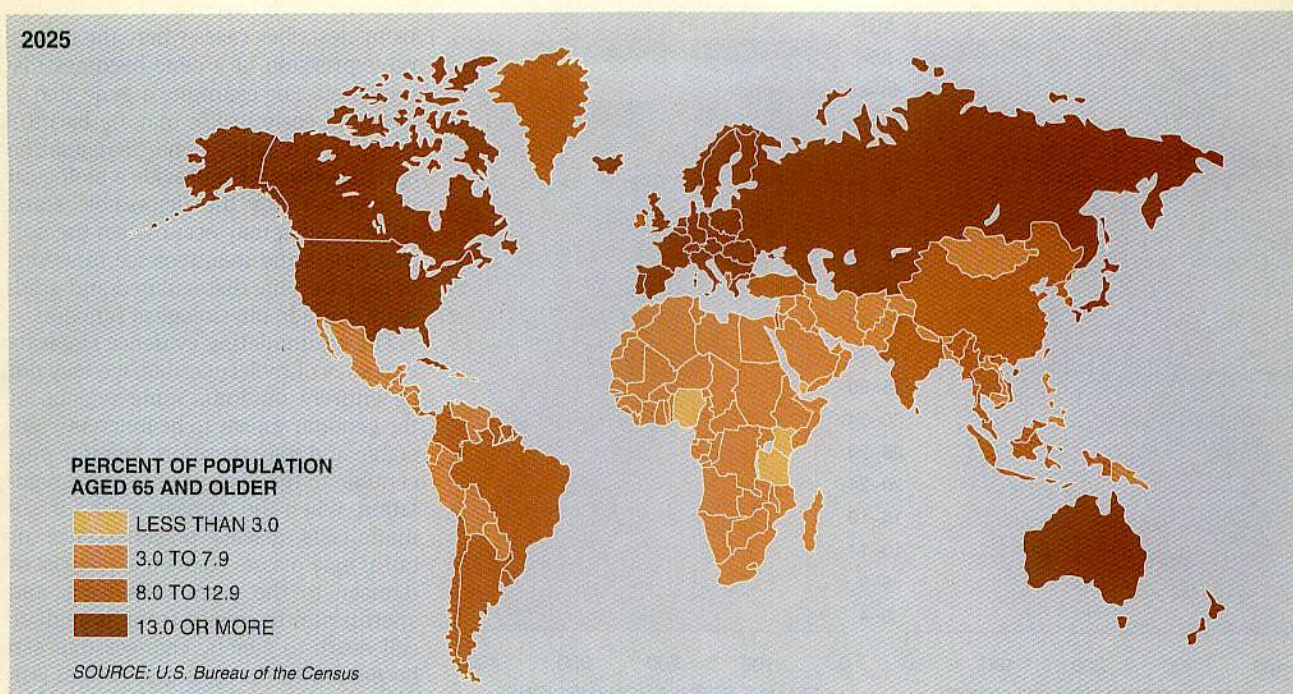
In many developing countries and in some groups within developed nations, human populations still face intense selection pressures. Consequently, some developing nations are not likely to reach equilibrium even by the middle

of the 21st century. Nevertheless, the pace at which the population ages will accelerate throughout the developing world for the next 60 years.

For example, in China, which has both the largest population and the largest number of elderly people, the population aged 65 and older will increase from 6.4 percent (71 million people) to about 20 percent (270 million people) by 2050. China will then contain more people over 65 than the U.S. now has at all ages. India, which has the second largest elderly population, should experience even greater proportional increases.

We must emphasize that the demographic momentum for both population growth and population aging is already built into the age structures of all nations: the people who will become old in the next half century have, of course, already been born. These demographic forces will present a formidable set of social, economic and health problems in the coming decades—many of which are as yet unforeseen by policymakers and are beyond the capacity of developing countries to handle.

By the middle of the 21st century the transformation to an aged population should be complete for much of humanity. No one yet knows whether medical science will thereafter succeed in postponing the age at which rapid increases in the death rate begin. Will the apex of the age distribution retain its shape but shift to older ages, or will mortality be compressed into a shorter time span?



also be dramatically older. This demographic transformation is occurring because mortality at young ages has diminished.

The social, medical and economic changes that accompany the aging of the population will pose significant problems.

The answer, which could profoundly affect economic and health issues, depends on whether there is an upper limit to longevity and a lower limit to the death rate.

For decades, the question of how low death rates can go has puzzled researchers. In 1978 demographer Jean Bourgeois-Pichat of Paris calculated that the average human life expectancy would not exceed 77 years. He arrived at that figure by theoretically eliminating all deaths from accidents, homicides, suicides and other causes unrelated to senescence. He then estimated the lowest death rates possible for cardiovascular disease, cancer and other diseases associated with aging. In effect, he eliminated all causes of death except those that seemed intrinsic to human biology. Yet shortly after its publication, Bourgeois-Pichat's life expectancy limit had already been exceeded in several nations. Other demographers have speculated that life expectancy will soon approach 100 years, but their theoretical estimates require unrealistic changes in human behavior and mortality.

In 1990 we took a more practical approach to the question of longevity. Rather than predicting the lower limits to mortality, we asked what mortality schedules, or age-specific death rates, would be required to raise life expectancy from its current levels to various target ages between 80 and 120 years. To determine the plausibility of reach-

ing the targets, we compared those mortality schedules with hypothetical ones reflecting the elimination of cancer, vascular problems and other major fatal diseases. We demonstrated that as the actuarial estimate of life expectancy approaches 80 years, ever greater reductions in death rates are needed to produce even marginal increases in life expectancy.

Our conclusion was that life expectancy at birth is no longer a useful demographic tool for detecting declines in death rates in countries where mortality rates are already low. Furthermore, we suggested that the average life expectancy is unlikely to exceed 85 years in the absence of scientific breakthroughs that modify the basic rate of aging. Like others before us, we demonstrated that even if declines in death rates at older ages accelerate, the gains in life expectancy will be small.

Why is the metric of life expectancy so insensitive to declining old-age mortality in low-mortality countries? First, for as long as reliable mortality statistics have been collected, the risk of death has always doubled about every eight years past the age of 30. That characteristic of human mortality has not changed despite the rapid declines in death rates at all ages during this century. A 38-year-old man today has a longer life expectancy than one from a century ago, but he is still twice as likely to die as a 30-year-old man.

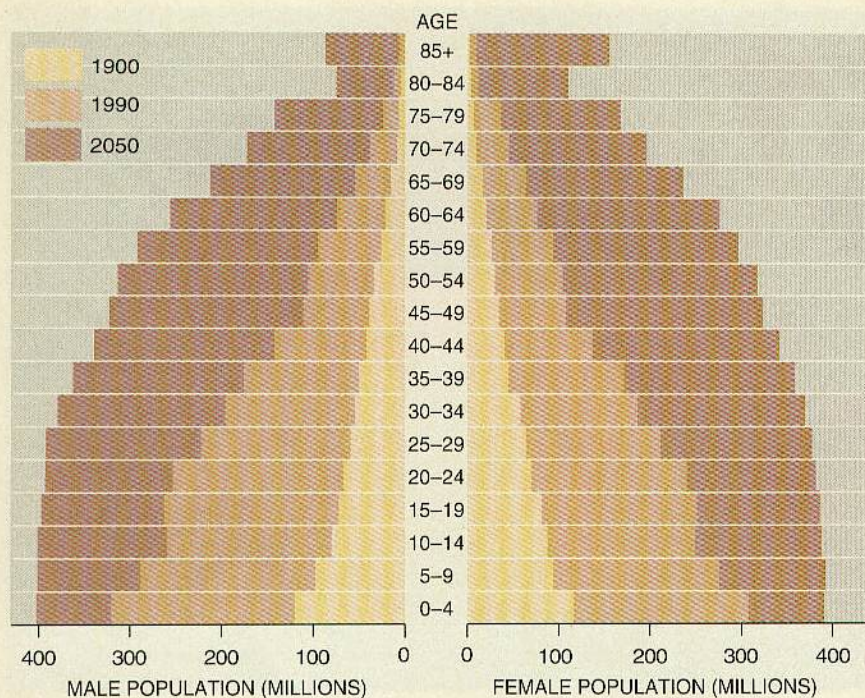
Moreover, there is no indication that humans are capable of living much past

the age of 110 regardless of declines in death rates from major fatal diseases. Thus, as death becomes ever more confined to older ages, the decline in death rates will inevitably stop. The point of deceleration occurs as life expectancy approaches 80 years.

Finally, in low-mortality countries, cardiovascular disease and cancer account for three of every four deaths after age 65. Those diseases are, in effect, competing for the lives of individuals, particularly at advanced ages. If the risk of dying from any single disease were reduced to zero, the saved population would simply be subject to high mortality risks from other causes—yielding a surprisingly small net gain in life expectancy. As deaths become concentrated into older ages, the competition among causes of mortality grows more pronounced.

Conceivably, however, medical researchers may learn how to slow the rate of senescence itself, thereby postponing the onset of degenerative diseases and the causes of old-age mortality. Toward that goal, many scientists working in the fields of evolutionary and molecular biology are now trying to learn why organisms become senescent.

In an influential paper written in 1957, evolutionary biologist George C. Williams, who was then at Michigan State University, proposed a mechanism for the evolution of senescence. His theory and subsequent predictions rested on two arguments. First, indi-



AGE STRUCTURE of the population is changing dramatically. For the past 100,000 years, the human age structure had the shape of a narrow pyramid. Since 1900, it has become wider and more rectilinear because relatively larger numbers of people in the growing population are surviving to older ages. By the middle of the 21st century it will be very nearly rectangular.

vidual genes are involved in multiple biological processes—a widely accepted concept known as pleiotropy. Second, he proposed that certain genes conferred survival advantages early in life but had deleterious physiological effects later. He then linked those assumptions to the prevailing concept that an individual's evolutionary fitness is measured by the genetic contribution that he or she makes to subsequent generations.

Williams then argued that an individual's odds of reproducing successfully would inevitably diminish over time because he or she would eventually die from an accident or some other uncontrollable cause. As individuals fulfill their reproductive potential, selection pressures should diminish, and any genes that had damaging effects later in life could not be eliminated by natural selection. Williams argued that this process, called antagonistic pleiotropy, provided a genetic basis for aging.

Another theory, proposed in 1977 by biologist T.B.L. Kirkwood of the National Institute for Medical Research in London, is a special case of antagonistic pleiotropy. He assumed that organisms must always divide their physiological energy between sexual reproduction and maintenance of the soma, or body. The optimum fitness strategy for a species, he argued, involves an allocation of energy for somatic maintenance that is

less than that required for perfect repair and immortality. Senescence is therefore the inevitable consequence of the accumulation of unrepaired defects in the cells and tissues. Under Kirkwood's disposable soma theory, senescence is the price paid for sexual reproduction.

The dysregulation of genes may provide a mechanism that links the antagonistic pleiotropy and disposable soma theories into a unified concept of disease and senescence. Two concepts central to the modern paradigm of molecular biology are required: gene regulation and pleiotropy. It is assumed in molecular biology that genes are carefully regulated and that the proteins produced by gene activity are typically involved in multiple, often interacting processes. Over time, a gradual accumulation of random molecular damage could disrupt the normal regulation of gene activity, potentially triggering a cascade of injurious consequences. Richard G. Cutler, a gerontologist at the National Institute on Aging, has referred to this process as the dysdifferentiative hypothesis of aging.

The severity of the consequences will depend on how critical the affected processes are at the time of their dysregulation and the ability of the organism either to compensate for or to repair the damage. If the damage disrupts the regulation of cell growth or differentiation, cancer could result. Antagonistic pleio-

tropy describes cases where the temporal expression of a gene becomes dysregulated. For example, a gene that is essential early in life may be harmful if expressed later. Gene dysregulation and pleiotropy also provide a biological mechanism for the disposable soma theory. Aging may occur when the normal repair and maintenance functions of cells become dysregulated and gradually degrade physiological function.

The accumulating evidence suggests that sites of molecular damage may not be entirely random. Some regions of the genome appear to be inherently unstable and may therefore be more susceptible to the disruption of gene regulation. When the damage occurs in somatic cells, disease or senescence, or both, may occur. The consequences of damage to the germ cells (eggs and sperm) run the gamut from immediate cell death to genetic changes that can be passed to the next generation. Propensities for disease and competency of somatic maintenance and repair are probably inheritable traits.

If there is a biological clock that begins ticking when a sperm fertilizes an egg, it probably does not go off at some predetermined date of death encoded in the genes. Rather the breakdown in gene regulation is a product of purely random events acting over a lifetime on a genome that contains inherited instabilities. As our understanding of biomolecular mechanisms grows, it may eventually become possible to manipulate disease processes and to slow the rate of senescence, thereby extending the average life span.

Although its link to molecular mechanisms is uncertain, one method of lengthening life span is known: dietary restriction. Early in the 20th century, researchers found that laboratory rats fed a low-calorie diet lived longer than those allowed to consume food at will. Those findings have been repeated for several species, including mice, flies and fish. Work by Richard Weindruch and his colleagues at the National Institute on Aging and by Roy L. Walford and his colleagues at the University of California at Los Angeles has suggested that dietary restriction may slow some parameters of aging in nonhuman primates.

These studies suggest life span can be extended by postponing—without eliminating—the onset of fatal diseases. Caloric restriction does not alter the rate of physiological decline in the experimental animals, nor does it change the doubling time for their death rate. Instead the animals appear to live longer because the age at which their death rates begin to increase exponentially is delayed. Dietary restriction seems to

help preserve somatic maintenance for a longer time. Although it is not practical to expect enough people to adopt a calorically restricted diet to increase the average human life span, research may be able to identify the mechanisms at work and thereby extend longevity by other means.

Few observers had imagined that the demographic evolution of the human age structure would reveal a new set of diseases and causes of death. Will future reductions in old-age mortality reveal even more, new senescent diseases? Or will the prevalence of existing senescent diseases simply increase? Given the health care industry's focus on further reducing the impact of fatal diseases and postponing death, these issues will become critical to policymakers attempting to evaluate the consequences—both medical and economic—of an aging population.

One of the most important issues is whether the trend toward declining old-age mortality will generally benefit or harm the health of the overall population. In a controversial paper published 12 years ago, physician James F. Fries of Stanford University hypothesized that the biological limit to human life is fixed at about 85 years. Better life-styles and advances in medical technology, he said, will merely compress mortality, morbidity and disability into a shorter period near that limit. His underlying premise was that changes in diet, exercise and daily routines will postpone the onset age both of the major fatal diseases (heart disease, cancer and stroke) and of the debilitating diseases of old age (including Alzheimer's disease, osteoporosis and sensory impairments).

Fries's compression-of-morbidity hypothesis has since been challenged by many scientists who posit an expansion of morbidity. They argue that the behavioral factors known to reduce the risks from fatal diseases do not change the onset or progression of most debilitating diseases associated with aging. Further reductions in old-age mortality could therefore extend the time during which the debilitating diseases of aging can be expressed. In effect, an inadvertent consequence of the decline in old-age mortality may be a proportional rise in the untreatable disabilities now common among the very old. This view has been referred to as trading off longer life for worsening health.

The expansion-of-morbidity hypothesis serves as a consequence and a corollary to the evolutionary theories of aging. As a larger and more heterogeneous population survives into more advanced

ages, the opportunities increase for the known senescent diseases to become more prevalent. New diseases associated with age (possibly resulting from the pleiotropic effects of gene dysregulation) may also have a greater opportunity to manifest themselves.

The ramifications of the expansion-of-morbidity hypothesis are so alarming that an international organization of scientists has been formed under the direction of demographer Jean-Marie Robine of INSERM in France to test its validity. The group's focus is the complex relation between declining old-age mortality and the relative duration of life spent healthy or disabled. Robine and his colleagues have demonstrated that women in Western societies can expect to spend up to one quarter of their lives disabled and men up to one fifth. Wealthier people are more likely to live longer and be healthier than those who are less well-off.

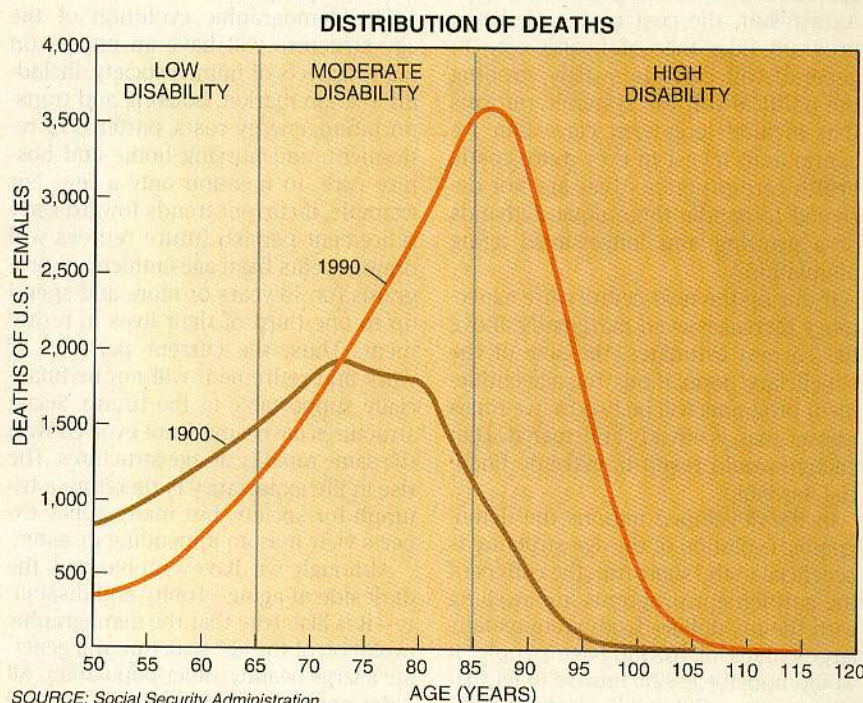
The data also suggested that recently the average number of years that people spend disabled has grown faster than those that they spend healthy. In other words, although people are enjoying more healthy years while they are young and middle-aged, they may be paying the price for those improvements by spending more time disabled when they are older. Because of the known problems of data reliability and comparability and of the short periods

observed, current trends in morbidity and disability must be interpreted with caution.

The dilemma we face as a society is that medical ethics oblige physicians and researchers to pursue new technologies and therapeutic interventions in efforts to postpone death. Yet that campaign will inadvertently accelerate the aging of the population. Without a parallel effort to improve the quality of life, it may also extend the frequency and duration of frailty and disability at older ages. Society will soon be forced to realize that death is no longer its major adversary. The rising threat from the disabling diseases that accompany most people into advanced old age is already evident.

There is every reason for optimism that breakthroughs in molecular biology will permit the average life span to be modified. Just how far life span could be extended by slowing the rate of senescence is the subject of much speculation and debate. No one has yet demonstrated that human senescence can be modified by any means.

It is also unclear how those breakthroughs might influence the quality of life. If slowing the rate of senescence postpones all the physiological parameters of aging, then youth could be prolonged and disability compressed into a short time before death. If only some



PATTERNS OF DEATH AND DISABILITY are shifting as an epidemiologic transition occurs in the aging population. Because of healthier ways of life and medical interventions, people are surviving longer with heart disease, stroke and cancer. Yet because of their extended survival, they may suffer longer from the nonfatal but highly disabling illnesses associated with old age.

parameters of aging are amenable to modification, however, then the added years may become an extension of disabled life in old age.

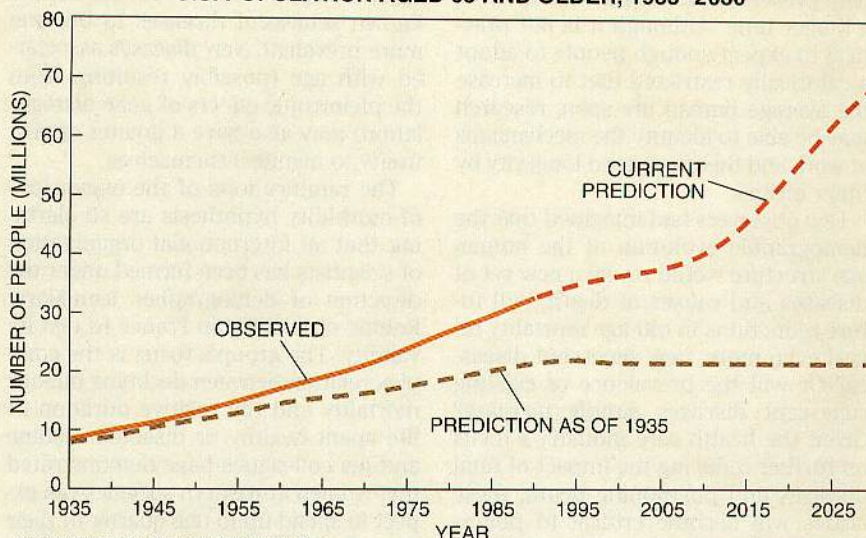
We can identify with certainty some of the social problems that an aging population will face. Two of the most difficult will be the financial integrity of age-based entitlement programs, such as Social Security and Medicare, and the funding of health care. Social security programs in the U.S. and other countries were created when the age structures were still pyramidal and life expectancies were less than 60 years. The populations receiving benefits from those programs are much larger—and living considerably longer—than was anticipated at their inception. Given that the demographic momentum for larger and longer-lived older populations already exists, it is inescapable that such programs cannot survive in their present form much beyond the second decade of the next century.

Because declining mortality allows most people to survive past the age of 65, Medicare will need to cover tens of millions of people in the U.S. Many of them will need coverage for several decades. Medicare has few effective restraints on the use of expensive acute care, which is critical for treating many fatal illnesses. Yet it covers almost none of the expense of chronic long-term care—the need for which will grow as rapidly as the population ages. As a result, the cost of the Medicare program (like that of health care in general) will escalate swiftly, eroding the political will for systemic reforms that include long-term care. Can we continue to invest in ever more costly health care programs that are not designed to handle the unique demands of a growing and longer-lived aging population?

If during the next century life expectancy increases even marginally above the current estimates, the size of the beneficiary populations for age-entitlement programs will be two to five times greater than is already anticipated. That change would result in extreme financial hardship.

In the developed nations the demographic evolution of the age structure is beneficial in the short run: the coffers of the entitlement programs are swelling with the tax dollars from an unusually large cohort of working-age people. It would nonetheless be unwise to let that temporary condition lull us into complacency. When the age structure in those nations becomes rectilinear, the ratio of beneficiaries to taxpayers will mushroom, and surpluses in entitlement programs will vanish.

U.S. POPULATION AGED 65 AND OLDER, 1935–2030



SOURCE: Social Security Administration

STRAINS ON SOCIAL PROGRAMS, such as Social Security and Medicare, will continue to emerge as the population ages and life expectancy increases. The number of beneficiaries in the Social Security program, for example, is growing much faster than was anticipated when the program was first conceived decades ago.

The financial integrity of age-entitlement programs has already been jeopardized in some countries. The worst problems will arise globally just after the year 2010, when the generation of baby boomers reaches entitlement age. The certainty of the demographic evolution of population aging will soon force governments to restructure all their entitlement programs.

The demographic evolution of the age structure will have an impact on many aspects of human society, including the job market, housing and transportation, energy costs, patterns of retirement, and nursing home and hospice care, to mention only a few. For example, if current trends toward early retirement persist, future retirees will draw benefits from age-entitlement programs for 30 years or more and spend up to one third of their lives in retirement. Thus, the current patterns of work and retirement will not be financially supportable in the future. Social structures have simply not evolved with the same rapidity as age structures. The rise in life expectancy is therefore a triumph for society, but many policy experts view it as an impending disaster.

Although we have emphasized the dark side of aging—frailty and disability—it is also true that the demographic evolution of the age structure will generate a large healthy, older population. All older people, both the healthy and the sick, will need the chance to contribute meaningfully to society. Achieving that end will require an economy that provides ample, flexible opportunities for experienced and skilled older persons,

as well as modifications in the physical infrastructures of society. Changes in attitudes about aging will be essential.

The medical establishment is continuing to wage war against death. Researchers in the field of molecular biology are still searching for ways to slow the basic rate of aging. Those efforts lead us to believe that the aging of the population will also continue and perhaps even accelerate. Everybody wants to live longer, and medicine has helped that dream come true. Only now is society beginning to comprehend what it has set in motion by modifying the natural selection forces that have shaped the evolution of human aging.

FURTHER READING

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