Dædalus

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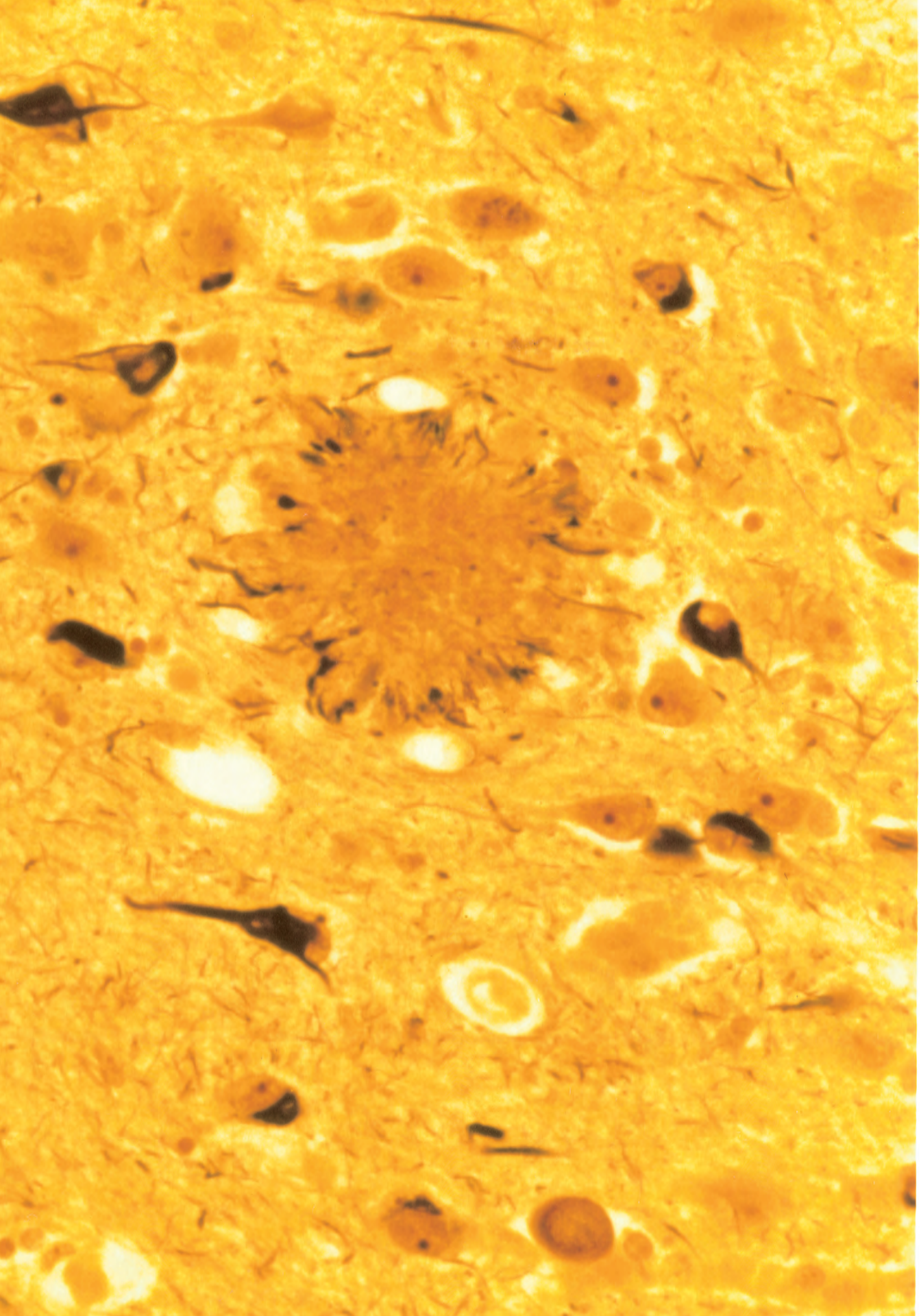

Inside front cover: A picture of progressive dementia, using a silver-stained section of the amygdala of the brain from a sixty-nine-year-old man with a nine-year history of the disease. In this image, darkly stained neurofibrillary tangles occupy much of the cytoplasm of selected pyramidal neurons, in contrast to the golden brown cytoplasm of numerous adjacent cytologically normal neurons. In the center, a senile plaque, consisting of a large compacted deposit of extracellular amyloid, is intimately surrounded by a halo of dilated, structurally abnormal (dystrophic) neurites – represented by the squiggly dark brown profiles. See Dennis J. Selkoe on *The aging mind: deciphering Alzheimer's disease & its antecedents,* pages 58 – 67: "The process of brain aging can contribute to the development of a clinically noticeable dementing illness, but aging by itself appears to be insufficient to cause the illness." Image courtesy of Dennis J. Selkoe.

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Announcement

Correction: In the Fall 2005 issue, "50 years," acknowledgment should have been made of Stephen R. Graubard, the editor who assigned and edited all but one of the essays that were reprinted in that issue.

Dædalus is designed by Alvin Eisenman

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The labyrinth designed by Daedalus for King Minos of Crete, on a silver tetradrachma from Cnossos, Crete, c. 350 – 300 B.C. (35 mm, Cabinet des Médailles, Bibliothèque National, Paris). "Such was the work, so intricate the place, / That scarce the workman all its turns cou'd trace; / And Daedalus was puzzled how to find / The secret ways of what himself design'd." – Ovid, *Metamorphoses*, Book 8

Dædalus was founded in 1955 and established as a quarterly in 1958. The journal's namesake was renowned in ancient Greece as an inventor, scientist, and unriddler of riddles. Its emblem, a maze seen from above, symbolizes the aspiration of its founders to "lift each of us above his cell in the labyrinth of learning in order that he may see the entire structure as if from above, where each separate part loses its comfortable separateness."

The American Academy of Arts & Sciences, like its journal, brings together distinguished individuals from every field of human endeavor. It was chartered in 1780 as a forum "to cultivate every art and science which may tend to advance the interest, honour, dignity, and happiness of a free, independent, and virtuous people." Now in its third century, the Academy, with its more than four thousand elected members, continues to provide intellectual leadership to meet the critical challenges facing our world.

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Chris Wilson

The century ahead

The twentieth century was, above all else, a century of population growth; the twenty-first century will be a century of aging. Between 1900 and 2000 the world's population quadrupled, from around 1.5 billion to over 6 billion. Most of this increase occurred after World War II. At present, it seems unlikely that the population will grow by more than about a further 50 percent. The most plausible forecasts see a population numbering between 9 and 10 billion by about 2050, with stability or decline in total population thereafter.

However, the population at older ages will increase far more quickly in the coming century than in the last. Indeed, the end of population growth and its replacement by aging are logically related. All rapidly growing populations are young. If each birth cohort is larger than the one before, there will always be plenty of young people.

Chris Wilson is a staff member of the World Population Program at the International Institute for Applied Systems Analysis in Laxenberg, Austria. One of Europe's most widely cited demographers, he is currently researching the causes and consequences of global demographic convergence.

Population growth was so characteristic of the recent past that we tend to regard it as the norm. However, for most of human history the long-run rate of population growth has been very close to zero. From the biblical Adam and Eve, it would have taken only thirty-two doublings of the population to reach over 8 billion. At the rate of population growth seen in the 1960s and early 1970s – over 2 percent a year, implying a doubling time of around thirty years – and given that the gap between generations is also usually about thirty years, such an increase could have taken place inside a millennium. Even James Ussher's 1650 estimate of October 23, 4004 B.C. as the date of creation implies we have been around much longer than that. And since *Homo sapiens* actually emerged one hundred and fifty thousand or so years ago, the rate of growth has obviously been close to zero.

Similarly, extrapolating the growth rates of the recent past into the future soon yields logically impossible figures. Ansley Coale once calculated that a growth rate of 2 percent a year sustained for five thousand years would lead to the sheer volume of human beings exceeding that of the solar system.

The absence of growth is a necessary but not sufficient condition for aging;

we also need long life expectancy. In populations before the modern medical era, relatively few people survived to reach three score years and ten. Thus, population aging is a novelty requiring both long lives and a low growth rate (i.e., low fertility). Though rare in the past, these conditions are now becoming the norm around the world.

When demographers try to understand the determinants of aging, they use one of social science's great generalizing models: the demographic transition. When a population modernizes, it undergoes, along with many other aspects of development, a set of interconnected changes called the demographic transition. According to this model, every population at some point has high fertility (mostly between four and six children per woman) and low life expectancy (mostly between twenty-five and forty years). With the spread of modern medicine and public health, mortality improves; as family planning and contraceptive use become the norm, fertility falls. Usually life expectancy rises first, with a delay before fertility declines. This difference in timing leads to substantial population growth before the two processes come back into balance.

This process of transition began in the late eighteenth and nineteenth centuries in Europe, the United States, and the other neo-Europes; it became a global phenomenon after World War II. Today, more than half of the world's people live in places where fertility is at or below the level needed for long-run intergenerational replacement (about 2.1 children per woman), and global life expectancy is approaching seventy years.

Trends in mortality can be followed in considerable detail for many European countries from the mid-nineteenth century, and for a few especially well-documented cases, as far back as the late 1700s. For Japan and the United States detailed information dates back to the early twentieth century. What these statistics reveal is both simple and striking. There has been an enormous reduction in mortality, with life expectancy for the two sexes combined now approaching, or even exceeding, eighty in most developed countries. Even more remarkably, this progress has been very regular for many decades. Jim Oeppen and James Vaupel have shown, for example, that the trend in "best-practice" life expectancy (i.e., the country with the longest life expectancy in each year) has been linear for more than 150 years.[1] In each decade the "state of the art" has increased about 2.5 years. Moreover, although there has been some variation at the national level, most developed countries have demonstrated strongly linear trends in life expectancy for the whole of the twentieth century.

Paradoxically, although this trend has been evident in mortality statistics for many decades, it is only in the last few years that it has been recognized. Demographers, actuaries, and others concerned with forecasting mortality had always hitherto assumed that life expectancy was approaching some asymptotic limit and would thus level off in the near future. But if there is some biological limit to extending longevity, there is no sign of it yet. As Oeppen and Vaupel point out, estimates of the maximum possible life expectancy made throughout the twentieth century were, on average, surpassed within five years of being made. This consistent error is of more than purely academic interest – pension-

1 Jim Oeppen and James W. Vaupel, "Broken Limits to Life Expectancy," *Science* 296 (2002): 1030 – 1031.

and health-care systems have been funded on the basis of large underestimates of the number of elderly people in the future.

The linearity of the upward climb in life expectancy has occurred in spite of the fact that very different age groups and causes of death have been involved in different eras. Before World War II, almost all progress took place in reducing infectious diseases, with the biggest impact for infants and children. In contrast, today much of the improvement is concentrated at old ages. Perhaps the best analogy for these remarkable changes is to be found in models of economic growth. Just as modern theory hypothesizes the existence of an endogenous rate of growth that is in some sense built into our economic system, so too there may be an endogenous rate of improvement in health, as measured by life expectancy. In any event, we have every reason to expect that continued increases in the average length of life will augment population aging.

There are, of course, exceptions to this optimistic picture. In the Soviet Union and its client states in Eastern and Central Europe, life expectancy stagnated from the 1960s until the end of Communism. It then worsened still further in many cases, in the immediate aftermath of revolution. In Russia and many of the post-Soviet states it remains low, especially for men. Male life expectancy in Russia today is roughly the same as it was in 1950: about sixty years. To put this stagnation into perspective, the equivalent figure for the United States has increased since 1950 by almost ten years from sixty-six to seventy-six.

In the post-Communist countries further west, however, the last decade has seen rapid improvements; life expectancy there will likely converge to levels seen in Western Europe within a few decades. The origins of the health crisis under Communism and its persistence in Russia, Ukraine, and the other post-Soviet states is a matter of heated debate in both the scientific and general literature. Whatever the cause, the crisis serves as a warning against unqualified Panglossian optimism. Likewise, the emergence of HIV/AIDS and the associated reemergence of tuberculosis make clear that all future estimates of improvement in public health must take into account the potential for severe reversals.

Overall, however, the last half-century has seen unprecedented convergence in mortality patterns around the world. While rich countries still lead in life expectancy, the gap between these leaders and most developing countries has shrunk substantially. In fact, there has been more convergence in demography than in any other aspect of modernization. For example, consider Latin America as a whole, where the United Nations estimates current life expectancy is seventy-two years, and GDP per head (adjusted for inflation and other factors) is below $4,000, according to the Organization for Economic Cooperation and Development. Now consider the United States. Life expectancy in the United States was seventy-two years as recently as the early 1970s. In contrast, the U.S. GDP per head exceeded $4,000 by 1900. Latin America is a century behind the United States in income growth, but only thirty to thirty-five years behind in life expectancy. We can make similar comparisons for most developing countries. And though the gaps in educational attainment or urbanization are somewhat smaller than in GDP per head, none of the other conventional quantitative indices of development has converged as rapidly as demography.

In recent decades there has also been a striking convergence in fertility, which has declined rapidly in most countries. More than half of the world's population now lives in countries or regions in which fertility is below the level needed for intergenerational replacement.[2] In most of Southern Europe (including Italy and Spain) and in most of Central and Eastern Europe, the total fertility rate (the number of children born per woman) is below 1.3. Similar values are now seen in Japan, South Korea, and many of the more developed parts of China. Even some countries that might seem unlikely candidates have experienced rapid fertility decline. In Iran, for example, fertility fell from over six children per woman to just over two between the mid-1980s and mid-1990s. In contrast, fertility in the United States has seemed to defy gravity, staying close to or even above the replacement level for the last two decades. Among the developing countries in which fertility is now lower than in the United States are China, Brazil, Thailand, and Tunisia. If the trends of the last twenty-five years continue for another decade or so, the U.S. fertility level will be well above the median for the human population as a whole.

The very speed of fertility decline in many countries will produce an exaggerated form of aging. While aging is an inevitable and global phenomenon, countries in which fertility has fallen rapidly will experience a form of 'super aging' in the middle decades of this century. The baby boom cohorts of Southern Europe or the pretransition cohorts in China are very large compared to those that followed, and their getting old will greatly exacerbate any problems that aging generates.

2 Chris Wilson, "Fertility Below Replacement Level," *Science* 304 (2004): 207 – 209.

There is also a sense in which aging can be 'locked in' as part of a country's demographic regime through a form of negative momentum. For example, in Southern Europe, the large number of baby boomers moving through the childbearing ages has disguised the very low fertility rate of recent decades. The largest age groups at present are those ages 25 to 39. In the coming decades, however, the much smaller cohorts born since the mid-1980s will be in the reproductive ages. Unless these cohorts (currently ages 0 to 19) have much higher fertility than their parents, the number of births in countries such as Italy and Spain will shrink even more rapidly in the future than it has so far. In contrast, the United States and other countries in which fertility has stabilized at close to the replacement level (in Europe, they include France and the Nordic countries) will face much less severe challenges from demographic disruption.

The future is always uncertain to some degree, but when trends have been so clear and so consistent for decades, they form a solid basis for prediction. It is very close to certain that aging will be one of the defining global phenomena in the twenty-first century. The ways in which societies choose to adapt to this new reality will test the old adage that "demography is destiny." Fatalism, however, is uncalled for – to a substantial degree we can still choose our future. However, demography does impose strong constraints on the range of feasible options. Taking these constraints into account is the basis for informed reactions to the challenges posed by aging.

Henry J. Aaron

Longer life spans: boon or burden?

'Aging angst' has become a booming industry among scholars. For example, the ethicist Leon Kass and others argue that, on a personal level, increasing longevity may deprive life of its savor and undermine the quest to achieve. Kass states, "If you push those [mortality] limits back, if those limits become out of sight, we are not inclined to build cathedrals or write the B Minor Mass, or write Shakespeare's sonnets and things of that sort."[1] Kass never says how much of an increase in longevity is too much, only that if science were able to slow aging, it would put humankind on a slippery slope to immortality, with all its seductive and corrosive effects.

Meanwhile, on a societal level, economists like Laurence Kotlikoff and Scott Burns worry that the growing percentage of the elderly in the population portends economic calamity:

> Let your mind wander toward the future. Move, slowly, to the year 2030 . . . . What do you see? You see a country [the United States] whose collective population is older than that in Florida today. You see a country where walkers outnumber strollers. You see a country with twice as many retirees, but only 15 percent more workers to support them. You see a country with large numbers of impoverished elderly citizens languishing in understaffed, overcrowded, substandard nursing homes. You see a government in desperate trouble. It's raising taxes sky high, drastically cutting retirement and health benefits, slashing defense, education, and other critical spending, and borrowing far be-

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1 Transcript of interview of Leon Kass by Morton Kondracke, <http://www.sagecrossroads.net/Default.aspx?tabid=60>. Parts of this essay draw on chapter 2 of Henry J. Aaron and Robert D. Reischauer, *Countdown to Reform: The Great Social Security Debate* (New York: Century Foundation Press, 2001). The views expressed here are my own and do not necessarily reflect those of the trustees, officers, or staff of The Brookings Institution.

> yond its capacity to repay. It's also printing tons of money to 'meet' its bills. You see major tax evasion, high and rising rates of inflation, a growing underground economy, a rapidly depreciating currency, and more people exiting than entering the country. They are leaving because they're sure things will get still worse.

What is going on here?

The prospect of living long lives, with physical and mental capacities intact, has long occupied the human imagination. To be sure, the ability to slow or prevent the onset of serious illnesses and even aging may create risks. But the harm that may result if something is done to excess does not require forgoing the good that results from doing the same thing in moderation. George Will illustrates this in his case for therapeutic cloning:

> Life . . . is lived on a slippery slope: taxation could become confiscation; police could become gestapos. But the benefits from taxation and police make us willing to wager that our judgment can stop slides down dangerous slopes.[2]

Warnings that a growing elderly population threatens national well-being are of a different character, but are also odd. Population aging can be delayed if birth rates remain high and the population continues to expand. Until population stabilizes, increasing longevity can coexist with a stable, low fraction of the population that is elderly. Of course, unlimited population growth creates problems of its own. It evokes specters of 'standing room only,' natural resource exhaustion, environmental degradation, and – at least for poor nations – inescapable poverty. Of course, population growth must end. When it does, increased longevity means an older population. Keynes had only half the story: in the long run we will, indeed, all be dead, but with rising longevity we will be old first.

Despite a widespread desire to prolong life, the human species for millennia made no progress toward fulfilling it. Even tiny increases in longevity sustained over the numberless generations of human existence would have resulted in life spans far greater than any now observed. Instead, until the modern economic era, few infants lived to experience what now would be called old age.

Nearly all of the current extension of life spans is a by-product of rising incomes – the result of the Industrial Revolution and the science that produced it. Before the Industrial Revolution, the elderly formed a small fraction of the population because people died young and birth rates were high.[3] In no European nation did as much as 5 percent of the population reach age 65 until the middle of the nineteenth century; in none did 10 percent of the population reach age 65 until after 1930. Now, projections indicate that by the year 2050 more than 20 percent of the population will exceed age 65 in most developed nations, and in several the proportion will approach or exceed 30 percent.[4]

Visions of the United States as a nation of doddering codgers notwithstanding, the U.S. population is projected to

2 George Will, "Column," *The Washington Post*, August 4, 2005.

3 Paradoxically, famine, which reduces life expectancy, could increase the proportion of the population that is elderly because it also causes birth rates to fall. For analogous reasons so could emigration of the young.

4 U.N. projections indicate that more than 35 percent of the population in Japan and Italy will be over age 65. According to U.N. projections, 20.9 percent of the population in the United States will be over age 65, the lowest proportion among developed nations.

remain among the youngest in the developed world because of its relatively high birth and immigration rates. Although the proportion of the U.S. population over age 65 will rise from 12.3 percent in 2005 to 20.6 percent in 2050, the labor force will grow, not shrink, by 29 percent over that period. In sharp contrast, the proportion of the Japanese population over age 65 is already 19.7 percent and is projected to rise to 35.9 percent by 2050. The Organization for Economic Cooperation and Development projects that the Japanese labor force will shrink by more than one-third between 2005 and 2050.[5]

So, if one embraces a dismal vision of the demographic future in the United States, then one must tremble at the truly unspeakable prospects confronting France, Germany, Japan, and Italy. In fact, it's hard to figure out where Americans, who according to Kotlikoff and Burns will be fleeing their wreck of a nation, would actually go. Those who see population aging as a source of collective calamity need to explain why the achievement of sustained economic advance and the deferral of death and of physical and mental decline – all age-old goals of human striving – is a global calamity.

To be sure, extended life expectancy will pose a variety of challenges. In all developed nations, public budgets bear more of the cost of care and support for the elderly than for children. Population aging will therefore tend to push up tax rates. Increased longevity could also create serious social and economic challenges if the years of extended life are ones of mental and physical infirmity; but prospective medical advances promise treatments and, possibly, cures for conditions that produce physical and mental decline. For the most part, the increase in life expectancy made possible by rising incomes, improved public health, and medical advances is a monumental achievement. In the United States, population aging, like the post–World War II baby boom, will doubtless require some quite significant economic and social adjustments, but the adjustments are straightforward and require no more than honest political leadership.

Longer life spans: boon or burden?

I shall begin this survey by recalling what growing old meant to previous generations in the United States and juxtapose a realistic image of what becoming old will mean for our children and grandchildren. I shall then outline the genuine economic problems that increasing longevity and population aging will raise and the steps that will be necessary to deal with them.

A scrim of forgetfulness shields us from the rather ugly reality of growing old in the America of just a few generations past. Let us draw back that curtain to examine what growing old meant for the generations born in 1860, 1890, and 1930.[6]

The 1860 cohort was born in a nation that still treated slavery as a constitutional right. A quarter of those born in 1860 died before turning age 20, half before reaching age 65. Living conditions and public sanitation were appalling by today's standards: few houses had indoor plumbing, and few cities had municipal water and sewer systems. Sur-

5 Thai Than Dang, Pablo Antolin, and Howard Oxley, "Fiscal Implications of Ageing: Projections of Age-Related Spending," Organization for Economic Cooperation and Development, Economics Department Papers No. 305, September 19, 2001. The Japanese labor force is projected to decline on average by 0.9 percentage points annually from 2000 to 2050.

6 Aaron and Reischauer, *Countdown to Reform*.

gery was uncommon and dangerous because surgical technique was primitive and anesthesia was dangerous. Inoculations were uncommon. Childhood diseases winnowed the young, and pneumonia was known as the 'widow's friend.'

By current standards, the 1860 cohort was a nation of educational dropouts, although the United States led the world in mass education. Out of every hundred students who started primary school, seventy finished, twelve completed high school, and three graduated from college. Economic growth was rapid but uneven. The U.S. economy underwent thirteen economic contractions between 1885 and 1925; many were catastrophic by modern standards. Output fell 7 percent following the 1893 panic, 8 percent during the 1907 – 1908 depression, and 6 percent on the eve of World War I. Since World War II, output has never fallen more than 3.7 percent in any recession.

Women gave birth to an average of more than five children. The backbreaking job of caring for children, husbands, brothers, sisters, and parents in a world without washing machines, vacuum cleaners, refrigerators, or dishwashers was borne, typically by women, until death and lightened only as family members died or moved away. Once married, few white women worked outside the home. Those who worked for pay almost invariably performed menial tasks. Many women, especially African American women, were domestics.

Old age was not a passage to a 'new mode of living,' but a continuation of what life had been when one was young. Three-quarters of men born in 1860 and still alive at age 65 continued to work for pay until death, disability, or economic catastrophe intervened. Such a catastrophe – the Great Depression – did intervene when the 1860 cohort was 69-years-old. By 1932, a quarter of the work force was unemployed. The elderly were more likely than the young to lose their jobs and less likely to find new ones. Protracted unemployment, bank failures, plunging stock prices, and collapsing real-estate values destroyed the savings of those in the middle and working classes who had scrimped and saved for retirement. Private charities were overwhelmed, and public charity dried up as state and municipal tax collections plummeted. Only a few Civil War veterans and their widows received small pensions; otherwise, private pensions were rare. The first Social Security check was not paid until the 1860 cohort reached age 80, and few were eligible for benefits. For the one-third of the 1860 cohort who survived to their sixty-ninth birthdays, the final years were generally grim.

America's 1890 cohort also lived through boom and bust. World War I ended a recession. With peace came another recession; unemployment reached 12 percent. The 1920s brought boom, except on the farm. The year 1929 ushered in twelve years that blighted what should have been this cohort's prime earning years. Too old to fight in World War II, the men of the 1890 cohort worked to support their sons at the front. Women left home for the paid labor force, freed from traditional jobs as secretaries, teachers, social workers, and nurses, to become machinists and assembly-line operatives.

Like its forebears, the 1890 cohort suffered high rates of infant mortality. Although this cohort benefited from steady, if undramatic, improvements in health and education, more than one-third of 20-year-old women and two-fifths of 20-year-old men did not

live to see their sixty-fifth birthdays. Eighty percent of unmarried elderly women and half of unmarried elderly men had been widowed. Four-fifths of this cohort finished primary school, one-fourth graduated from high school, but only one in twenty earned a college degree.

When this cohort reached age 65 in the mid-1950s, fewer than half had health insurance. Coverage was often uncertain because insurers could raise premiums sharply or refuse to renew coverage of those whose health had begun to deteriorate. Because health expenses of the elderly, even when adjusted for inflation, were less than one-tenth of what they are today, medical outlays were a threat only for the minority who became seriously ill. But in one of the most striking social changes of the late twentieth century, a spell in a nursing home became common. By the late 1970s, roughly a quarter of the 1890 cohort survivors were residing in nursing homes.

Congress passed the Social Security Act of 1935, subsequently increasing benefits and extending coverage in 1939 and again in 1950. Because of these liberalizations, members of the 1890 cohort received benefits far greater than the earmarked payroll taxes they and their employers had paid. Still, benefits were modest – only about 32 percent of taxable earnings of full-time covered workers. And since roughly half of U.S. jobs were not covered until the 1950 legislation broadened coverage, many members of the 1890 cohort did not receive benefits at all. Furthermore, private pensions covered only about a quarter of members of the 1890 cohort. Even workers who were covered typically received meager benefits because most had not worked long enough under these plans to have earned meaningful benefits. With insufficient income to retire, two-thirds of surviving men from the 1890 cohort were still working at age 65, nearly half at age 70, and 30 percent at age 75. More than one-third had incomes below official poverty thresholds.

The 2.6 million American children born in 1930 enjoyed advantages unavailable to previous generations. Nearly all finished primary school. Seven in ten graduated from high school. Partly because of the G.I. Bill for Korean War veterans, one man in five and one woman in nine graduated from college. Women no longer automatically withdrew from the labor force after marriage; those who did often reentered when still young. Just over one-third worked outside the home when they were age 30, but three-fifths did at age 50, and two-fifths still worked for pay at age 60.

If the educational achievements of the 1930 cohort were striking, the economic advances were breathtaking. Between the end of World War II and the mid-1970s, output per person more than doubled. At the start of their working lives, members of the 1930 cohort earned hourly wages three times higher than members of the 1890 cohort had earned in their first jobs. By the time the 1930 cohort turned age 65, their average earnings had risen by another one-third. Post–World War II recessions, though numerous, were shallow compared with the economic paroxysms of earlier eras. Furthermore, unemployment compensation, also created by the Social Security Act of 1935, cushioned the shock for those who did lose jobs – for up to six months in normal times and even longer during recessions.

Higher incomes, medical advances, and improved working conditions combined to boost life expectancy for the 1930 cohort. Two-thirds of men and over

three-quarters of women born in 1930 lived to celebrate their sixty-fifth birthdays. Four-fifths of 65-year-old men and three-fifths of 65-year-old women still lived with a spouse.

As they approached retirement age in the mid-1990s, members of the 1930 cohort had options and resources few of their parents had enjoyed. Most had assets that provided substantial financial security. Social Security benefits, averaging $8,500 a year for individuals and $12,000 for couples, were fully protected against erosion by inflation. One-third of the 1930 cohort received private pensions, although the amounts were modest – a median of less than $7,000 a year. Further, more than four in five members of the 1930 cohort owned their own homes at retirement. Most had benefited from the postwar real-estate boom that tripled the real value of owner-occupied housing between 1950 and 1995. The 1930 cohort also had better protection against medical costs than ever before. Medicare, enacted in 1965, provided basic health insurance coverage for the elderly and the disabled while eight in ten also had supplementary coverage.

Increasingly workers retired years before they died. One-third of men in the 1930 cohort stopped working before age 62, two-thirds before age 65. Average living standards approximated those of younger adults. Averages, however, concealed large disparities: only 4.3 percent of elderly couples were poor in 1996, compared to 18 percent of elderly single men, 20 percent of elderly single women, and 36 percent of elderly single African American women. Whatever the future holds for the final years of the 1930 cohort, its circumstances represent a revolutionary improvement over the experiences of their predecessors.

America's 1960 cohort was better educated than any of its forebears. Only one in eight dropped out of high school. Half attended college and nearly one-fourth earned a bachelor's degree. The fraction of the 1960 cohort with postbaccalaureate education matched the share of the 1860 cohort who had completed high school. But not all advanced at the same pace. African Americans were only two-thirds as likely as whites to earn a college degree, and barely half of Hispanics completed high school.

Even if the earnings of men with little education grew more slowly than their parents' pay had, the 1960 cohort earned more on their first jobs than their parents had three decades earlier. The jobs filled by members of the 1960 cohort also required less brawn and more brain than had jobs in the past. Three-fifths of men and 90 percent of women in the 1960 cohort worked in white-collar or service-sector jobs. Still, roughly one-quarter of men and a small but growing fraction of women worked as craftsmen, mechanics, miners, machine operators, laborers, truck drivers, or in other physically strenuous jobs that become increasing difficult to perform as one ages. Women were better educated, worked more hours, stayed in the labor force with fewer interruptions, and earned much more than women had previously. As a result, more will be entitled to their own private pensions and to Social Security based on their earnings rather than their husbands'.

Members of the 1960 cohort have told pollsters that they hope to retire earlier than have past generations. Unfortunately, they have done little to prepare economically for that event. By 2000, only 31 percent of those born between 1954 and 1964 had nonhousing assets worth more than $100,000, and 49 percent had accumulated less than $50,000,

a sum that would support an annuity of less than $4,000 a year. In their failure to save, the 1960 cohort differ little from their forebears, who began to save, if at all, only in their forties and fifties. Members of the 1960 cohort may find it even harder to save when they reach those ages, though, because many married late and deferred childbearing. As a result, many will face tuition bills and other costs of childrearing until late in their lives.

On the bright side, more members of the 1960 cohort will have more sizeable pensions than previous generations. The declining fraction of employees with pensions tied to previous earnings, so-called defined-benefit plans, will find them more secure than in the past because the Employee Retirement Income Security Act of 1974 set vesting rules and the Pension Benefit Guaranty Corporation guarantees all or much of promised pensions. On the other hand, the massive shift to pensions whose value depends on the market price of stocks and other assets, so-called defined-contribution plans, means that the pensions of the 1960 cohort will face the risk of losing value just when they are needed. If the pensions are not converted into annuities, these risks will persist even after benefits are being paid. More than previous cohorts, members of the 1960 cohort will also confront the possibility that they will outlive their assets. One-fifth of men who reach age 65 are projected to be alive at age 90, and half of women alive at age 65 are expected to live past their eighty-seventh birthdays.

If members of the 1960 cohort retire when they say they will, those who reach retirement age will spend an average of roughly one-third of their adult lives in retirement. But retirement patterns may change as rising budgetary costs force cutbacks in publicly financed pension and health benefits. Out-of-pocket medical expenditures may discourage older people from leaving primary jobs as soon as they now do or from withdrawing from the labor force completely.

Undeterred by the demonstrated incapacity of even the brightest people to anticipate future conditions or events, many claim to see clearly into the distant future. David Cutler, a Harvard professor and dean, once spoke disparagingly of "spreadsheet policy analysis," the extraordinary disposition of some analysts to take seriously the mindless extrapolation of unreliable assumptions decades or even centuries into the future. What should be clear to all who try to anticipate the implications of population aging for today's and tomorrow's newborns is that only a few things are clear.

One, the proportion of the population that is elderly will increase. This trend is almost certain because the large cohorts of baby boomers who will start reaching age 65 in 2008 are already alive. Almost as certain is that tomorrow's elderly, like today's, will be mostly women. Of those over age 65, 58 percent are female; of those over age 85, 69 percent are female. Female life expectancy exceeds male life expectancy by about five years. As women are also typically younger than their husbands, women are more likely to outlive their husbands and can anticipate about ten years of widowhood.[7] It is also likely that life expectancy will con-

7 The Social Security Administration estimates that among couples in which the husband is 65 and the wife is 63, 54 percent of women and 45 percent of men will outlive their spouses by a year or more (the remainder will die in the same year). Women will outlive their husbands by an average of 10.84 years; widowers will out-

tinue to increase, though by how much remains highly uncertain.[8] However, a drop in longevity, caused by widespread obesity or a global pandemic that science is unable to control, is not out of the question.

In any event, the physical and mental condition of the elderly during these added years counts more than the mere number of years added to the human life span.[9] A nation swarming with doddering seniors incapable of working or even of caring for themselves would face nasty challenges. On the other hand, a nation replete with mentally and physically active elders who might even delay retirement a few years would enjoy an extraordinary economic and social opportunity. The speed and character of advances in medical knowledge give reason for optimism, if not for confidence, that increased longevity will lengthen life, not prolong dying. Understanding and controlling the processes that underlie Alzheimer's disease and other forms of senile dementia and arthritis are within the reach of medical science.

Subject to these uncertainties, the nation of 2050 is quite likely to be richer and better educated than its forebears, even if the rate at which longevity increases slows. Growth of per capita income will continue as the fruits of information technology, such as data processing that abets advances in molecular biology, continue to spread.[10] According to estimates by Kevin Murphy and Robert Topel, the welfare gain from increased longevity between 1970 and 2000 was worth about as much as all economic growth over that period.[11] Factors other than advances in health care contributed to this increase, of course. But improvements in the treatment of heart attacks and reductions in the number of low–birth weight infants yielded benefits worth about six and five times the added cost of medical care respectively.[12] And eliminating half the deaths from heart disease or cancer would produce benefits greater than annual GDP to current and future Americans. Moreover, these estimates make no specific allowance for enhancements in the quality of life that would result from better medical care.

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live their wives by an average of 9.75 years. Personal communication from Stephen Goss, chief actuary of the Social Security Administration.

8 James Vaupel thinks that today's newborns will typically live into the next century. Other demographers simply extrapolate trends of the more or less recent past and predict that life expectancy will continue to increase one or two years with each passing decade. Jay Olshansky and various colleagues worry that obesity, pandemics, or other events will reverse the increase in life expectancy.

9 Alexander M. Capron, "Ethical Aspects of Major Increases in Life Span and Life Expectancy," and Margaret Battin, "Comments," in Henry J. Aaron and William B. Schwartz, *Coping with Methuselah: The Impact of Molecular Biology on Medicine and Society* (Washington, D.C.: Brookings Institution Press, 2004), 198–234, 235–246.

10 J. Bradford DeLong, Claudia Goldin, and Lawrence F. Katz, "Sustaining U.S. Economic Growth," in *Agenda for the Nation*, ed. Henry J. Aaron, James Lindsay, and Pietro Nivola (Washington, D.C.: Brookings Institution Press, 2003), 17–60.

11 Kevin M. Murphy and Robert H. Topel place the gain from increased longevity at $3.2 trillion a year. GDP rose from just over $1 trillion in 1970 to just over $9 trillion in 2000. See Kevin M. Murphy and Robert H. Topel, "The Value of Health and Longevity," The National Bureau of Economic Research, Working Paper 11405, June 2005.

12 David M. Cutler and Mark C. McClellan, "Is Technological Change in Medicine Worth It?" *Health Affairs* 20 (5) (September/October 2001): 11–29.

It is possible that future advances may not bring benefits as large as those of past innovations. The twenty-first century has a tough act to follow: the twentieth century saw massive reductions in infant and childhood mortality, the introduction of artificial joints and CT and MRI scans, and the virtual elimination of broad classes of infectious diseases. But this century has opened auspiciously with the sequencing of the human genome, an event that may reveal the fundamental processes of particular illnesses and of biological aging and senescence and heralds the possibility of individualized medicine, where treatments are tailored to the specific biological characteristics of each person. Even if the twenty-first century does not live up to the more overheated expectations of some observers, there is good reason to hope that Alzheimer's disease, diabetes, and some forms of cancer will become curable or even preventable. These improvements will be costly, however. In fact, they are likely to be so expensive that they will force extremely difficult and divisive political choices and economic tradeoffs. But technical advance will be a cruel tease if few can afford it.

Total spending on the products made possible by scientific revolutions typically increases, even as the prices of these products fall. The automobile, the airplane, television, and the computer reduced the price of moving a person or a ton of merchandise a mile, of hearing an opera or seeing a drama, and of carrying out an arithmetic computation. At the same time, they raised total spending on these activities because they raised the standards of quality, thus increasing the quantities that people demanded. No one bewailed the growth in the share of income devoted to transportation, entertainment, or computation, however. Instead, as other, less satisfying forms of consumption gave way to the new technologies, people celebrated the improvement in living standards.

So also demand for medical treatments has dramatically increased as medical advances have improved the chances for beneficial outcomes while reducing the price of achieving these outcomes. Largely because of such advances, total U.S. spending on health care multiplied more than ninefold and tripled as a share of GDP between 1960 and 2003. There is every reason to expect future medical advances to add to age-adjusted, per capita spending on health care. Population aging will amplify this growth, but advances in medical technology are likely to remain the principal force driving up health-care spending.[13]

If health-care spending were to continue growing at the same rate as in the past half century, about 2.5 percentage points a year faster than the growth of per capita income, the fraction of income devoted to health care would reach 33.6 percent in 2030 and 36.1 percent in 2040. Increases in health-care spending would claim half of income growth by 2022 and all of it by 2051. If Medicare and Medicaid spending were to rise at the same rate, outlays on these two programs alone would rise from 4.2 percent of GDP in 2005 to 11.5 percent by 2030, and 16.1 percent by 2040.[14] For

13 Per capita health-care spending rises until patients are in their eighties and then it actually falls.

14 Henry J. Aaron and Jack Meyer, "Health," in *Restoring Fiscal Sanity: The Long-term Challenge*, ed. Alice Rivlin and Isabel Sawhill (Washington, D.C.: Brookings Institution Press, 2005). These projections are taken from the Congressional Budget Office.

purposes of comparison, all income and payroll taxes combined will comprise 16 percent of GDP in 2006.

These projections suggest that taxes, premiums, and cost sharing will have to increase and that coverage will have to be restricted. A more difficult problem arises from the fact that most health care is consumed during episodes of illness when total spending is so high that any adequate insurance plan will cover all, or nearly all, costs at the margin. In this situation, patients have economic incentives to seek, and conventionally reimbursed providers have every incentive to assure that patients receive, all care however high the cost or low the benefit.

Health-care rationing curbs such high-cost, low-benefit care for well-insured patients. Most people and all politicians recoil now at the prospect of health-care rationing. This reaction is misplaced because such rationing would improve welfare by redirecting resources from uses that produce benefits smaller than cost and make them available for services that produce benefits greater than cost. Whether the nation can ration health care accurately and fairly, though, is far from certain, but trends in health care indicate that a national debate about health-care rationing is inescapable.[15]

Even with higher cost sharing and well-designed rationing, Americans – and citizens of all other advanced nations – are going to end up paying far more than they now do for health care. Population aging will intensify this trend.

Even if maturity brings certain compensations and opportunities,[16] no one welcomes the loss of physical and mental capacities associated with aging. But the problems that aging individuals face is not the cause of 'aging angst.' Rather, it stems from a sense that a large increase in the fraction of people who are 'old' will make life much less attractive for the young. The fear is that the elderly will be economically inactive and otherwise unproductive, that they will not have saved enough during their economically active years to provide for themselves during their inactive years, and thus, that they will impose crushing tax burdens on the declining fraction of the population who are economically active.

It is certainly possible for nations to bring calamity on themselves through mismanaged policies, as the histories of Argentina throughout the twentieth century, most of Africa after the end of colonial rule, and the Russian empire under communism clearly attest. But we can manage the problems of population aging easily. To do so, American policymakers need to keep a few basic facts in mind. First, apart from borrowing or lending from foreigners, all national consumption comes from currently produced goods and services. How that consumption is divided between the economically active and inactive depends on the relative size of these two groups and their relative living standards. Second, consumption by the economically inactive can be financed either by their own past savings or by current taxes on the economically active. Third, past savings are responsible for today's capital

15 Henry J. Aaron, William B. Schwartz, and Melissa Cox, *Can We Say No: The Challenge of Rationing Health Care* (Washington, D.C.: Brookings Institution Press, 2005).

16 George E. Vaillant, *Aging Well: Surprising Guideposts to a Happier Life from the Landmark Harvard Study of Adult Development* (Boston: Little, Brown, 2002).

stock, which influences today's productive capacity. Finally, past savings also can be used to support today's elderly.

The lesson of these simple economic relations is straightforward. Americans can prepare now to meet the macroeconomic 'challenge' of aging by insisting on public policies to promote high national saving. That will add to tomorrow's productive capacity. High saving would also reduce borrowing from abroad, which generates debts to foreigners that tomorrow's active workers will have to either repay or pay debt service.

Yet recent economic policy has moved resolutely in the direction of encouraging consumption with permanent tax cuts. Recent discussions of pension reform have also lacked explicit proposals to encourage future generations of workers to delay retirement, which would reduce pension claims. By extending drug coverage to the elderly and disabled, Congress has further committed the nation to providing a needed benefit but failed to pay for it, thereby increasing borrowing and deepening the future fiscal challenges of population aging. Measured over the next seventy-five years, the Medicare Modernization Act will also add to federal borrowing an amount nearly twice the projected shortfall in Social Security. Thus, current policy has aggravated, rather than ameliorated, the fiscal problems of population aging.

The first step in dealing with the 'aging problem' is to avoid public policies that enlarge it.

The second step is to recognize that the U.S. 'aging problem' is among the smallest in the developed world.

The third step is to recognize that although population aging will present some fiscal challenges, it is the by-product of a monumentally beneficial achievement – increased longevity – and an inevitability – declining birth rates.

Longer life spans will doubtless create some problems. But as the old saying goes: Consider the alternative.

Longer life spans: boon or burden?

Sarah Harper

Mature societies: planning for our future selves

As the new millennium begins, the world is entering into demographic maturity. Western Europe now has more people over age 60 than under age 15. Asia will follow by the year 2040, the Americas shortly after. But while Western Europe took more than a century to go through this demographic transition, Asia will move through it in less than twenty-five years. By 2050, more people globally will be over age 50 than under age 15.

The extent of population aging is truly staggering. By 2030, nearly half of Western Europe's population will be over age 50, with a life expectancy at 50 of another forty years. That is, half of this population will be between 50 and 100, a quarter over 65, and 15 percent over 75. Yet, in terms of numbers, it is to the developing world we must look. Two-thirds of the world's older population already resides in developing countries, with the absolute numbers of older people in these regions projected to double to just under a billion within twenty-five years and increase to 2 billion by the middle of the century. The majority of these individuals are already born. Indeed, we are tomorrow's elderly.

We are not talking here about the so-called age wave. Many people mistakenly believe that population aging is solely the result of the baby boom generation moving its way up the population pyramid. Rather, demographic maturing is a global trend that heralds long-term shifts in individual and societal behavior – changes that are likely to restructure societies for much of the foreseeable future.

Powering this maturing trend, in reality, are dramatic declines in fertility and increases in the normal life span. By the mid-1980s, most Western-style countries were experiencing historically low fertility levels. Initially, calendar measures of fertility indicated a plateau in reproduction during the 1930s and 1940s – what we consider the end of the classical demographic transition – before a further drop to levels significantly below replacement level occurred. However, generational measures reveal that

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fertility levels had been continuously declining, even through the end of the transition.

Perhaps fundamental norms regarding the desirability of having many children changed radically, or perhaps the economic structure of modern societies reduced the need to bear a lot of children. In any case, low fertility seems to be a characteristic of postindustrial societies: fertility in Europe, for example, remains below the replacement level of 2.1, despite increases in some countries toward the end of the twentieth century. Most strikingly, the past two decades have also seen a steady fall in fertility in Asia and Latin America. Total fertility rate, the number of children a woman of reproductive age will bear in her lifetime, has now dropped to 1.4 in Hong Kong, 1.5 in Singapore, and 1.8 in Korea, Taiwan, and Thailand. Sri Lanka and Chile stand at 2.1 and Brazil at 2.3.

While maximum life span has remained more or less constant, twentieth-century social, economic, and medical innovations have also enabled more people to achieve the maximum life span. Until very recently, for example, the second longest-lived person was born in 1701, with a life span of 113 years. During the same period, however, the number of British centurions alive at any one time was in the hundreds. This figure has now increased to some thirteen thousand, with eighty-seven thousand predicted by 2050. Between now and 2050, the number of centurions in Japan will also rise, from twenty-eight thousand to 1 million. Currently, the world has two hundred and sixty-five thousand centurions; by 2050, it will have 3.8 million. Overall, life expectancy has improved. In 1880, a female baby in Europe could expect to live to age 47; her great-granddaughter born one hundred years later can expect to live to 78. Her baby brother had a life expectancy of forty-four years; his great-grandson now has one of seventy-one. There is thus evidence of a rectangulization of the life curve in the West, with a growing percentage of the population reaching out toward the current maximum life span.

The eventual convergence of the maximum and normal life spans should be seen as a great success. For most individuals born in a society to reach the natural limit to human life in good health and with few frailties is a major achievement for any society. To accomplish this on a global level would be *the* achievement of civilization. For then we would have conquered poverty, disease, famine, and war.

We have already felt the impact of the factors *associated* with demographic aging – falling fertility and mortality, and increasing longevity – and those *contributing* to it – advances in living standards, education, public health, and medicine. But population aging promises to influence decision making even more in the new century and at every level – individual, national, and international. The social, economic, and political areas that this shift may affect include: the labor market, saving and consumption patterns, family and household structure, social interaction and networks, demand for health and welfare services, supply of housing and transportation, leisure and community behavior, and even geopolitical order. Thus, individuals and governments, in both developed and developing countries, must understand the reality of population aging in order to plan societal frameworks and policies appropriate for the demographic challenges and opportunities ahead.

Currently, the *demographic burden hypothesis* dominates public rhetoric. This

hypothesis focuses on four pervasive myths. The first sees Western health-care systems folding under the strain of caring for growing numbers of older people. The second myth fears the ratio of workers to retirees will become so lopsided that many Western economies will collapse. The third envisions families as loose, multigenerational collections of individuals, experiencing more emotional strain, as fewer children are available to take care of elderly parents. Finally, the fourth mistakenly believes that aging is a feature of the developed world alone, with little relevance to developing countries.

An aging population naturally raises concern about increasing health- and social-care costs.[1] U.S. forecasts, for example, show health-care costs accounting for almost 33 percent of GDP by 2030.[2] However, as George Leeson points out, a number of cross-national studies have considered the determinants of health-care costs, but only one has found population age structure – that is, the proportion of the population aged 65 and over – responsible, along with income, lifestyle characteristics, and environmental factors.[3]

Part of the reason for the false rhetoric lies with the methodologies used in some research studies.[4] Often, these studies simply calculate how much health care people at each age use.[5] These current patterns are then applied to the demographics of an aging population to forecast the effects of changing demographics on the cost of health care. The results imply that age contributes between 0.3 and 0.8 percent of annual expenditure growth.[6] But this simplistic way of assessing age-cost effects ignores the fact that the amounts of health care utilized by different age groups also change over time. In fact, a number of studies have revealed that health-care expenditures are concentrated in the period immediately prior to death.[7] Thus,

1 G. W. Leeson, "The Demographics and Economics of UK Health and Social Care for Older Adults" (Oxford Institute of Aging, University of Oxford, 2004), <http://www.ageing.ox.ac.uk/publications/papers/oia%20wp%20304.pdf>; R. Lee and J. Skinner, "Will Ageing Baby-Boomers Bust the Federal Budget?" *Journal of Economic Perspectives* 13 (1) (1999): 117 – 140; B. J. Soldo and M. S. Hill, "Intergenerational Transfers: Economic, Demographic, and Social Perspectives," in *Aging, Kinship, and Social Change*, vol. 13 of the *Annual Review of Gerontology and Geriatrics*, ed. G. L. Maddox and M. P. Lawton (New York: Springer, 1994).

2 S. T. Burner, D. R. Waldo, and D. R. McKusick, "National Health Expenditure Projections Through 2030," *Health Care Financing Review* 14 (1) (1992): 1 – 29; M. J. Warshawsky, "Projections of Health Care Expenditures as a Share of the GDP: Actuarial and Macroeconomic Approaches," *Health Services Research* 29 (3) (1994): 293 – 313.

3 Leeson, "The Demographics and Economics of UK Health and Social Care for Older Adults."

4 S. Petrou, J. Henderson, T. Roberts, and M.-A. Martin, "Recent Economic Evaluations of Antenatal Screening: A Systematic Review and Critique," *Journal of Medical Screening* 7 (2) (2000): 59 – 73.

5 M. Seshamani and A. Gray, "The Impact of Ageing on Expenditures in the National Health Service," *Age and Ageing* 31 (4) (2002): 287 – 294.

6 OECD, "Reforming Public Pensions," Social Policy Studies no. 5 (Paris: Organization for Economic Cooperation and Development, 1988); U.-G. Gerdtham, "The Impact of Aging on Health Care Expenditure in Sweden," *Health Policy* 24 (1) (1993): 1 – 8; M. L. Barer et al., "Trends in Use of Medical Services by the Elderly in British Columbia," *Canadian Medical Association Journal* 141 (1) (1989): 39 – 45.

7 K. McGrail et al., "Age, Costs of Acute and Long-Term Care and Proximity to Death: Evidence for 1987 – 88 and 1994 – 1995 in British

as we postpone death to later and later ages, the health-care costs associated with preceding ages should grow lighter.

Within the United States, in particular, the growing elderly population portends especially large increases in health-care costs because of both the Medicaid and Medicare programs. Currently, Medicare provides almost universal coverage of hospital and physician expenses to those over age 65, while Medicaid provides medical care to eligible persons of all ages, as well as nursing home expenses for the elderly. But the United States spends a lot on health care in general. In addition, it shifts the payment for health care from private to public sources at age 65.[8] Thus, the fault for the feared Medicare crisis lies not so much with the increasing numbers of old people, especially since forecasts show a long-term decline in U.S. disability rates.[9] Instead, the responsibility belongs to rising per capita health-care expenditures in a policy framework that makes these a public liability.

Likewise, the aging of populations also inspires fear of serious fiscal issues, caused in part through a sharp growth in real spending on pensions.[10] Yet, as with many studies of the effects of population aging, these studies rely on current dependency ratios and retirement rates,[11] rather than acknowledging that these measures are period- and cohort-specific and can therefore change. Dalmer Hoskins, Secretary-General of the International Social Security Association, stresses that instead of focusing on the 'burden' of the aging, we must pay attention to the rising number of persons who are able to work but made prematurely inactive.[12] Indeed, as with health-care costs, it is not demographic aging per se, but current policy frameworks, that are liable. The continued commitment by many governments to generous public pensions with high replacement rates both facilitates and encourages retirement at or before age 65, despite evidence of increased longevity.[13]

Columbia," *Age and Ageing* 29 (3) (2000): 249 – 253; C. O'Neill et al., "Age and Proximity to Death as Predictors of GDP Care Costs: Results from a Study of Nursing Home Patients," *Health Economics* 9 (8) (2000): 733 – 738.

8 B. Bosworth and G. T. Burtless, eds., *Aging Societies: The Global Dimension* (Washington, D.C.: Brookings Institution Press, 1998).

9 K. G. Manton, L. S. Corder, and E. Stallard, "Chronic Disability Trends in Elderly United States Populations: 1982 – 1994," *Proceedings of the National Academy of Science* 94 (1997): 2593 – 2598; V. A. Freedman and L. G. Martin, "Understanding Trends in Functional Limitations Among Older Americans," *American Journal of Public Health* 88 (10) (1998): 1457 – 1462; Leeson, "The Demographics and Economics of UK Health and Social Care for Older Adults."

10 Leeson, "The Demographics and Economics of UK Health and Social Care for Older Adults"; OECD, "Ageing and Income: Financial Resources and Retirement in 9 OECD Countries" (Paris: Organization for Economic Cooperation and Development, 2001); R. Lee and S. Tuljapurkar, "Population Forecasting for Fiscal Planning," in A. J. Auerbach and R. Lee, eds., *Demographic Change and Fiscal Policy* (Cambridge: Cambridge University Press, 2000); European Commission, "Public Finances in EMU – 2002," *European Economy no.* 3 (Luxembourg: Office for Official Publications of the European Communities, 2002).

11 OECD, "Ageing and Income."

12 D. Hoskins, "Thinking About Ageing Issues," *International Social Security Review* 55 (1) (2002): 13 – 20.

13 P. S. Heller, *Who Will Pay? : Coping with Aging Societies, Climate Change, and Other Long-Term Fiscal Challenges* (Washington, D.C.: International Monetary Fund, 2003).

Changing these policy frameworks will involve combating the perception that the elderly are economically inactive, both as producers and consumers. Laboratory-based research suggests the decline in physical and mental activity between ages 20 and 70 is negligible; in general, variations *within* age groups far exceed those *between* age groups.[14] Despite this evidence, considerable data indicate that negative perceptions of older workers are still partially responsible for prompting early retirement.[15]

Given the apparent increase in healthy life expectancy for current older cohorts, older men and women should be able to remain economically active longer. Industrialized countries may, therefore, burden themselves with their unwillingness to adapt fiscal and social policies to changing social and cultural attributes. Despite an increasingly tight labor market in many developed countries, employers have reacted very slowly to this. In fact, many have thus far relied on immigration as the panacea for falling birth rates. However, this movement of Asian and Eastern European migrants into Western labor markets is hardly sustainable, as economic growth in the source countries will allow these countries to retain more of their own workers. Further, many predict that countries like China themselves will attract highly skilled labor, especially in the growing and valuable IT sector.[16]

The elderly are also thought to consume less, perpetuating anxiety that population aging will result in slower economic growth. However, the longer individuals work, the more likely it is their consumption rates and patterns will also change. Currently, those over 50 spend primarily on leisure activities rather than on consumer goods. But consumer goods obviously have a limited life span, and as people live longer, they will need to replace what they purchased in their twenties and thirties. Ensuring older workers a place in the labor pool would enable them to consume more later in life. Again, changing preconceived notions of older cohorts and their economic behavior is the key to capitalizing upon the potential of population aging.

The possibility that families will undergo more strain is also ever present in discussions about population aging. These arguments suggest that increasing longevity means greater numbers of older people requiring care, at the same time that declining fertility is shrinking the reservoir of family members available to care for the old. This places pressure on the middle-aged to cope with both dependent children and aging parents.

Rather than the demise of the family, however, more heterogeneous forms of family are emerging from the reality of population aging. These alternative structures include multigenerational

14 C. P. Bird and T. D. Fisher, "Thirty Years Later: Attitudes Toward the Employment of Older Workers," *Journal of Applied Psychology* 71 (1984): 515–517.

15 C. S. Forte and C. L. Hansvick, "Applicant Age as a Subjective Employability Factor: A Study of Workers Over and Under Age Fifty," *Journal of Employment Counselling* 36 (1) (1999): 24–34; E. M. Crimmins, Y. Saito, and S. L. Reynolds, "Further Evidence on Recent Trends in the Prevalence and Incidence of Disability Among Older Americans from Two Sources: The LSOA and the NHIS," *Journal of Gerontology Series B: Psychological Sciences and Social Sciences* 52B (1997): S59–71.

16 D. Arkless, "2005 Manpower Presentation to IBM," Paris, October 2005.

relationships and members not formerly defined as kin.[17] Naturally, these new family arrangements are generating questions about each person's role in the intergenerational transmission of social and economic resources and each person's responsibility for taking care of dependent adults. Extensive work in the United States and Europe has revealed a high level of social and economic transfers despite significant changes in family structure.[18] For example, Vern Bengtson's study shows that while members of Generation X were more likely to have grown up in a home with divorced parents, a full-time working mother, and fewer than two siblings, intergenerational influences remain strong. In fact, alternative and multigenerational families are performing the functions of nuclear families where necessary.

Cross-sectional work in Europe has also demonstrated at least an intention on the part of middle-aged children to take care of older kin. Data from France, for example, show that despite the government's fears of a breakdown in family solidarity, 80 percent of middle-aged children said they would either provide housing for needy parents and in-laws or care for them in their own homes.[19] Scandinavian research has also highlighted the importance of kinship within a modern welfare state, reporting both increased contact with family members and a significantly more positive view of the family as a supportive institution.[20]

As pressing as the concerns of aging in developed countries are, developing countries face the greatest challenge. In forty-five years, three-quarters of the 2 billion elderly people in the world will live in developing countries. Currently, Africa has 40 million people over age 60, Latin America and the Caribbean have 41 million, and the Asia-Pacific region has 600 million. Moreover, these countries are aging very quickly: it will take, on average, twenty-three to twenty-four years for the elderly to go from comprising 7 to 14 percent of the population in many developing countries, a jump that took 115 years for France to achieve. Worse, the elderly in developing countries will probably not be the active, healthy retired but frail, dependent elders.

Older people are among the poorest in every developing country. With the lowest levels of income, education, and literacy, they lack savings, assets, and land; have few skills or capital to invest in productive activity; and have very limited access to jobs, pensions, or other benefits. In fact, many developing countries have yet to establish even minimal social insurance schemes. Healthcare provision, for example, will pose a real challenge. With the epidemiological transition now underway globally, the emphasis is shifting from infectious and parasitic diseases to chronic and degenerative diseases. The World Health Report 2003 identified cardiovascular disease and lung cancer as two new major global epidemics – brought on not only by environmental factors such as smok-

17 V. L. Bengtson, "Beyond the Nuclear Family: The Increasing Importance of Multigenerational Bonds," *Journal of Marriage and the Family* 63 (2001): 1 – 16.

18 G. W. Leeson, "Changing Patterns of Contact with and Attitudes to the Family in Denmark," *Journal of Intergenerational Relationships* (forthcoming).

19 J. Chwalow, A. Bagnall, C. Baudoin, and F. Elgrably, "France," in I. Philp, ed., *Family Care of Older People in Europe* (Amsterdam: IOS Press, 2001), 27 – 48.

20 Leeson, "Changing Patterns."

ing and diets high in saturated fat, but also because individuals are now living long enough to develop those diseases. Though commonly perceived as a Western disease, cardiovascular disease is now taking more lives in developing countries than in the developed world. But since most health-care systems in these regions are struggling to address acute diseases, including HIV/AIDS and tropical diseases, and tackle infant, child, and maternal mortality, they have no spare resources to develop much needed preventative public-health programs, let alone long-term care strategies.

The second large area of real concern is material security. Most people in less developed countries have no prospect of a secure and sustainable income in their old age. Few, even among workers, receive any public benefits. In contrast to the 84 percent of those over age 60 in OECD countries who receive a pension, under 20 percent in many Latin American countries, under 10 percent in Southeast Asia, and under 5 percent in parts of sub-Saharan Africa do.[21] Not only is political impetus missing, but substantial practical obstacles stand in the way of extending formal insurance coverage to workers in both rural and informal sectors. These range from lack of adequate record keeping identifying the appropriate recipients to the logistics of actually delivering benefits.[22] Even countries with more developed systems tend to exclude large sections of the work force.[23]

Even so, it would be foolhardy to ignore the urgency of developing institutions and policies appropriate for a world that will have 1 billion older adults within the next twenty-five years.

As the baby boom generation moves through old age and dies, and if current low rates of fertility and mortality continue, most developed countries and some less developed countries will probably become fully mature, 'age symmetric' societies over the next few decades. These societies will be historically unique in both their demographic profile and their promise of long, healthy lives for many individuals.[24] Governments have the first two decades of the twenty-first century to develop frameworks for these mature societies.

Currently, three broad approaches exist. Governments can introduce ameliorative policies and simply wait to see what happens when a more age-symmetric population arrives; they can attempt to manipulate population struc-

21 World Bank, "Averting the Old Age Crisis: Policies to Protect the Old and Promote Growth," World Bank Policy Research Report (Oxford: Oxford University Press, 1994).

22 J. H. Schulz, "Economic Security in Old Age: A Family-Government Partnership," in J. Randel, T. German, and D. Ewing, eds., *The Ageing and Development Report: Poverty, Independence and the World's Older People* (London: Earthscan, 1999), 82–97; International Social Security Association, "Report of the Asian Regional Round Table, Meeting on Social Security Protection of the Rural Population in Developing Countries" (Kuala Lumpur: International Social Security Association, 1980).

23 U.S. Social Security Administration, "Social Security Programs Throughout the World," Research Report no. 65 (Washington, D.C.: U.S. Social Security Administration, Office of Policy, Office of Research, Evaluation, and Statistics, 1997).

24 Currently, there are concerns about child and adolescent obesity and diabetes. Given the ability to prolong life, a fall in healthy life expectancy, rather than in life expectancy itself, is possible. This may mean the growth of a frailer, more disabled older population.

tures through increased fertility or immigration; or they can explore and take advantage of the many opportunities a mature population offers. Governments should pursue the latter approach and seize the chance to harness the experience, expertise, and creativity of older people.

Rather than defining themselves only by their demography, societies that take the latter approach will benefit from the possibility of age integration, or more interaction between successive cohorts. This potential to utilize the capabilities of such a large spectrum of cohorts, in different stages of the life course, is unprecedented. Moreover, integration could be a consistent and relatively stabilizing force within societies as they mature. However, these benefits will only accrue if those influencing our governments and economies today turn from fearing such a future and work toward creating the framework for tomorrow's mature societies. Such a framework would include banning age discriminatory practices; encouraging age-integrated behavior; and fully recognizing the eventual frailty, and finality, of old age.

As Matilda Riley argued some thirty years ago, the removal of structural age barriers, such as those to work and education, is essential to an age-integrated workplace – where age does not constrain entry, progress, and exit;[25] and individuals of different ages interact within the same set of structures or roles. Unfortunately, even in those countries with anti–age discrimination legislation, employers are generally reluctant to hire older workers.[26] Until the middle of last century, however, an age-integrated workforce was considered a versatile workforce,[27] and ample evidence pointed to the valuable contributions that older workers made to economic activity.[28] It was widely acknowledged that while capacity changed with age, one could overcome this through retraining and adaptations in the work environment.[29] Such evidence is all the more abundant today.[30] Even within the manufacturing sector, where concern that older workers are not as productive is highest, research shows those aged 55 and older are as productive as those aged 35 to 54, and more productive than those under 35.[31]

25 M. W. Riley, "Age Strata in Social Systems," in R. H. Binstock and E. Shanas, eds., *The Handbook of Aging and the Social Sciences*, 2nd ed. (New York: Van Nostrand Reinhold, 1976).

26 Princeton University Conference on Discrimination in Labor Markets, *Discrimination in Labor Markets*, ed. O. Ashenfelter and A. Rees (Princeton, N.J.: Princeton University Press, 1973); L. A. Bennington and R. Wein, "Anti-discrimination Legislation in Australia: Fair, Effective, Efficient or Irrelevant?" *International Journal of Manpower* 21 (1) (2000): 21 – 33.

27 E. Belbin, "Methods of Training Older Workers," *Ergonomics* 1 (1958): 207 – 211.

28 S. Harper and P. Thane, "The Consolidation of 'Old Age' as a Phase of Life, 1945 – 1965," in M. Jefferys, ed., *Growing Old in the Twentieth Century* (London: Routledge, 1989), 43 – 61.

29 A. T. Welford, *Ageing and Human Skill* (London: Oxford University Press, 1958); E. L. Meier and E. Kerr, "Capabilities of Middle-Aged and Older Workers: A Survey of the Literature," *Industrial Gerontology* (Summer 1976): 147 – 155.

30 T. Warr, "Age and Employment," in H. C. Triandis, M. D. Dunnette, and L. M. Hough, eds., *Handbook of Industrial and Organizational Psychology*, vol. 4, 2nd ed. (Palo Alto, Calif.: Consulting Psychologists Press, 1994), 485 – 550.

31 J. Hellerstein, D. Neumark, and K. Troske, "Wages, Productivity and Worker Characteristics: Evidence from Plant Level Production

Besides general reluctance on the part of employers to hire older workers, there is also evidence that institutional support for age-structured careers has stagnated.[32] Fewer jobs provide increasing rewards and security for longer tenure. Instead, more variable and flexible patterns of career progression and termination have become the norm. Indeed, younger cohorts, currently in early and mid-life, are already growing accustomed to a less rigid labor market with greater access to part-time and flexible working patterns as well as the need to continually update skills.[33]

While the workplace is still slow to move toward more age-inclusive frameworks, some examples exist. The Norwegian model, for one, lays out the key elements of a long-term strategy for facilitating an integrated workforce. These elements include a financial incentive and benefit system that encourages the retention of older workers, more flexible working patterns, continual training and education, and increased dialogue between employers and employees.

Another development of mature societies is the multigenerational, or 'bean pole,' family. It is estimated that three-quarters of all adults will become grandparents.[34] This represents a considerable historical shift. In the United States, for example, nearly one-fifth of all children born in 1900 were orphans before reaching age 18. In contrast, more than two-thirds of those born in 2000 will still have both sets of grandparents at 18.[35] Similarly, at age 30, one-fifth of the 1900 cohort had a living grandparent; three-quarters of those born in 2000 will have at least one living grandparent at 30. The age-integrated family, with members stretching from birth to well into their eighties and nineties, appears to be a growing phenomenon in many Western societies. Bengtson even suggests that in terms of providing support across the life course, multigenerational bonds are becoming more important than nuclear family ties for many Americans.[36]

These multigenerational families have wider policy implications. Despite what considerable public rhetoric predicted, wars between the generations have not erupted. Younger cohorts have not risen up to protest policies that benefit older adults, even if these policies seem counter to their own interests. Anne Foner offers two reasons why people of working age in the United States favor programs that benefit older people. First,

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Functions and Wage Equations," *Journal of Labor Economics* 17 (3) (1999): 409 – 446.

32 K. Loscocco, "Age Integration as a Solution to Work-Family Conflict," *Gerontologist* 40 (3) (2000): 292 – 300.

33 D. Gallie, *Equal Opportunities for Women and Men in Europe? : Eurobarometer 44.3 : Results of an Opinion Survey* (Luxembourg: Office for Official Publications of the European Communities, 1998).

34 R. Giarrusso, M. Silverstein, and V. L. Bengtson, "Family Complexities and the Grandparent Role," *Generations* 20 (1) (1996): 17 – 23; D. Dench, J. Ogg, and K. Thomson, "The Role of Grandparents," in R. Jowell, J. Curtice, A. Park, and K. Thomson, eds., *British Social Attitudes : The 16th Report* (Aldershot: Ashgate, in association with the National Center for Social Research, 1999).

35 P. Uhlenberg, "Intergenerational Support in Sri Lanka," in T. K. Hareven, ed., *Aging and Generational Relations : Life-Course and Cross-Cultural Perspectives* (New York: Aldine de Gruyter, 1996).

36 Bengtson, "Beyond the Nuclear Family: The Increasing Importance of Multigenerational Bonds."

younger people have a stake in protecting public programs for older adults because these programs relieve them of financial responsibility for the elderly people in their own families. Second, younger adults wish to preserve these programs for their own old age.[37]

Of more importance than these self-interested motives, however, is how these policies are mediated within kinship roles and relationships.[38] While public programs operate at the national level, most people actually experience them at the individual, family, or community level. Age-integrated families naturally advance opportunities for younger people to have contact with and knowledge of their older relatives. Through these intimate intergenerational relationships, older people are no longer the other. Thus, even though individuals do not directly benefit from age-friendly policies, these policies feel rewarding because their relatives, with whom they have affective intergenerational ties, are benefiting.

Also, younger people may well receive direct or indirect benefits from these programs. The entitlements for older adults that these programs dispense at the macro level often result in a circulation of benefits from older to younger family members. The relative financial security afforded the old by public programs permits them to contribute to their children and grandchildren. So rather than parents becoming rich at the expense of the young, the young benefit both directly and indirectly from the public funds received by older adults. The possibility for increased interaction between successive generations, which arises from population aging, thus impacts society as well as families.

It is clear that during the last quarter of the twentieth century, the early retirement of healthy, active adults had profound implications for societal preconceptions, and ultimately the definition, of men and women in their fifties and early sixties. In fact, the notion of the 'Third Age,' which Peter Laslett conceived as commencing during an individual's sixties as he or she withdrew from full-time employment, is now seen as beginning at 50. Or even as the Third Age Foundation boldly asserts in a recent UK report: "Older people is the term used for the purposes of this report to refer to those aged 40-plus." Given that individuals at 40 can expect to live fifty more years, are we really to believe that two-thirds of the adult population are now 'old'?

Yet, particularly in Western societies, financial and other services are now labeling those over 50 as the 'older' consumer group; communities are building special housing for those over 50; and governments in many countries are even providing pensions to individuals from age 50. At least one UK local authority now allocates social services for the elderly to those age 50 and older on the grounds that this is politically correct. And many in the sales sector say that identifying a late-life product as one "for those in their fifties" is a good sales technique, as the older consumer will be more likely to purchase a product apparently targeted at those some twenty years younger. However, the social, cultural, and economic dynamics driving this are not necessarily good for the individual.

37 A. Foner, "Age Integration or Age Conflict as Society Ages?" *Gerontologist* 40 (3) (2000): 272 – 276.

38 S. Harper, *Ageing Societies: Myths, Challenges and Opportunities* (London: Hodder Arnold, 2005).

Female menopause, which usually begins around age 50, was the original rationale for making age 50 the transition into old age. Men came to be included in this transition because of the often associated, though never causally associated, change of roles and relationships that occurred around that age. The economic benefit of early retirement was also partially responsible for including men. However, with the aging of life transitions – the delay in finishing full-time education, forming stable adult unions, and becoming a parent – most individuals in their fifties are still active parents, partners, and contributors to community and economic life, as they were when they were in their thirties and forties.

Indeed, given the multiplicity of roles and diversity of life-course experiences, most individuals will spend much of their adulthood moving back and forth among a spectrum of responsibilities with no sense of an abrupt transition at 50. One woman in her fifties, for example, seamlessly shifted between being a granddaughter and grandmother, parent and child, worker and caregiver, and wife. Another, the wife of a U.S. senator, had her last child naturally at 51. Though pregnant and nursing, she was eligible for old people's housing; in England, she would have been targeted for social services and considered to be in her Third Age.

In our eagerness to claim that we have reached later life, we have created an 'age' that starts at 50 and may continue for nearly half a lifetime. As a result, we marginalize the reality of true old age – the increasing frailty and approach of death that comes to the very old. Historians Thomas Cole and W. Andrew Achenbaum have suggested that this process of marginalization began in the nineteenth century. During this period, the acceptance of death as a natural part of the human condition, which could enrich and inform all life, was slowly replaced by the view that willpower and scientific knowledge could postpone physical decline indefinitely. By the middle of the twentieth century, scientific responsibility had completely trumped personal responsibility: modern science, having discovered the 'problem' of old age, resolved to solve it.[40] As Cole remarked, "Unable to infuse decay, dependence, and death with moral and spiritual significance, our culture dreams of abolishing biological aging."[41]

This dream has conjured up a notion of the body not based on human experience. Since we do not see infirmity as a common experience of all stages of human life that is then expanded in later life, the frailty of the old sets them apart from the young.

As societies age, it is important that we recognize the full potential length of active adulthood and enable most individuals to contribute as long as they are able. However, it is equally important that we then accept a period, brief or long, of morbidity and disability at the end of our lives as the reality of old age. Then, societies can redirect resources to those elders in real need, ensuring accommodation and financial, social, and health-care services, regardless of chronological age. This is the reality – old age as an integral part of adulthood.

40 W. A. Achenbaum, *Old Age in the New Land: The American Experience Since 1790* (Baltimore: Johns Hopkins University Press, 1979); T. R. Cole, *The Journey of Life: A Cultural History of Ageing in America* (Cambridge: Cambridge University Press, 1992), 129.

41 Cole, *The Journey of Life*.

Most developed countries began the twentieth century with populations that aged from one old for every ten young, to one old for every two young by the late 1980s, to one old for every young person by the early years of the twenty-first century. Developing countries saw a dramatic increase in absolute numbers of older people, moving from a few million to nearly a billion in that time. Europe also saw three months of life expectancy added at birth every year.

The speed and magnitude of these changes are unprecedented, and their full implications are difficult to grasp. Laslett talked of a cultural lag, in which society has yet to catch up with the current reality of population aging. Riley speaks of a structural lag, whereby institutions have yet to adapt. It is clear that there is also an individual lag, whereby people have yet to readdress their life-course activities in light of their significantly increased potential for long life.

As mature societies develop, we must be wary of confounding age with cohort and life course, implying that those in later life must consistently act in certain ways because of their age. While age-integrated mature societies may display certain characteristics because of their demographic profile, we must also acknowledge the complexities of cohort and period effects: each cohort brings with it specific life dimensions, dynamics, and histories; and each time period introduces particular institutional and structural contexts. All mature societies now have the exciting possibility of age integration. Mature societies are not those that have large numbers and proportions of old people; they are societies in which people live longer.

Paul B. Baltes

Facing our limits: human dignity in the very old

An ancient Greek myth captures a dilemma that still faces us today. The goddess of the Dawn, Eos, persuades Zeus to make her earthly lover Tithonus immortal. But she forgets to ask Zeus to preserve the health and vitality of her lover. As a result, though Tithonus lives on and on, his body and mind begin to fail. Finally, and with pain in her heart, Eos moves her former lover into a separate chamber where he lives forever out of sight.

Like Eos in the myth, some scholars now entertain the prospect of an indeterminate, if not 'limitless,' human life span.[1] But should this dream become a reality, still other students of aging foresee a growing number of Tithonuses – very old and frail people, bereft of mind, body, and human dignity.

The Janus face of aging becomes apparent when we compare what, pursuant to earlier work by Bernice Neugarten and Peter Laslett, my colleagues and I have called the 'Third' and 'Fourth Age' of the human life span.[2] These are dynamic and heuristic concepts, approximations that change with time and exhibit large individual variations. Currently, in developed countries, the Third Age begins, on average, at about age 60; the Fourth Age generally starts around age 80.

In recent decades, a powerful coalition of gerontological scientists, policymakers, and social-technological advances

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1 J. Oeppen and J. W. Vaupel, "Broken Limits to Life Expectancy," *Science* 296 (5570) (May 10, 2002): 1029 – 1031.

2 P. B. Baltes, "On the Incomplete Architecture of Human Ontogeny: Selection, Optimization, and Compensation as Foundation of Developmental Theory," *American Psychologist* 52 (4) (April 1997): 366 – 380; P. B. Baltes and K. U. Mayer, eds., *The Berlin Aging Study: Aging from 70 to 100* (New York: Cambridge University Press, 1999); P. B. Baltes and J. Smith, "New Frontiers in the Future of Aging: From Successful Aging of the Young Old to the Dilemmas of the Fourth Age," *Gerontology* 49 (2) (March/April 2003): 123 – 135.

has contributed to major increases in longevity and improvements in the quality of life for individuals in the Third Age.[3] But theoretical and empirical evidence suggests that further improvement may become more difficult as more and more people reach the Fourth Age. These oldest-old are manifesting a new level of biocultural incompleteness, vulnerability, and unpredictability in their everyday behavior – testing the limits of human functioning as well as of science and policy.

After offering some of the scientific evidence for this contrast between the Third and the Fourth Age, I shall sketch some of its implications, not only for the individual but also for societies. Depending on characteristics such as average age and age distribution, countries will differ in productivity and health costs. Indeed, a population's age distribution may even affect how much money a country has available for international developmental aid.

Thus, it is not surprising that debates over the future of aging differ in marked ways, for instance, in the United States and in Germany. Because a much larger proportion of Germany is elderly, and because of the added problem of lower rates of fertility, German scholars and policymakers have been forced to face some hard questions: How can a society distribute scarce educational and medical resources justly among people at all stages of life? How can a society maintain its productivity when a growing proportion of its labor force population, because of its older age, is less fit for innovative labor and productivity?

In industrialized countries over the last century, we have witnessed truly astonishing increases in average life expectancy, from about forty-five years in 1900 to close to eighty years in 2000. Of special importance is new evidence that life expectancy is increasing in the older as well as younger age ranges.[4] Thirty years ago, an 80-year-old would live, on average, another four years; today, an 80-year-old can expect to live longer for double that time. If this upward trend in life expectancy continues more or less linearly, nearly half the people born today – especially women – could theoretically reach an age close to 100.

While certain genes play a key role in determining the life spans of human beings, genetic factors by themselves cannot explain this rapid increase in life expectancy in the twentieth century. Changes in the human genome occur gradually, over much longer periods of time than a century. Rather, better living conditions have been primarily responsible for the recent increase in human life expectancy. These technological, social, and cultural changes have permitted fuller utilization of the plasticity built into the human genome.

As more people live to older ages, they are demonstrating remarkable potential. Researchers at the Max Planck Institute for Human Development have found that people in their sixties and seventies still possess considerable intellectual and cognitive resources. Of course, intelligence is not a single, homogeneous capacity, and not all aspects of intelligence in the elderly show posi-

3 P. B. Baltes and M. M. Baltes, eds., *Successful Aging: Perspectives from the Behavioral Sciences* (New York: Cambridge University Press, 1990); Baltes and Mayer, eds., *The Berlin Aging Study*; M. W. Riley, R. L. Kahn, and A. Foner, *Age and Structural Lag: Society's Failure to Provide Meaningful Opportunities in Work, Family, and Leisure* (New York: J. Wiley, 1994); J. W. Rowe and R. L. Kahn, "Human Aging: Usual and Successful," *Science* 237 (4811) (July 10, 1987): 143 – 149.

4 Oeppen and Vaupel, "Broken Limits."

tive changes – but some do. On one hand, the mechanics of the mind, like computer hardware, dictate the sheer speed and accuracy with which it processes information. This capability – which is also key to learning – grows rapidly during childhood, but begins to wane in early adulthood. On the other hand, intelligence also includes a kind of 'crystallized' pragmatics, which, like computer software, reflects culture-specific knowledge such as language, professional skills, and practical reasoning about human affairs.[5]

Older adults can retain and even improve the crystallized pragmatics of the mind, provided they do not suffer from brain disorders. This is especially true for those bodies of pragmatic knowledge that individuals cultivate.[6] Thus, even as our minds lose their basic potential for peak performance in the basic mechanics of the mind, we can concentrate our efforts in areas where we have already achieved mastery and where 'new' learning is not the most critical component.

In the same vein, many older adults evince heightened emotional intelligence and interpersonal social cognition.[7] Indeed, wisdom, often considered the peak of human excellence in mind and character, is one of the elderly's most impressive potential characteristics.[8] It goes without saying that simply growing old is not enough to become wise. However, with life experience and the necessary personal qualities and patterns of thought, adults in their sixties and seventies often address problems requiring wisdom extremely well.

The positive aspects of experience are also evident in certain areas of professional expertise. Older composers and conductors, for instance, are often among the best in their fields. As long as an aged person remains professionally active, unaffected by specific age-associated illnesses such as stroke, and works in an area where the pragmatics rather than the mechanics reign, age often has little or no effect on specialized professional knowledge.

5 P. B. Baltes, U. M. Staudinger, and U. Lindenberger, "Lifespan Psychology: Theory and Application to Intellectual Functioning," *Annual Review of Psychology* 50 (1999): 471 – 507; R. B. Cattell, *Abilities: Their Structure, Growth, and Action* (Boston: Houghton Mifflin, 1971); F. I. M. Craik and T. A. Salthouse, eds., *The Handbook of Aging and Cognition*, 2nd ed. (Hillsdale, N.J.: Erlbaum, 2000); S.-C. Li et al., "Lifespan Transformations in the Couplings of Mental Abilities and Underlying Cognitive Processes," *Psychological Science* 15 (3) (2004): 155 – 163; P. A. Reuter-Lorenz, "New Visions of the Aging Mind and Brain," *Trends in Cognitive Sciences* 6 (9) (September 1, 2002): 394 – 400; K. W. Schaie, *Developmental Influences on Adult Intelligence: The Seattle Longitudinal Study* (New York: Oxford University Press, 2005); P. C. Stern and L. L. Carstensen, *The Aging Mind: Opportunities in Cognitive Research* (Washington, D.C.: National Academy Press, 2000).

6 Baltes and Baltes, eds., *Successful Aging*; R. T. Krampe and P. B. Baltes, "Intelligence as Adaptive Resource Development and Resource Allocation: A New Look Through the Lenses of SOC and Expertise," in R. J. Sternberg and E. L. Grigorenko, eds., *The Psychology of Abilities, Competencies, and Expertise* (New York: Cambridge University Press, 2003), 31 – 69.

7 L. G. Aspinwall and U. M. Staudinger, eds., *A Psychology of Human Strengths: Fundamental Questions and Future Directions for a Positive Psychology* (Washington, D.C.: American Psychological Association, 2003); B. T. Hess and F. Blanchard-Fields, eds., *Social Cognition and Aging* (London: Academic Press, 1999).

8 P. B. Baltes and U. M. Staudinger, "Wisdom: A Metaheuristic (Pragmatic) to Orchestrate Mind and Virtue Toward Excellence," *American Psychologist* 55 (1) (2000): 122 – 136; R. J. Sternberg and J. Jordan, eds., *A Handbook of Wisdom: Psychological Perspectives* (New York: Cambridge University Press, 2005).

The elderly are also amazingly well-equipped to adapt and stay positive despite the increasing restrictiveness of their activities and physical abilities. In fact, many older people claim they feel just as healthy as younger people, even though – objectively speaking – they are not. Often, their ability to establish new standards of comparison makes this feeling possible. For example, after people survive a heart attack, they are likely to compare themselves to others who have died.[9]

This 'adaptive self-plasticity' has positive effects on well-being. It also corresponds to the theory of selective optimization with compensation, developed at the Max Planck Institute for Human Development.[10] According to this theory, people at all ages of life engage in selection, optimization, and compensation. However, in older people, the mastery of these skills can become a fine art, as in the case of the pianist Arthur Rubinstein.

At 80, Rubinstein was asked how he managed to still give such excellent concerts. Over the course of several interviews, he offered three reasons. First, he played fewer pieces – an example of selection. Second, he practiced these pieces more often – an example of optimization. Finally, he played slow movements more slowly, to make it appear as though he were playing the piano faster in the fast movements than he was actually able to – an example of compensation. People who apply selection, optimization, and compensation as behavioral strategies feel better about themselves and go further in life, especially when the mechanics of mind and body start to decay, as they inevitably do in old age.

Gerontologists and policymakers are thrilled with these pieces of good news. However, not everyone – particularly not the very old people themselves – share this optimism. After all, why is it that most people don't want to be old? Why do people always want to be a bit younger than they actually are as they grow older? And why does the discrepancy between actual age and desired age increase dramatically as we reach our seventies, eighties, and nineties? Ninety-year-olds in Berlin, for instance, said they would have liked to stay, on average, between the ages 65 and 70.

Late in his life, the Italian philosopher Norberto Bobbio coined the phrase "happy gerontologists,"[11] suggesting that some aging researchers were so optimistic because they had not yet taken a proper look at the older-old. But since then, a number of gerontologists have in fact shifted their focus from the 'young-old' to the 'oldest-old.' In the Berlin Aging Study, for example, more than fifty medical experts, psychologists, sociologists, and economists repeatedly assessed over a period of ten years approximately five hundred older people ranging in age from 70 to 100.[12]

The results confirmed what Bobbio had suggested: Although some of the

9 J. Heckhausen and J. Krueger, "Developmental Expectations for the Self and Most Other People: Age Grading in Three Functions of Social Comparison," *Developmental Psychology* 29 (3) (May 1993): 539 – 548.

10 Baltes and Baltes, eds., *Successful Aging*; A. M. Freund and P. B. Baltes, "Life-Management Strategies of Selection, Optimization, and Compensation: Measurement by Self-Report and Construct Validity," *Journal of Personality and Social Psychology* 82 (4) (April 2002): 642 – 662.

11 N. Bobbio, *Old Age and Other Essays* (Cambridge: Polity Press, 2001).

12 Baltes and Mayer, eds., *The Berlin Aging Study*.

older-old remain very agile and emotionally well-off, their numbers begin to dwindle as they grow older. Physical and mental capacities increasingly diminish the older someone gets, clearly contradicting the belief that today's elderly are necessarily spared the negative aspects of aging.

Aside from deteriorating health, sensory systems, and bodily strength, one of the first things to decline markedly in the oldest ages is the capacity to learn. Experiments where subjects must learn a new memory enhancement technique demonstrate this: whereas the young-old tend to do very well, many people over 85 are unable to learn the technique unless it is simplified in major ways.[13] In the Fourth Age, even people considered mentally fit for their age have difficulty learning especially if the concepts are complex.

Moreover, people in the Fourth Age have a more fragile self-image than younger-old people. In the oldest-old, self-regulatory adaptability diminishes, largely because the gap between the desired and the real becomes too large in scope and magnitude. When the Fourth Age is reached, indicators of well-being such as life satisfaction, social integration, a positive attitude toward life, and aging satisfaction start to fall as a whole.

When looking at the whole of human functioning, the evidence for sizeable losses in the Fourth Age is impressive.[14] For instance, compared to people in the Third Age, almost five times as many people over 85 suffer from chronic impairments and exhibit low functional scores across a wide range of physical, cognitive, and social indicators.[15] These data confirm that life's journey tends to take a turn for the worse as one reaches, and even exceeds, the biological limits of human adaptability. The fact that the physical and mental capabilities of same-age old people have improved substantially in recent years can at best buffer the negative effects of old age, but not eliminate the basic trend. In other words, the magnitude of the aging effect is much larger than that of historical improvement.

The dramatic increase in dementia makes the losses of the Fourth Age particularly visible. According to the Berlin Aging Study and other studies, less than 5 percent of 70-year-olds suffer from some form of dementia, including Alzheimer's.[16] But this percentage increases to 10 to 15 percent of 80-year-olds and to about 50 percent of 90- to 100-year-olds. Currently, no scientific evidence seems to indicate a 'historical cohort effect' for Alzheimer's-induced dementia – that is, a shift toward later ages.

Dementia, especially Alzheimer's disease, leads to a gradual deterioration in many basic human characteristics, including intentionality, independence, identity, and social integration. These characteristics play a key role in defining human dignity and allowing individuals

13 R. Kliegl, J. Smith, and P. B. Baltes, "On the Locus and Process of Magnification of Age Differences During Mnemonic Training," *Developmental Psychology* 26 (6) (1990): 894 – 904; T. Singer, U. Lindenberger, and P. B. Baltes, "Plasticity of Memory for New Learning in Very Old Age: A Story of Major Loss?" *Psychology and Aging* 18 (2) (June 2003): 306 – 317.

14 J. Smith et al., "Two-Wave Longitudinal Findings from the Berlin Aging Study: Introduction to a Collection of Articles," *Journal of Gerontology: Psychological Sciences* 57 (6) (November 2002): 471 – 473.

15 Baltes and Smith, "New Frontiers."

16 H. Helmchen et al., "Psychiatric Illnesses in Old Age," in Baltes and Mayer, eds., *The Berlin Aging Study,* 167 – 196.

to exercise autonomously their 'human rights.'

We are now faced with a new challenge: to conserve human dignity in the later years of life. In the Fourth Age, gerontology's leitmotif, "Add more life to years, not more years to life," seems to have met a new barrier. This barrier has its roots in our evolutionary past.[17]

Up until the present time, biological evolution operated by selecting and optimizing the reproductive abilities of humans during early adulthood. As a consequence of this primary focus on reproductive fitness, there was little opportunity for improving the genome for the older ages of modern times. Not surprisingly, therefore, the self-preserving regulatory mechanisms of the human genome deteriorate with age. These biogenetic losses of human aging are less obvious in the Third Age because culture- and technology-based improvements have been successful in compensating for this evolution-based deficit.

These genome-based deficits, more conspicuous and prevalent in the Fourth Age, limit the countervailing effectiveness of cultural factors, including education and medicine. Older people, in general, need much more practice than young people to achieve similar progress in a cognitive task. Moreover, the number of illnesses increases. Multimorbidity and general losses across the board become hallmarks of the oldest ages.[18]

Is this but a transitory state? Scientists should be careful with predictions. However, without a doubt, one of the core questions gerontology faces today is to what extent further scientific developments can offer new insights and ways to ameliorate the biological deficits of the Fourth Age. Theoretically, of course, the advent of new forms of biotechnology gives us hope of genetically altering the biogenetically 'incomplete' architecture of the life course to make it more amenable to cultural and psychological influences. However, based on present-day evidence, such speculations also put us on shaky ground – not only because of the unpredictable effects of genetic engineering, but also because of the ethical-religious debate on human nature.

Because of the complexity of the human genome, any attempt to intervene in this system risks producing undesirable side effects. Moreover, a multitude of biogenetic factors, including their interaction with numerous behavioral and environmental parameters, influences the aging process and many of its associated diseases. An increasing number of random effects are also a part of the story. This makes gene therapy more complicated for these diseases than for 'simpler' monogenetic diseases. Though gene therapy currently holds a lot of promise for treating monogenetic diseases, these kinds of diseases are demographically less significant, affecting only a small fraction of the aging population.

Many biomedical scientists agree that knowing the genetic factors involved in the aging process does not automatically mean that a quick and standardized means of 'artificially' perfecting the biogenetic architecture of the aging process is available. These factors are simply too complex and often differ from individual to individual. Nonetheless, it seems fair to argue that in the long term only biomedicine has a chance of truly trans-

17 Baltes, "On the Incomplete Architecture of Human Ontogeny"; C. E. Finch, *Longevity, Senescence, and the Genome* (Chicago: University of Chicago Press, 1990).

18 Baltes and Mayer, eds., *The Berlin Aging Study*; Baltes and Smith, "New Frontiers."

forming old age into a Belle Époque. Because of the reduced scope of biological potential, improved environmental conditions and age-friendly behavioral strategies alone will not suffice.

For the younger-old, those in the Third Age, the prospects seem bright. With new approaches involving various methods of biocultural co-construction,[19] with systematic efforts at optimizing the strengths of older individuals, with new aging-friendly institutional structures, with innovative efforts at developing conceptions of productivity that extend beyond the narrow sense of economic productivity, modern societies have the potential to create a better future for the younger of the older ages and thereby empower individuals to become 'successful agers.' And because of large individual variability in the genome and culture, it is also likely that we will continue to witness outstanding individual success stories in the oldest ages as well.

For most of the older-old, however, the prospects are not so bright. From my point of view, "Hope with a mourning band" may be the motto best suited to this situation. How to combine longevity with a high quality of life and human dignity in the oldest ages is the new frontier. As demographers celebrate each month gained in the lives of the oldest-old, researchers focused on improving quality of life worry about the associated increase in the gap between longevity and vitality.

That the situation is not hopeless is illustrated in the "compression of morbidity model" articulated by Fries.[20] It proceeds from the assumption that there is a 'current' biological limit to life span, around eighty-five to ninety years. With that assumption, science and society could take increasing the quality of life rather than the quantity of life more seriously. One way to do so would be by compressing major events of illness into the few years preceding natural 'biological' death. Such a strategy allows people to maintain their human dignity longer without necessarily extending life span.

In my view of the evidence, Fries' vision still seems a realistic alternative. Though only when we move to an average life expectancy of eighty-five to ninety years will we know if it is truly possible. Using this model as a framework along with the newest evidence on the dysfunctional states of the oldest-old, I suggest that we tone down our quest to extend longevity in favor of raising the quality of life within the present frame of life expectancy. And even if there is no maximum biological limit to life, such a limit can still become

19 P. B. Baltes, P. A. Reuter-Lorenz, and F. Rösler, eds., *Lifespan Development and the Brain: The Perspective of Biocultural Co-Constructivism* (New York: Cambridge University Press, forthcoming); E. Jablonka and M. J. Lamb, *Evolution in Four Dimensions: Genetic, Epigenetic, Behavioral, and Symbolic Variation in the History of Life* (Cambridge, Mass.: MIT Press, 2005); S.-C. Li, "Biocultural Orchestration of Developmental Plasticity Across Levels: The Interplay of Biology and Culture in Shaping the Mind and Behavior Across the Lifespan," *Psychological Bulletin* 129 (2003): 171–194; Riley, Kahn, and Foner, *Age and Structural Lag*.

20 J. F. Fries, "Aging, Natural Death, and the Compression of Morbidity," *New England Journal of Medicine* 303 (3) (July 7, 1980): 130–135; J. F. Fries, "The Compression of Morbidity: Near or Far?" *Milbank Memorial Fund Quarterly* 67 (2) (1989): 208–232; J. F. Fries, "Measuring and Monitoring Success in Compressing Morbidity," *Annals of Internal Medicine* 139 (5 [Part 2]) (September 2, 2003): 455–459; J.-M. Robine and J.-P. Michel, "Looking Forward to a General Theory on Population Aging," *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* 59 (2004): M590–M597.

a part of our decision making as self-directed individuals.

Besides begging the question of more quantity versus better quality of life in the oldest ages, this new frontier will also intensify debate over the meaning of life, forms of death and dying, and the impact that investing scarce resources in increasing life span will have on other sectors of society. Though these are beyond the scope of this essay, it is important to discuss at least the last issue briefly in order to place human aging in a larger societal and global context. Too often aging researchers and policy analysts overlook the implications of individual and population aging for the distribution of resources.

For instance, to what degree does the growing investment into extending and supporting the lives of the elderly limit the resources available for improving the lives of children and youth, or for reducing the gap between the rich and the poor? Also, are we hampering global progress by allocating an increasingly larger share of our resources to keeping the oldest in our own industrialized country alive rather than helping developing countries? For a gerontologist, these are questions that do not win the approval of most of his peers. However, I believe these are the kinds of questions that will increasingly shape scientific and public discourse about individual and population aging in the twenty-first century.

Countries such as Germany – where increasing longevity and low fertility rates will result in a disproportionately older population within the next fifty years – must also consider the effect of population aging on their national productivity and global competitiveness. Such societies are likely to experience a reduction in their potential for innovation, as the average age of workers grows from about 45 to 55. Experimental aging research on cognitive plasticity clearly demonstrates the considerable negative and rather immutable effects of age on the speed and accuracy of information processing and the potential to learn new skills and knowledge, especially at high levels of expertise. Also, the older we get, the more the body calls on cognitive resources – for instance, when keeping one's balance or thinking while walking on uneven terrain.[21] This 'mortgaging of the mind' by the body leaves less of our mind available for mental activities of the usual kind.

We humans are inherently curious and committed to living long and well, if not for eternity. Yet, even as *homo faber*[22] allows us to grow older and older, we must consider the scientific evidence that indicates a developmental limit to human life in the oldest ages and accept that "less may be more."

Hesiod is said to have expressed a similar thought: "If one chooses the right half, half may be more than the whole."

21 U. Lindenberger, M. Marsiske, and P. B. Baltes, "Memorizing While Walking: Increase in Dual-Task Costs from Young Adulthood to Old Age," *Psychology and Aging* 15 (3) (September 2000): 417 – 436.

22 J. Mittelstraß, "Science and Culture," *European Review* 4 (1996): 293 – 300.

Linda Partridge

Of worms, mice & men: altering rates of aging

The word 'aging' has very different implications for wine and women. Matters can improve or deteriorate over time, and the everyday usage of the term can cover either situation.

In the natural world things tend to get worse over the course of adulthood. In mammals and birds, for instance, many long-term field studies have revealed that both likelihood of survival and production of offspring decline later in life. There are exceptions, to be sure. Especially where animals start to procreate before they are fully grown, as is the case in many fish, chances of survival and fecundity can improve over at least part of adulthood.

But in the organism in which most people are really interested, humans, there is a clear deterioration in function over time, after puberty. This is the sense in which I will use the word 'aging' in this essay, as the intrinsic decay in function that sets the ultimate limit to life span.

At first sight, aging does not require any special explanation. Machines wear out and fail, so why not living things too? Aging does indeed involve accumulation of damage. The molecules that make up bodies acquire lesions, as do whole cells and tissues. Decrements in the ability to run or to solve problems quickly, for example, reflect this accumulation of damage.

If this were the whole story, then we would expect similar organisms to age at roughly similar rates, but they do not. For instance, bats and monkeys are peculiarly long-lived mammals for their body size; birds, in general, also live much longer than comparably sized mammals. These differences persist in captivity and are thus properties of the species rather than of the individual lifestyle. The rate at which organisms age can, therefore, evolve. By changing the rate at which unrepaired damage accumulates with time, certain biological processes are able to influence the rate of intrinsic decline. It is the activity of these processes that can change during evolution.

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Considering its genetic basis and evolutionary change, aging has some very odd characteristics. It is unconditionally deleterious; yet, as far as we know, no genes have evolved to directly cause accumulation of damage, lowered fertility, and death. Earlier ideas that the aging of individuals could be beneficial to the species, by removing the old to make way for the young, are now very largely discredited. If aging itself is a disadvantage, then it cannot evolve by natural selection in its favor and must instead evolve as a side effect of something else.

Several of the intellectual giants of theoretical populations genetics and a large amount of empirical research in the laboratory and the field have produced quite a clear picture of the two routes through which aging can evolve.

First, it can appear as a side effect of mutation pressure, in a process known as mutation accumulation. Sporadic alterations to the genetic material transmitted from one generation to the next can often cause genetic diseases. Some of these diseases, however, become manifest only as the bearer of the mutation gets older, as in the case of Huntington's disease. The later in life that the mutation's bad effects appear, the greater the mutation's chance of reproduction because external hazards such as infections, predators, and accidents will cause attrition of the bearers of the mutation and not all of them will survive long enough to express its effects. Natural selection therefore acts more weakly to remove these later-acting mutations from the population. The balance between the occurrence of mutations and their removal from the population by natural selection is hence shifted; later-acting mutations can achieve a higher frequency in the population than can equivalent mutations that also affect the young. These late-acting, deleterious mutations can therefore cause aging.

In addition, aging can evolve as a side effect of earlier success. If a mutation benefits the young, perhaps by making them more fecund, natural selection can act in its favor, even at the price of a higher subsequent rate of aging. Again, death by natural hazards means that more of the mutation's bearers will survive to gain the early benefits than to show the elevated rate of aging.

In either case, whether as a side effect of mutation pressure or of earlier reproductive benefits, the intrinsic rate of aging seems to evolve according to the level of external hazard. In an environment where external risks minimize the likelihood of survival beyond age 10, natural selection will not eliminate a mutation that causes problems for 11-year-olds from the population. However, if the external hazards ameliorate so that survival to age 14 becomes routine, then natural selection against the mutation will become stronger.

We therefore expect to find slow-aging creatures in less hazardous environments. This is roughly what we observe in nature. Species that are well protected such as tortoises and turtles, species that can fly such as birds and bats, and mammals that live in trees instead of on the ground are all capable of longer lives.

Aging involves change in the organism with time, which has often led people to think that it is a process like development or growth. Our evolutionary understanding of aging says otherwise. Aging evolves in response to extrinsic hazard, with more rapid aging arising in more dangerous environments, as a side effect of accumulating more mutations that either affect an organism later in life or cause more intense early reproduction. Aging is therefore not a programmed process like development or

growth, with a well-orchestrated hierarchy of genetic control ensuring that the right things happen in the right place and at the right time to make a well-formed organism. The genes that affect aging did not evolve to control it; therefore it is a much more haphazard and variable kind of process. In humans, the sorts of damage that accumulate are very distinct in different parts of the body, and the rate at which these forms of damage accumulate can vary greatly between individuals. People develop different problems from each other as they age, and they die of diverse causes. Because aging is such a complex process, it probably involves a large variety of parallel processes. Therefore, many rather than few genes likely influence aging.

Aging's complexity has long colored scientific and medical attitudes toward it. Scientists have tended to assume that aging is too difficult and intractable a trait for experimental analysis. Although aging is the major risk factor for multiple diseases, including major killers such as cancer, circulatory disease, and neurodegeneration, the medical community treats these diseases separately rather than as different manifestations of a single, underlying aging process. Aging is seen as inevitable, too complicated to do anything about, and best treated by piecemeal intervention into its undesirable manifestations.

Tempering pessimism about the prospects for intervening in the aging process are the dramatic improvements in health during aging that have occurred in industrialized human societies worldwide. The rate of aging certainly has a genetic and evolutionary basis, but life span can vary depending upon the environment encountered. Beginning in the mid-nineteenth century, survival rates have risen steadily, probably mainly because of various public-health measures such as improved sanitation and hygiene and a great reduction in the impact of infectious diseases. These increases in survival have affected all age groups. Thus not only average life expectancy but also the longevity of the oldest segment of the population has increased. Furthermore, there is no sign that the upward swings in survival rates are slowing down for any age group, which suggests that there is no impending limit to maximum human life span. Such a limit may exist, but at the moment we cannot see what it is.

People of all ages are certainly healthier than they used to be, although the approaching wave of obesity in the young, if unchecked, may counter this trend. But is the improvement in health the result of the slowing of the aging process itself? The figures suggest not. If we define a population's death rate as deaths per thousand in a year, then this rate increases with chronological age – aging. Were aging checked, we would see a decline in this latter rate, say over ten-year blocks of time, but we do not. Rather, survival at all ages has increased, with no evidence of a slow down in the rate of aging itself. Human death rates tend to be somewhat elevated at birth, fall to a minimum around the age of puberty, and start to rise steadily thereafter. This pattern has remained unchanged over the one and a half centuries during which life expectancy has lengthened, and the rate of increase in death rates after puberty has not declined. Health during aging is better, but the underlying aging process seems to have eluded modification. There is good news and bad.

As a result of longer life expectancy, industrialized societies face many challenges, particularly for health-care systems. Although older people are now

healthier, health-care demand is steadily growing as more people reach the older ages at which aging-related health problems occur. This provides strong motivation for the biomedical community to undertake scientific research that will ameliorate the impact of aging-related disease and disability. In recent years, the discovery that mutations in single genes can greatly lengthen healthy life span in experimental animals has galvanized research into the mechanisms of aging. This has come as a surprise to many, and it has opened up new vistas in our understanding of how healthy life span is controlled.

The process of discovering the single gene mutations that extend life span started with a tiny roundworm called *Caenorhabditis elegans*. It was in the context of studies of these worms' development that the first mutation came to light. Before becoming reproductive adults, the worms can take two different developmental routes. If conditions are good, they grow straight through to adulthood and start reproducing. If, on the other hand, the worms are crowded or short of food, they arrest their development and form a *dauer* larva. *Dauer* larvae stop feeding, store fat, resist various environmental stresses, and are very long-lived. Because of these characteristics, they can sit out hard times and resume normal development when conditions ease. Initially, work on *dauer* development produced mutations in single genes that caused the developing worms to form *dauers* even under good conditions. Different, weaker mutations in these same genes, however, made the adult worm long-lived. And one of these mutations turned out to be in a gene that encoded a part of a signaling pathway that was clearly similar in its evolution to the insulin and insulin-like growth factor signaling pathways of mammals.

These findings were fascinating. They revealed that adult life span could be under simple genetic control, and that a signaling pathway more familiar for its effects on the regulation of blood sugar and growth in mammals could also affect life span in a lowly invertebrate. However, the full implications of these findings took some time to become apparent. For starters, the long life of the mutant adult worms was probably simply a reexpression in the adult of the traits that make for long life in *dauer* larvae. If this were true, then the findings would unlikely to be of any relevance to mammals, which do not have *dauer* larvae or their equivalent. Furthermore, it was quite unclear how this insulin-like pathway could have any bearing on life span.

There matters rested for some years until an insulin-like signaling pathway came to light in another invertebrate inhabitant of research laboratories, the fruit fly *Drosophila*. Again, the initial discovery occurred during studies of development rather than of aging. This insulin-like signaling pathway controls growth in the pre-adult period. If the activity of the pathway is elevated, growth rate and the size of the adult are increased. Conversely, lesions in components of the pathway result in dwarf adult flies. Because of this work, scientists were able to make mutations in several genes that encode components of the pathway and measure the effect on adult life span. Some of these mutations did indeed increase life span. The results for *Drosophila* were also interesting, for instance, in showing that lesions in the pathway seemed to increase life span much more in females than in males and to impair the fecundity of females.

But the findings from the fly had broader implications. The worm and the fly are very different kinds of organisms; they are only distantly related to each other and have diverged over millions of years of evolutionary time. If the genes of the insulin-like pathway could affect life span in both, then it was distinctly possible that the pathway could control life span in mammals as well.

Some straws in the wind were already suggesting that insulin-like growth factor signaling could play a role in the control of mammalian life span. It was known, for instance, that several mutations that cause lesions in the development of the pituitary gland and in the signaling by the growth hormone produce long-lived dwarf adult mice. Then, in 2003, two key papers implicated both the insulin-like growth factor signaling pathway and the insulin signaling pathway in the control of life span in the mouse. In one, scientists manipulated the gene that encodes a receptor on the surface of cells that responds to signals from the insulin-like growth factor. They reduced the number of copies of this gene from two copies to one throughout the mouse. The resulting female, but not male, mice lived longer. In the other, scientists removed the receptor on cell surfaces that reacts to insulin from the mice's fat cells, their white adipose tissue. The engineered mice were leaner and longer-lived.

These results had huge implications. The insulin or insulin-like growth factor signaling pathway, which is present in all multicellular animals, appears to have conserved one of its functions, the control of life span, over the very large evolutionary distances between the invertebrate worm, the fly, and the mouse. The pathway is therefore a strong candidate for the control of human life span. Furthermore, the evolutionary conservation of the pathway means that we can use powerful analytical methods with fewer ethical implications on the invertebrates, with all their advantages of relatively short life spans (about three weeks in the worm and three months in the fly, as opposed to three years in the mouse), simplicity, and low maintenance costs to understand how this pathway might control mammalian life span. This has long been the Holy Grail of aging research. But it opens up a major paradox.

How can a mutation in a single gene produce such a large increase in life span if many genes influence the multiple, parallel processes that control the rate of aging?

A clue may come from the effects of diet. Dietary restriction is an environmental intervention that, along with mutations in single genes, has long been known to extend life span in laboratory rodents. Its effects have been conserved over evolution; first discovered to extend life span in laboratory rats in 1935, dietary restriction has since been shown to have a similar effect in organisms as diverse as yeast, worms, flies, and mice as well as other less intensively studied species. During dietary restriction the amount of food an animal consumes is reduced dramatically. In rats and mice a reduction to about 50 percent of voluntary levels can produce a substantial increase in life span. Dietary restriction does not merely reverse the effects of a sedentary existence and overeating. In worms, flies, and mice dietary restriction also reduces fecundity; female mice subjected to strong dietary restriction become completely infertile.

This correlation between the longer life span that results from dietary restriction and lowered fertility has led some to suggest that reduced fertility is an

evolved, adaptive mechanism for sitting out hard times. Reproduction is expensive in nutrients and can compromise the survival of the parents. If food is scarce, parents cannot produce many offspring anyway and their offspring's likelihood of survival is low. Under these circumstances, it may pay for a parent to lower its reproductive rate and thereby increase its own chance of survival until the food supply improves.

Numerous species show greater longevity and reduced fecundity in response to lowered food intake. However, we do not know precisely what it is about lower intake that influences longevity, for instance, whether the overall intake of energy or particular dietary components are critical. Nor do we fully understand how the animal senses the change in nutrition and how it uses this information to change its internal state and extend its life span. For these reasons, it is not yet certain if different animals achieve these responses to dietary restriction in the same way, as we would expect if the processes at work have been conserved over large evolutionary distances. Thus, we cannot exclude the alternative possibility – that these responses have evolved independently in different lineages. Still, there is intense interest in the mechanisms by which dietary restriction lengthens life span in laboratory organisms.

Dietary restriction also keeps laboratory rats and mice healthy for longer, delaying the impact of aging-related disability and disease. Dietary restriction enables animals to remain active and able to reproduce for longer, maintains better immune function, slows down the changes in the musculoskeletal system that alter body shape as the animal ages, preserves the structure and function of the nervous and endocrine systems, and reduces the frequency of cancers and other diseases. In the fly, too, several lines of evidence, including the ability to reproduce for longer, suggest that dietary restriction helps animals remain youthful. For these reasons, it seems that dietary restriction really might slow down the aging process itself. We might, then, have two interventions – conserved by evolution – that can increase life span by slowing down the aging process. But if aging occurs through multiple, parallel pathways of accumulation of damage, how could dietary restriction slow down all of them? As for the effects of the insulin or insulin-like growth factor pathway, the evolutionary and mechanistic findings seem to be at variance with each other.

We are making steady progress toward understanding how lesions in the insulin or insulin-like growth factor signaling pathway and dietary restriction extend life span. We have already made some important findings. One is that these interventions do not slow down the rate of living; the metabolic rate of these long-lived animals is normal. Nor does the reduction in reproductive rate we often see in conjunction with the increase in life span appear to play any simple direct role. We have also sequenced all the genomes of the worm, fly, and mouse, opening up powerful avenues to understanding how genes control vital processes. While we have a long way to go to this goal, it is becoming clear that when life span is extended, the genes that control processes such as detoxification and turnover of damaged molecules, resistance to stresses, immunity and inflammation, and the metabolic pathways by which nutrients are stored and used can all show changes in expression. Some of these may be irrelevant to the extension of life span; only

experimental work will eventually reveal which are the critical changes, where in the organism they occur, and how exactly they lead to increased survival.

The discovery of interventions – be they diet or single gene mutations – that have the potential to improve health during aging is a massive step forward. The current intense interest in their mode of action will undoubtedly continue amidst high hopes that the results will translate into medical practice. But do these interventions really slow aging, or could they be prolonging life in some other way?

Since aging is the accumulation of unrepaired, irreversible damage, we would expect an organism that has aged more slowly to have less cumulative damage. Flies that have lived at different temperatures demonstrate this phenomenon. Flies – reared to adulthood at a single intermediate temperature and then kept at different temperatures as adults – live longer the lower the temperature at which they are kept. Flies are much too small to regulate their own body temperature and immediately adopt the temperature of the environment in which they live. Lower temperatures slow down the rates of all chemical processes, including, presumably, those leading to death.

Low temperatures extend the life span of flies by slowing down the rate of aging. If flies that have been in the cold for varying lengths of time are moved to higher temperatures, their death rates are lower than those of flies that have been previously kept at the higher temperatures. Also, the degree of protection that they enjoy is greater the longer that they are left in the cold before being transferred to the warmer environment. Similarly, the death rates of flies switched to a cooler environment after living in high temperatures are permanently elevated above those of flies with a history of life in a cooler environment; and the longer that they have stayed in the warmer conditions the higher their death rates. These findings demonstrate that, in flies, warmer temperatures induce more irreversible damage that leads to death; in other words, they elevate the rate of aging.

This kind of experimental approach, where animals are switched between regimes partway through adult life, has revealed some surprising findings about dietary restriction in flies. When flies that have been on a normal diet are subjected to dietary restriction, they adopt within forty-eight hours the lower death rates of flies that have been on restricted diets. Similarly, when flies that have been on restricted diets are fully fed for the first time, they show within forty-eight hours the elevated death rates characteristic of flies that have been on normal diets throughout adulthood. It does not seem to matter how late in life they switch; after a short lag their life spans show no memory of nutritional history, and the death rates of flies that have switched diets converge with those of the flies that have been kept permanently in that nutritional regime. At least in flies, then, dietary restriction does not slow down aging. Rather, dietary restriction somehow acts acutely to make the flies less likely to die from the damage that they have accumulated.

We do not know if dietary restriction has similarly acute effects on death rate in mammals because we have not carried out the appropriate experiments yet. If the effects were acute, then we would expect the switch in death rates after a change in nutritional regimes to take longer in mammals than in flies, perhaps on the order of some weeks. At first sight, the idea that dietary restric-

tion acts acutely to reduce death rates seems incompatible with the finding that it also delays the impact of aging-related disability and disease. However, these two findings can be reconciled. Dietary restriction could act acutely to lower the likelihood that aging-related damage will lead to the appearance of a lethal, aging-related pathology. The lag period during which death rates switch over to those characteristic of the new nutritional regime would then represent the period during which individuals that have acquired a lethal pathology are lost from the high-risk population, with a switch to dietary restriction, or gained by it, in a switch to full feeding.

We have not yet determined, even in flies, if the reduced activity of the insulin or insulin-like growth factor signaling pathway extends life span by slowing the rate of aging or through an acute effect. Answering that question will require an experiment switching the activity of the pathway partway through adulthood, which is now coming within the realms of technical feasibility. These are important questions for the future. The acute effects of dietary restriction in the fly have unexpectedly revealed the existence of a type of intrinsic risk factor whose mechanistic basis requires elucidation. If similar acutely acting interventions exist for humans, we could eliminate the adverse long-term consequences of some risky habits.

So far, we have not proven that dietary restriction or single gene mutations extend life span by reducing the rate of accumulation of aging-related damage in any organism. The information that we have about their effects is compatible with the alternative possibility that they increase life span by making animals less likely to die of damage already accumulated, leaving the rate of aging itself unaffected. Perhaps the pervasive effects of altered temperature for ectothermic animals and the slow, cumulative effects of evolution really are the only means by which the rate of aging can be altered.

The fact that we may not yet be able to alter its rate in humans is disappointing. But the discovery that there are interventions that can act acutely to delay the impact of multiple forms of aging and pathology is in some ways even better. For those with unhealthy habits, it may mean it is never too late to adopt a lower-risk lifestyle.

Hillard Kaplan

The life course of a skill-intensive foraging species

The study of model organisms, such as flies and worms, is becoming an increasingly important tool in aging research. They are ideal for experiments that manipulate rates of aging because they are small, do not live long, reproduce rapidly, and have relatively uncomplicated genomes. To the extent that many of the basic processes governing aging are similar across a wide range of organisms, we can learn much about human aging through the study of simpler creatures.

But in other ways, human beings themselves are also model organisms. We live and have lived in a diverse array of environments; we can know our ages without observing our own births; we leave historical, archaeological, and paleontological records; and we can report on the phenomena that are relevant to the aging process. We certainly know much more about aging in humans than we do about aging in any other mammal, with the possible exception of laboratory rats and mice.

In fact, the study of human aging and its evolution is especially promising today. It is revealing clues not only to our evolutionary history, but also to the forces shaping life span evolution among living creatures in general.

More specifically, research with the few remaining people that still practice a traditional hunting and gathering lifestyle is providing critical information about the life histories of our ancestors and the selection pressures that acted on them. Humans lived as hunter-gatherers for the vast majority of their evolutionary history (the genus *Homo* has existed for about 2 million years). Modern hunter-gatherers, who are affected by global socioeconomic forces, are not living replicas of our stone age past. Yet, in spite of their variable historical, ecological, and political conditions, foraging peoples exhibit remarkable similarities, especially in the realms of development and aging, suggesting that in our species natural selection has produced a

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characteristic life history. This life history is quite distinct from that of our closest living primate relatives.

The life course of a skill-intensive foraging species

When we compare humans to other living beings, our brains and our attendant mental abilities stand out. The human brain, which weighs about three pounds, is about three times as large as that of a chimpanzee, our closest living relative and arguably one of the most intelligent nonhuman species. Any attempt to account for our unique evolutionary path will also have to explain why human brains became so large and why we became such good learners.

In addition to our large brain size, humans are also distinctive because of our very long lives. As children, we learn that a human year is equal to seven 'doggy' years, a recognition that our pets live shorter lives and age faster than we do. After their first decade of life, our dogs show signs of aging – hearing and vision loss, arthritis, incontinence, and increased likelihood of cancerous tumors.

Comparing the life span of people living in remote societies without Western medicine and that of wild chimpanzees and gorillas is particularly revealing. Among chimpanzees, the risk of dying is high during infancy, decreasing rapidly after infancy to its lowest point (about 3 percent per year) at about age 13, the age of first reproduction for females – and increasing sharply thereafter. In contrast, mortality among people still living as hunter-gatherers drops to a much lower point (about 0.5 percent per year) and remains low without any increase between about 15 and 40 years of age. Mortality rates then rise slowly, with a very rapid jump in the sixties and seventies.

As a result, about 60 percent of hunter-gatherer children survive to adulthood, compared to 35 percent of chimpanzees. Chimpanzees also have a much shorter adult life span than humans. At the age when they first reproduce, chimpanzees live, on average, an additional fifteen years, compared to thirty-eight more years among human foragers. Importantly, women spend more than a third of their adult life in a postreproductive phase, whereas very few female chimpanzees even reach a postreproductive phase. Overall, fewer than 10 percent of chimpanzees last to age 40, but more than 15 percent of hunter-gatherers make it to age 70. In this sense, a human year is equal to about two 'chimpanzee years.'

Chimpanzees also age faster, displaying signs of aging in their thirties that humans show in their sixties. In contrast, elderly people in foraging societies are surprisingly vigorous, as I learned during my first few days living with Ache hunter-gatherers in Paraguay. As a young single male, I shared a fire with 'Grandfather Jaguar,' who was about 70 at the time. While he had outlived his prime hunting days, he still chased small game and dug six-foot holes with a stick to capture armadillos, who were themselves digging as fast as possible to escape him. He was also a font of wisdom, advising younger hunters and introducing me to culinary delicacies like large rodent feces cooked with fat and hearts of palm.

Delayed aging and long adult life spans appear to be evolved characteristics of our species. Adult mortality risks are remarkably uniform across human societies, even in those without access to Western medicine. Although the ancestors of the aboriginal peoples of South America, Africa, and New Guinea went their separate ways more than ten thousand years ago, the risk of dying at each age is very similar from society to socie-

ty, and not that different from historical Europe's prior to modern medicine.

How do we explain our large brains and long lives? For more than a half-century, we have known that larger brains are associated with longer life spans among mammals in general. But for humans in particular, we must examine our primate ancestors to understand the evolution of our larger brains and longer lives. As the primate order evolved and different species radiated across the world's warmer areas, there was a series of four grade shifts that increased 'encephalization' – brain size relative to body size – and slowed life histories.

The first grade shift began about 60 million years ago with the evolution of prosimians, who probably lived longer because living in trees afforded them more safety. The second grade shift came about 35 million years ago with the evolution of the monkey lineage. It involved a huge increase in both brain size and life span. The development of the ape lineage – represented today by chimpanzees, gorillas, and orangutans – was the third major grade shift in primate brain size and longevity. Apes can live almost twice as long as most monkeys and have much bigger brains, even after adjusting for their larger body size. The fourth grade shift occurred with the divergence of the hominid line, particularly the evolution of genus *Homo* and its extreme manifestation in modern humans.

This coevolution of brains and life spans makes sense when one considers that natural selection depends on both costs and benefits. Brains are very costly: in the extreme case, humans expend about 65 percent of all resting energy in supporting the maintenance and growth of the brain during the first year of life. Brains evolving by natural selection must provide benefits that pay for their costs. If the average cost of the brain outweighed the benefits it provided, individuals with smaller brains would leave more descendants than the larger brained members of their population, and the average brain size of the population would shrink.

Since learning transforms present experiences into better future performance, we can see brain development – especially of the cerebral cortex, which expanded disproportionately during primate evolution – as an investment in the future. During primate evolution, brain size appeared to increase as the learning required by the feeding niche grew more intense. The monkey radiation, for example, involved a shift from the prosimians' smell- and hearing-based insect eating to a reorganized sensory system with binocular, color vision able to find many different plant foods, all captured by dexterous hands and manipulated through hand-eye coordination. Because monitoring and exploiting the fruits and leaves of different trees demanded more learning, brain size, accordingly, had to evolve.

During the third shift, apes adopted a diet that emphasized ripe fruits, which requires even more environmental monitoring and more complex, extractive foraging techniques. These higher prerequisites for learning explain why apes take longer than monkeys to become competent foragers. For example, even though chimpanzees can provide most of their own caloric needs by age 5, those under age 7 are unable to utilize and make tools for termite fishing, and therefore still rely on their mothers for protein, even after weaning. Orangutans also depend on their mothers for about seven years. Today, as conservation-minded scientists attempt to reintroduce

captive-born orangutans into the forest, they are discovering how much learning must take place before these creatures are able to survive on their own.

During their evolutionary history, humans took the learning-intensive diet to an extreme. Mounting evidence from various sources, including digestive anatomy, digestive biochemistry, bone isotope ratios, archaeology, and observations of hunter-gatherers, shows that humans are specialized in the consumption of calorie-dense, low-fiber foods that are rich in protein and fat. Although there is considerable variation across societies depending on their ecology, modern foragers all differ considerably in diet from chimpanzees. The majority of forager diets is meat, accounting for about 60 percent of their calories. In contrast, chimpanzees obtain only about 2 percent of their food energy from hunted foods.

Resources such as insects, roots, nuts, seeds, and difficult-to-extract plant parts such as palm fiber or growing shoots comprise the next most important food category for foragers. These resources tend to be embedded in a protective context, such as underground or in hard shells. Such 'extracted' foods make up about 30 percent of the forager diet as opposed to 3 percent of the chimpanzee diet.

In contrast to hunted and extracted resources, which are difficult to acquire, collected foods such as fruits, leaves, flowers, and other easily accessible plant parts form, on average, 95 percent of the chimpanzee diet but only 8 percent of the human forager diet. The data suggest that humans specialize in rarer but more nutrient-dense resource packages – such as meat, roots, or nuts – whereas chimpanzees specialize in easily attainable but less nutritionally dense plant parts.

The life course of a skill-intensive foraging species

Comparative data on digestive anatomy confirm that these contemporary differences reflect long-term adaptations. Gorillas and chimpanzees have a very long large intestine and *caecum*, which promote the bacterial fermentation necessary for the conversion of plant cellulose in leaves and other structural plant parts into a source of dietary protein. Humans, on the other hand, have very short large intestines, incapable of processing cellulose in large quantities, and very long small intestines, geared toward digesting fat. Although the data are still relatively scarce, this dietary shift apparently began with the origin of the genus *Homo* about 2 million years ago, about the time the hominid brain expanded beyond that of apes.

Because of the learning-intensive nature of their feeding niche, humans take even longer than apes to become competent foragers. Whereas chimpanzees can meet most of their caloric needs by age 5, humans produce fewer calories than they consume for close to twenty years! In fact, the total calories that human parents must provide for their offspring increases from birth to about age 14, as children grow but remain unproductive. A Machiguenga man in Amazonian Peru once complained that his two older boys, 12 and 14, ate faster than he could bring food in, requiring him to hunt more than ever before.

Yet however burdensome childrearing may sometimes seem to parents, this long period of dependence pays off in the long run. Human adults are much more productive than chimpanzees. Net production (the surplus after one's own food consumption is taken into account) climaxes at about 1,750 calories per day for human adults, compared to 250 for adult female chimpanzees. However, humans generally do not reach that level of productivity until about age 45. Fe-

male Hiwi foragers from Venezuela, for example, do not reach their peak rate of root acquisition until their late thirties; meanwhile, 10-year-old girls only attain 15 percent of the adult maximum.

Changes in hunting yields with age demonstrate the skill- and learning-intensive nature of human hunting as well. For instance, the amount of meat Ache men obtain per hour spent hunting does not peak until the mid-thirties. Ten-year-old boys only obtain about 1 percent as much as the adult maximum, and even full-grown 20-year-olds only acquire 25 percent of the adult maximum.

Between 20 and 40, human hunters go through the equivalent of graduate school and a period of on-the-job training. This is because humans, unlike other predators, rely more on knowledge than on physical prowess. For example, Ache men know that the paca, the large rodent whose feces Grandfather Jaguar so adeptly prepared, lives in burrows with a main entrance and up to seven hidden escape hatches. When jaguars attempt to dig a paca out, it can run away unscathed through one of its alternative exits. But rarely does it fool an Ache hunter, who calls the other hunters to the hole while quietly and systematically searching for each escape route. When the other hunters arrive, a younger, less-skilled hunter is given the task of ramming a large log into the main entrance. Simultaneously, each of the remaining hunters dives on top of his respective escape hatch while his hands form a noose – allowing the fleeing paca's head through his open hands only to grab its throat and suffocate it.

Typically, human hunters do not even specialize in a single species but regularly exploit more than a dozen different kinds of prey, each with its own feeding habits, grouping patterns, and escape strategies. The skill and cleverness with which hunter-gatherers capture and prepare food may help explain why brains and longevity evolve together. Feeding niches that demand high levels of learning and information processing should not only select for bigger brains, but also for increased longevity because the brain costs a lot early in life, providing benefits only later in life. To illustrate the importance of longevity to a learning-intensive feeding niche, consider what would happen if a human forager group suffered the same mortality rates that chimpanzees do. Less than 10 percent of chimpanzees live to the age at which human productivity peaks. Humans depend on their parents for a long time, creating a large calorie deficit that they only gradually pay back during their highly productive middle adulthood years. With a chimpanzee life span, a human forager group would go into calorie deficit and become extinct.

Several other distinctive features of human history also make sense in light of this dietary niche. Physical growth in humans differs from growth in chimpanzees and gorillas. At first, it is faster: human newborns weigh about seven pounds, whereas newborn gorillas and chimpanzees weigh about four to five pounds. Moreover, human newborns are much fatter, having almost four times the fat that mammals of comparable weight have. The difference in the rates of brain growth may well be responsible for this difference in neonatal body size. At birth, a human's brain is about the same size as an adult chimpanzee's – huge considering that an adult chimpanzee weighs fifteen times more than a human newborn. Humans are probably born with relatively bigger bodies than other primates and more stored energy in the form of fat in order

to support the brain's rapid postnatal growth.

Following infancy and early childhood, humans grow more slowly than chimpanzees. In fact, growth is almost arrested for human children during middle and late childhood. By age 10, chimpanzees have caught up to and surpassed human children in body size. Perhaps human growth in middle childhood is slow because children do very little work and therefore do not need large bodies. Instead, they learn through observation and play. It is only in adolescence, when their brains are almost ready for large bodies, that humans undergo growth spurts and achieve their adult body sizes.

The flows of food and other services – both within and among families – that support this long period of dependency is also particular to humans. Unlike our ape relatives who give females the entire burden of feeding and caring for infants, humans engage in a sexual division of labor, a practice that appears to have ancient roots. Although the details vary from society to society, all existing hunter-gatherers and peoples who depend on a mix of foraging and farming worldwide cooperate in raising children. Typically, men focus on hunting but do some gathering as well, while women participate in a mix of activities such as gathering, food preparation, and childcare.

Why is a division of labor so fundamental to the human way of life? After all, women are physically capable of being adept hunters and sometimes do so when it is necessary. In one family of forager-farmers in Amazonian Peru, for example, the eldest daughters hunted with their father in order to feed the family because the only son was the youngest child. But unlike other predators, who often hide their young in a den while they hunt, human hunter-gatherers carry their babies with them from place to place – as do all primate mothers. This makes hunting a very risky activity for women with babies – and without contraception, women spend most of their adult lives prior to menopause either pregnant or lactating. Also, since traditional human hunting is so learning intensive, it only pays to hunt if one spends many years doing so. If women were to hunt only when they were not pregnant or nursing, they would get less food from hunting than from gathering because of their lack of experience.

The life course of a skill-intensive foraging species

Males probably became providers when hunted foods became an important part of human diets. The protein and fat from meat along with the carbohydrates obtained from plant foods created a balanced diet. Although the proportion of food provided by men and women varies across hunter-gatherer societies, men acquire, on average, about twice as many calories and seven times as much protein as women. After taking into account their own consumption, women supply only 3 percent of the calories to offspring while men provide the remaining 97 percent.

This assistance from men enables women to focus their energy on providing high quality childcare, resulting in almost double the survival rate for human children than for chimpanzees. In addition to behavior, women's physiology is consistent with an evolutionary history of extensive male parental investment. Unlike other primates, human females lower their metabolic rates during pregnancy and store fat, the result of receiving provisions. Human female foragers also tend to work less during lactation unlike female primates who heighten mortality risk by working more during lactation.

This extensive cooperation between human men and women would only make evolutionary sense if the reproductive performance of spouses were linked. Even though divorce is common in many hunter-gatherer societies, marriages stabilize once children are born. The long period of dependence on parents means that at any one time, most parents are raising several dependent offspring of different ages. This puts pressure on couples to stay together. In each of the five small-scale societies where I have lived and worked, men and women reported that stepparents are often less motivated to take care of stepchildren. And those who divorce and remarry while raising children frequently argue with new spouses over the division of resources among their joint children. Avoiding those conflicts is an additional incentive for a couple to stay together and have all or most of their children together.

Although men are physiologically capable of reproducing throughout their lives, most men appear to undergo 'behavioral menopause.' Among Ache hunter-gatherers, for example, if a couple had at least two children together, the woman's last birth was the same as her husband's last child in 90 percent of cases. Grandfather Jaguar had his last child more than two decades before he died.

From a biological perspective, menopause, whether physiological or behavioral, seems like an odd phenomenon. Most organs – such as the heart, lungs, liver, and brain – tend to age at roughly the same rate. This makes sense, since only whole organisms reproduce and all organs are necessary for survival. Chimpanzees and other primates can experience menopause, but in the wild almost all die before this age. Humans, and some whales, are rare in ceasing to reproduce with many years of life remaining.

Some have suggested that menopause evolved because women would leave more genetic descendants by helping as a grandmother than by continuing to reproduce. As women age, the reasoning goes, their pregnancies are less likely to succeed and they are more likely to die in childbirth. Indeed, among foragers, there are no 'golden' retirement years; both grandmothers and grandfathers spend significant time helping to raise children. Although older people switch to less physically demanding and more knowledge-intensive activities as they age, they still work very hard until death.

Still, an elderly person in a foraging society generally has a very short interval between the time that he or she is first unable to contribute and death. Stories like the one two brothers in Peru told me are not uncommon: When asked how their mother died, they said she had been very old and beginning to falter. One day while collecting bark in the forest, she collapsed at the foot of the tree. When she did not return, the brothers searched for her and found her almost unconscious. They ended her misery with one quick blow. Although they hated putting her to death, they expressed no remorse and agreed she would not have wanted to live any longer. Likewise, in lowland Bolivia only several months ago, an elderly man bled to death after falling logs crushed his leg. His nephew, in recounting the story, mentioned in passing that it was probably better that he died, since he would not have been able to work with a damaged leg.

While grandparents undoubtedly play an important role in traditional societies, this doesn't seem to account for menopause. Jocelyn Peccei points out

parenting duties alone could explain the two decades women survive after their last birth, since it will take their last child that long to become fully economically independent. In addition, attempts to measure the beneficial effects of aging people on the survival and reproduction of children and grandchildren have shown that they are not great enough to offset the costs of not having more children.

Overall, the aging of the human reproductive system remains a mystery. For example, why do females lose more eggs before rather than after puberty? In a process called *atresia*, eggs in the ovary 'die' at a relatively constant rate (with a brief acceleration in death rates just before menopause) until menopause when none are left. Perhaps atresia helps select among eggs so that only the most viable are fertilized; a slower rate of cell death might lead to poorer embryo quality and wasted investment in less viable offspring. Given the intense and prolonged investment that humans make in children, selection may have favored more quality control in our species.

Even though many mysteries are not yet solved, it is clear the human life course is an integrated adaptation to a specialized niche. Human digestive physiology and anatomy, nutritional biochemistry, brain growth and cognitive development, the timetable of physical growth, productivity throughout the life cycle, parental and grandparental investment, reproduction, and ultimately, the life span are coadapted to a learning-intensive feeding niche. It is this adaptive complex that has allowed people to colonize all the world's environments.

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The human life course is highly structured, consisting of at least six stages: the fetal and infant period, childhood, adolescence, early adulthood, middle adulthood, and old age. Fetal development and infancy – the first stage – is distinctive in its commitment to brain growth, supported by large fat reserves in both baby and mother. By age 5, infants have completed most of their brain growth and language learning, giving them the tools necessary for a lifetime of learning. The second stage, childhood, is characterized by very slow physical growth, a large allocation of energy to building the immune system, several important phases of cognitive development facilitated by play and other forms of practice, very low productivity, and very low mortality. Complete reliance on family, which reduces exposure to mortality hazards and allows time for learning, is another unique feature of human childhood. The slow physical growth of children eases the burden for families; faster growth would only raise the cost of rearing children until they are ready to provide food for themselves. The third stage, adolescence, follows. During this period, the brain and the rest of the body become ready for adult productivity: the body grows rapidly to adult size, the reproductive system matures, and the final phases of cognitive development occur.

'On the job training' distinguishes the fourth stage, from early adulthood to prime adulthood in the mid- to late thirties. Productivity increases significantly during this period, while mortality rates remain low and virtually constant. Men and women cooperate in a division of labor to raise children. Middle age, the fifth stage, is a period of simultaneous parenthood and grandparenthood. Dependency loads on parents peak around age 40 and gradually diminish over the next twenty years, as does productivity. The sixth stage, 'old age,' commences around 60. During this seventh decade of life, physical deterioration proceeds

rapidly and brain aging becomes evident, followed by a dramatic increase in mortality rates. Parenting is finished, and both work effort and productivity decrease. Nevertheless, older adults attempt to be as productive as possible, allocating more time to skill-intensive but less energy-intensive activities.

If this set of stages characterizes the average life course of human beings for the last one hundred thousand to two hundred thousand years, it is likely selection shaped the processes of maintenance and repair from the intracellular level to the whole-organism level to achieve a life span that would include all six stages. It may be that the sixth stage – old age – is actually an artifact of selection to maintain the body and mind in good condition through the first five stages of life. The sixth stage could thus result from the impossibility of nature designing a body that collapses the moment the aged person is no longer highly functional. Another possibility is that the sixth stage has been positively selected for because of the fitness benefits produced during old age.

This detailed understanding of our life course sheds light on how evolution has shaped the life spans of other organisms. The majority of long-lived organisms do not have large brains and a learning intensive feeding niche. For the most part, evolutionary biologists have focused on 'extrinsic' mortality risks to explain longevity: some types of organisms face higher risks of dying from predation, disease, and accidents. When the risk of dying from these extrinsic causes is high, it does not pay to repair the body and slow down aging. Steven Austad, for example, has proposed that primates live longer than comparably sized mammals because living in trees make them less vulnerable to predation.

Such risks may be important determinants of the aging process, but the human story also directs us to think about the *value* of living to older ages and why that should vary from one organism to another. In the human case, it appears that the value of slowed aging is related to the investment in learning and the shift in productivity as one ages.

The value of living to older ages may help explain the five-thousand-fold variation in the life spans of insects, from mere days to more than a decade. All the longest-lived insects are queens in social colonies, in species as diverse as ants, bees, and termites. These social insects make a large investment in their physical plant – building tunnels, storage rooms, reproductive chambers, and defensive structures. It takes time to build such a plant and to grow a large workforce, but once it is completed, a large colony can be extremely productive. This may be why insect queens can sometimes live over a decade. Other factors may also affect the value of long life.

Besides helping us to understand the present, we can also use our evolutionary past to look to our future. When compared to other species, human life histories have a characteristic pattern, yet they also vary systematically with the environment. Perhaps the most dramatic example is the pattern of changes accompanying modernization – a pattern often referred to as *the secular trend*. Improved nutrition and decreased disease loads have systematic effects on human developmental physiology: physical growth is more rapid and maturation begins earlier, resulting in greater stature and body weight and earlier puberty in girls. This change probably reflects adaptive flexibility in growth and maturation that evolved in response to

variation in food supply and disease assault rates during human evolutionary history.

Slower aging may also be a response to better nutrition and a lower burden of infectious disease. On the other hand, heart disease, diabetes, and cancer because of excess fat and lack of exercise are more prevalent than ever – and possibly also the result of evolved responses. Past activity regimes and the variability in food supply may have selected for human appetites and nutritional biochemistry designed to store fat and increase blood lipid levels when food is abundant. Those very adaptations, however, reduce the life span in the context of modern activity regimes and virtually unlimited food access.

Behavior also responds to modernization. With more education comes increases in longevity, though more schooling lowers earnings early in adulthood. Just as in the learning-intensive foraging niche, though, earnings then rise later in adulthood. It is not known why higher levels of education, on average, correlate to longer life, but the effect is rather strong. The large educational gradient in longevity throughout evolutionary history may be partly responsible for this effect. Or, it may be due partially to a lifestyle change that is consciously or unconsciously concerned with living longer.

Whether we are now reaching the upper limit of our flexibility in the life span is the subject of an ongoing and lively scientific debate. With respect to stature, and perhaps age of menarche, the data suggest we may be nearing the limit. But there appears to be more scope for variation in the life span, given investments in medical technology designed to reduce disease and the effects of aging.

And better knowledge of the human genome is likely to lead to manipulations of genes and gene products, resulting in large-scale life span increases. Greater understanding of cell and DNA repair mechanisms that have evolved by natural selection may also play an important role in lengthening life. If the difference in rates of aging between humans and our ape relatives is any guide, another leap in life span could well be possible in our near future.

The life course of a skill-intensive foraging species

Dennis J. Selkoe

The aging mind: deciphering Alzheimer's disease & its antecedents

Last scene of all, that ends this strange
eventful history,
Is second childishness, and mere
oblivion …

– Shakespeare, *As You Like It*

Before the last century, only a small portion of the human population survived into the eighth decade of life. Those few individuals who successfully avoided the myriad causes of adult mortality – principally, infectious diseases, trauma, and cardiovascular failure – were expected to face a steady attrition of their most human qualities: memory, reasoning, judgment, abstraction, and language. In the popular mind, and even among scientists and philosophers, the idea that great age inevitably brought about an inability to think clearly was widely accepted. But intensive research into the pathology and biochemistry of the aging brain during the last few decades has revealed that specific diseases cause major impairment of cognition late in life and that the process of aging per se results only in relatively subtle changes in certain mental functions.

This reinterpretation of the nature of the aging mind has profound implications on both the personal and societal levels. In contrast to the assumption inherent in Jacques' soliloquy, the passing of time does not by itself destroy our ability to think cogently. Rather, certain diseases that devastate those areas of the brain serving memory and cognition become increasingly prevalent after age 70 or so. For example, the two major causes of late-life dementia in most developed nations, Alzheimer's disease and multiple small strokes (multi-infarct dementia), afflict just a few individuals in their forties or fifties, but the numbers rise very substantially in the mid-sixties and beyond.

In this sense, aging, the passage of time, does contribute to the develop-

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ment of dementing diseases in at least two broad ways. First, over time, the brain accrues molecular and cellular defects in neurons and glia, which reduce its physiological reserve, just as occurs in muscle cells with age. This process makes the brain more susceptible to loss of function if and when a neurological disease is imposed. Second, some of the specific diseases that cause dementia require great time to produce enough brain abnormalities, or lesions, to compromise function. For instance, in Alzheimer's disease and certain other dementias, a lot of time is needed to reach a critical tissue concentration of particular proteins that allows for their polymerization into potentially toxic forms. In short, the process of brain aging can contribute to the development of a clinically noticeable dementing illness, but aging by itself appears to be insufficient to cause the illness.

Life expectancy at birth in the United States and in many other developed nations has risen from roughly fifty years in 1900 to more than seventy-five years in 2000, an unprecedented 50 percent increase in just one century. This sudden jump in average longevity is the result of major improvements in public health, intensive biomedical research, and subsequent pharmacological, surgical, and lifestyle interventions. It is by no means assured that life expectancy will continue to rise in the coming century, with the threat of highly resistant infectious diseases and an emerging epidemic of obesity and associated metabolic disease.[1] Nevertheless, the sheer number of humans now surviving beyond eighty years and the accompanying social and economic stresses demand that the scientific community focus far more attention on the determinants of successful aging and the prevention of age-linked disease – particularly in the brain, which helps regulate non-neural organ function.

Based on personal observation, many people have come to realize that the aging process does not usually wreak havoc on the mind. But as recently as thirty years ago, gerontologists and neuroscientists were not at all sure of this conclusion and continued to catalog a complex array of relatively minor deficits in the numbers and biochemical properties of brain cells in aged mammals, including humans. Understandably, scientists focused mostly upon the health of neurons, the excitable cells in the brain that convey signals through electrochemical impulses – for example, a response to light impinging upon the photoreceptor cells of the retina or to sound waves vibrating the hair cells of the inner ear. Because the long cytoplasmic extensions of neurons, the axons and dendrites, pass information from one place to another in the brain, age-related defects in the innumerable molecules that allow them to do so could lead to cognitive failure. Indeed, scientists have documented a host of quantitative and qualitative changes in neuronal receptors, enzymes (specialized proteins that catalyze chemical reactions), structural proteins, and lipids in the brains of aged rodents, lower primates, and humans.

But when one counts the actual numbers of surviving neurons in aged versus middle-aged or young brains, most brain regions show very little or no significant neuronal attrition. This recent realization flies in the face of the long-held assumption that neurons steadily die out during the life span, a conclusion based

1 S. J. Olshansky et al., "A Potential Decline in Life Expectancy in the United States in the 21st Century," *New England Journal of Medicine* 352 (2005): 1138 – 1145.

on what we now recognize as technically flawed cell-counting methods. For example, the number of pyramidal neurons in certain areas of the hippocampus, a seahorse-shaped brain region critical for memory, does not decline appreciably in older humans.

On the other hand, the number of neurons in the *substantia nigra* – a small cluster of neurons in the brain stem that secrete the neurotransmitter dopamine – does decline steadily with age, perhaps because these cells produce the pigment neuromelanin as a by-product of their dopamine metabolism, a process that results in the excessive oxidation of proteins and lipids. The age-related dysfunction and loss of *substantia nigra* neurons likely contributes to the decreased speed and fluidity of movement and somewhat stooped, shuffling gait that very old people often display. This finding provides an example of the relationship of the aging process in the brain to diseases of the elderly. Age-associated nigral cell loss, which may normally amount to 30 to 50 percent or so of these neurons, is not sufficient to induce the clinical syndrome of Parkinson's disease. However, this level of attrition may reduce the physiological reserve enough so that a superimposed insult, e.g., the presence of an inherited mutation in a specific gene or prolonged exposure to an environmental toxin, may elevate the degree of nigral cell loss to some 70 to 80 percent, enough to produce clinically apparent symptoms of Parkinson's disease. But it must be added that the loss of neurons during normal aging in the *substantia nigra* is more severe and predictable than one observes in many other regions of the brain such as the cerebral cortex.

Even when the absolute number of neuronal cell bodies does not decline substantially, the brains of older mammals reveal a remarkable array of cellular and molecular alterations. There are defects in nuclear and mitochondrial DNA; in many different proteins, particularly enzymes; and in the lipids of the membranes enveloping cells and internal organelles. What bearing do these diverse molecular changes have on the mind?

For most of us, the answer is very little. In aged people without Alzheimer's disease and other mind-threatening illnesses, the clinical effects of biochemical and anatomical alterations seem to be modest. In many studies reporting age-related neurochemical deficits – such as a reduction in a particular enzyme or in certain proteins or RNA molecules – the levels or functional activities in elderly adults have ranged from 5 to 30 percent below those in young adults. And though a 30 percent loss might seem quite high, such gradual declines over several decades often have little measurable effect on thinking. Indeed, positron emission tomographic (PET) scans and functional magnetic resonance imaging (fMRI) scans show that the brains of healthy people in their eighties are almost as active metabolically as those of people in their forties. In some brain regions such as parts of the frontal cortex, healthy aged humans may even exhibit more metabolic activity, though it is unclear whether this seemingly paradoxical rise in activity represents the brain's attempt at compensation for some neuronal loss or just a nonspecific and potentially adverse recruitment of remaining local neurons.[2] Overall, the aged brain tolerates relative-

2 R. L. Buckner, "Memory and Executive Function in Aging and AD: Multiple Factors that Cause Decline and Reserve Factors that Compensate," *Neuron* 44 (2004): 195 – 208.

ly small deficits in neuronal structure and function rather well, although certain mental functions required for highly specialized activities – such as the rapid visual-motor tasks required to pilot a 747 or perform complex surgery – may become compromised in older humans.

Epidemiological and neuropsychological studies generally paint a similar picture to that emerging from neurobiological research. Estimates of the prevalence of senile dementia – the progressive loss of cognitive function after roughly age 65 – vary widely, but most data suggest that a large majority of individuals in their seventies and eighties are free of significant cognitive loss that interferes with daily function. And analyses of healthy elderly adults reveal only subtle declines in performance on tests of memory, perception, and language. One decrement on which numerous studies agree, however, is a reduction in the speed of some aspects of cognitive processing. Hence, septuagenarians are often unable to quickly retrieve certain details of a particular past event – say, the precise date or place – although they are often able to recall the information minutes or hours later. Given enough time and an environment that keeps anxiety at bay, many healthy elders score almost as well as young or middle-aged adults on tests of mental performance. A measure of guarded optimism emerges from investigations of 'normal brain aging': one may not learn or remember as rapidly later in life, but one may learn and remember nearly as well.

The range of brain diseases that express themselves as a progressive loss of intellectual function is remarkably broad. Vascular, metabolic, infectious, neoplastic, traumatic, and degenerative disorders can all present with symptoms of dementia.

At different times over the course of the last century, various disorders have assumed greater or lesser relative importance in contributing to late-life dementia. In the early 1900s, for example, neurosyphilis was considered a common cause of dementia; Alzheimer's disease had not yet been recognized as a specific brain disorder. More recently, the proportion of dementia cases attributable to one or more strokes has declined because of the successful control of hypertension and hyperlipidemia and the gradual reduction in some types of cardiovascular disease. When Alzheimer's disease comes under reasonable medical control, other disorders will assume greater relative importance in the differential diagnosis of late-life dementia.

But in developed countries today, Alzheimer's disease is still by far the most common basis for senile dementia, accounting for some one-half to two-thirds of all cases. For several decades after Alois Alzheimer reported his index case, a 53-year-old woman from Frankfurt, the disorder was classified as a rare 'presenile' dementia, that is, a dementia having its onset prior to roughly age 65. But in the mid-1960s, three British scientists – Garry Blessed, Bernard Tomlinson, and Martin Roth – conducted landmark clinical-pathological correlative studies that made clear what some earlier investigators had suspected: common senile dementia is usually associated with the classical findings in the brain that Alzheimer had described. The term 'senile dementia of the Alzheimer type' was subsequently coined, but nowadays, 'Alzheimer's disease' designates this syndrome, regardless of the age of onset. For research purposes, one still refers to 'early-onset AD' and 'late-onset AD,' divided arbitrarily at age 65, but little evidence exists that these are fundamentally distinct biological processes or that

we could not ultimately treat them as one entity.

In the United States, multi-infarct dementia has long been considered the second most common basis for late-life dementia, even though Parkinson's disease-associated dementia plus a related disorder, Lewy body dementia (named after the characteristic neuronal lesion that defines Parkinson's disease), are now equally if not more prevalent. Careful microscopic analyses of autopsied Parkinson's disease brains often reveal the features of AD or else AD plus Lewy body dementia, confounding precise diagnostic classification. Nevertheless, 'pure' Alzheimer's disease is still the most common neuropathological basis for late-life dementia in the United States and most developed countries. A number of less common causes of dementia, including frontotemporal dementia and Creutzfeld-Jakob disease, share certain pathological or biochemical features with AD, but they are etiologically distinct.

Virtually everyone beyond late middle age has worried that an occasional memory lapse – a name forgotten or an object misplaced – could represent the earliest sign of AD. But such momentary losses, with recovery of the detail within minutes and a complete awareness of the lapse, are usually not progressive. In contrast, the repeated inability to remember recent, minor episodes of daily life – a call from a friend, a trip to the department store, the paying of a bill, a brief news story – can represent the earliest harbinger of AD. In a condition now referred to as 'mild cognitive impairment (MCI)-amnestic type,' the individual shows a subtle, intermittent decline in episodic memory but is otherwise intact cognitively and performs very well in everyday life. Evidence from structural and functional magnetic resonance imaging of MCI-amnestic brains suggests that the neuronal dysfunction is restricted to the hippocampus and a small number of other brain structures connected to it. Studies of the fate of MCI-amnestic subjects over time suggest that roughly 12 to 15 percent of them 'convert' to clinically diagnosable, mild AD each year, meaning that these individuals begin to exhibit signs of a more general disturbance of recent memory as well as disorientation to time and place, decreased attention span, confusion in executing complex tasks, and sometimes, difficulty in finding words. This slow progression of cognitive symptoms occurs in an individual who appears fully alert and demonstrates no abnormalities of the motor system, e.g., decreased mobility, stiffness, and slowed gait, until later in the disease.

What causes this initially subtle but ultimately devastating loss of higher cortical function? The answer has begun to emerge from three decades of intensive neuropathological, biochemical, and genetic research. While there is still earnest debate about the detailed sequence of events, the majority of scientists researching AD now believe that the misfolding, aggregation, and accumulation of a small protein of forty-two amino acids, the amyloid ß-protein (Aß), initiates a complex cascade of molecular and cellular changes that compromise neuronal function in brain regions serving memory and cognition.

According to this scenario, widely referred to as the 'amyloid (or Aß) cascade hypothesis,' a chronic imbalance between the production and the clearance of this otherwise normal protein arises in the brain long before the first symptoms of dementia. This accumulation leads to the self-association of Aß into 'oligomers' (doublets, triplets,

quadruplets, etc.), which in turn can assemble into filamentous polymers ('amyloid fibrils') that clump together to form the cores, or spherical centers, of tiny plaques. These amyloid deposits are gradually surrounded by degenerating axons and dendrites (collectively called neurites) and activated brain inflammatory cells (microglia and astrocytes), completing the formation of so-called neuritic plaques.

During this slowly evolving process, some of the neurites within and adjacent to the emerging plaque develop rigid intracellular filaments, or 'paired helical filaments,' that are composed of a neuronal protein called tau. Tau filaments also accumulate in large bundles that comprise the neurofibrillary tangles found inside many neuronal cell bodies in the hippocampus and cerebral cortex, as well as in certain subcortical neurons that send their axons to these areas. In short, the accumulation and self-assembly of the Aß protein is believed to initiate a series of first functional (biochemical) and then structural (anatomical) changes in selected neurons, to the ultimate detriment of the thinking process.

Perhaps the most compelling evidence for this Aß hypothesis has come from identifying and characterizing genetic mutations that cause rare inherited forms of AD. It is a truism of modern biomedicine that searching patients' genomes for faulty genes opens up the study of diseases of previously unknown cause and mechanism. For example, until the cloning of the Huntington gene in 1993, no one had any real clue as to what might be killing off certain brain neurons in patients with Huntington's disease. In this and many other heritable diseases, the unbiased search of the human genome for the genes responsible for the disease allowed scientists to subsequently formulate biochemical hypotheses about what actually kills cells. But in the case of Alzheimer's disease, the opposite sequence occurred: progress in the 1980s in understanding the biochemistry of the disease identified the proteins that comprise the plaques and tangles, providing geneticists with key clues to the location of the DNA mutations that might cause Alzheimer's disease.

In 1991, researchers discovered the first mutation responsible for AD on chromosome 21, specifically in the gene that encodes the amyloid precursor protein (APP), the parent protein of Aß. In addition to the fact that APP molecules give rise to the Aß fragments that form the neuritic plaques, a crucial clue that the APP gene might be the site of an AD-causing defect came from a disorder at the opposite end of the life span: Down syndrome. Humans with Down syndrome, or trisomy 21, the most common form of chromosomal duplication compatible with life, invariably develop the plaques and tangles of AD in their thirties and forties. This is because they harbor three copies of the APP gene in all of their cells, rather than the usual two copies. The extra copy of the APP gene results in a roughly 50 percent increase in the cellular levels of the APP protein throughout life and the consequent start of Aß deposition in the Down syndrome brain as early as age 10.

Another powerful clue pointing to the APP gene had come from studying a family in the Netherlands with a history of multiple brain hemorrhages caused by the severe build-up of the Aß protein in cerebral blood vessels. In 1990, scientists discovered that a mutation in the APP gene that changes a single amino acid within the Aß region of APP was responsible for this rare disorder, demonstrating for the first time that

mutations in APP could cause Aß accumulation.

With all of this knowledge in hand, geneticists scrutinized the APP region of chromosome 21 in a few families with a hereditary form of AD that led to the onset of dementia in the fifties. In one such family, they discovered a 'missense' mutation in APP that changed one amino acid near the end of the sequence encoding the Aß region to another. The study of other families with early onset of AD revealed additional APP missense mutations, most of which occurred in amino acids either at the beginning or at the end of the forty-two-residue Aß region. Tellingly, geneticists did not find any AD-causing mutations away from the Aß region of this large (770–amino acid long) precursor protein, indicating that the mutant amino acids might lead to increased cutting of APP at the beginning or end, resulting in the heightened production of the Aß fragment.

As these genetic findings were emerging, a major biochemical discovery was made: all cells normally produce the Aß peptide throughout life. Thus, Aß is the product of healthy APP metabolism in all of us, implying that unknown factors – genetic, environmental, or both – can increase its production or decrease its degradation in those individuals who develop AD, all of whom have too much Aß in their brains.

Putting together these two key observations – that healthy cells continually make Aß and that rare mutations within its precursor, APP, can cause AD – led to groundbreaking experiments. Inserting a gene that bore an AD-causing APP mutation into cultured cells resulted in significantly greater Aß production. Scientists could now study many details of the production and metabolic fate of Aß in simple cell models. They could also use such cells to screen large libraries of drug-like molecules and pinpoint compounds that lower Aß production without damaging the cells. And through the wonders of genetic engineering, scientists could also create 'transgenic' mice that express a human APP gene bearing an AD-causing mutation. After considerable trial and error, the latter approach generated several highly useful mouse lines that mimic several, but not all, features of AD in their brains, including the abnormalities of neurites and glia around the amyloid plaques. As they age, these mice develop deficits in cognition such as difficulty remembering how to negotiate mazes efficiently. Taken together, these and many other experiments have produced a wealth of evidence that AD can arise at least in part from an imbalance in the economy of the Aß protein in brain regions important for memory and cognition. The practical outcome has been to encourage scientists to find ways to lower Aß levels in humans.

Still, there are many unanswered questions about the Aß hypothesis. What causes the imbalance in Aß levels in the brains of the large majority of AD patients who do not have known genetic mutations? For example, can environmental factors influence the brain's Aß levels? Does the Aß peptide begin to aggregate inside the neuron before the Aß oligomers are exported into the extracellular space and then bind back to the cell? Which type of brain cell – neurons, microglia, or astrocytes – is the first to respond adversely to the excess of Aß in the local microenvironment? Precisely why do neuronal extensions, i.e., axons and dendrites, respond with an aggregation of their tau protein? Are the resultant tau aggregates the prime culprits in compromising neuronal function and ultimately killing the neurons? And perhaps most perplexing, how does the en-

tire process select for neurons serving memory and cognitive function?

Answering all of these questions in detail should not be necessary in order to treat or even prevent Alzheimer's disease. Because human genetic data and the modeling of the effects of the faulty genes in engineered mice have continued to support the Aß hypothesis, scientists in both academia and the biopharmaceutical industry have spent the last decade devising strategies to interrupt the Aß cascade at an early point in its development.[3] Without knowing precisely how Aß compromises the functions of selected neurons, they have searched for compounds that can decrease brain Aß levels, initially in mouse models.

Three broad approaches have been conceptualized. First, one could partially inhibit one of the two specialized enzymes, ß-secretase and γ-secretase, that cut APP to release the Aß region. Second, one could allow these reactions, which occur normally in all of us, to proceed unimpeded but instead prevent a single Aß protein, a monomer, from binding with another to form oligomers, the small aggregates that appear to initiate the amyloid build-up and the associated short circuiting of neurons. Third, one could attempt to 'clear' the brain of various forms of Aß, including monomers, oligomers, and larger amyloid deposits.

The first approach – inhibiting the protein-cutting enzymes that generate Aß – is somewhat analogous to the use of statin drugs to decrease cholesterol production. Several groups have identified inhibitors of ß-secretase, the enzyme that cuts APP first. But these inhibitors require modification to make them more potent yet still able to penetrate the blood brain barrier and achieve effective levels in brain tissue. At this writing, there are no such ß-secretase inhibitors ready for human testing. Scientists have also discovered many small molecules able to inhibit γ-secretase, the enzyme that makes the second and final cut of APP. Unfortunately, most of these molecules also interfere with the cutting by γ-secretase of a protein called 'Notch' that is crucial for the normal functioning of most cells. However, the serendipitous discovery that certain anti-inflammatory drugs like ibuprofen can gently 'tweak' γ-secretase to lower the production of Aß42, a particularly noxious form of Aß, without decreasing Notch cleavage has helped researchers continue to pursue this approach. And since the anti-inflammatory properties of such drugs are not responsible for this selectivity, scientists have identified and are now testing in humans derivatives that solely tweak γ-secretase. Early trial results suggest that these specialized 'γ-secretase modulators' may indeed slow cognitive decline, at least in some AD patients.

The second approach, preventing the self-assembly of Aß into oligomers and fibrils, makes good theoretical sense but has received less attention. While some compounds have performed well in test-tube experiments, very small assemblies of Aß (dimers and trimers) can already interfere with synaptic function and behavior, raising concern that a partial inhibition of Aß aggregation might stabilize such small species and actually worsen the disorder.

The third approach – clearing Aß from the brain – has progressed the furthest to date, advancing into human trials. Here,

3 D. J. Selkoe and D. Schenk, "Alzheimer's Disease: Molecular Understanding Predicts Amyloid-based Therapeutics," in *Annual Review of Pharmacology and Toxicology*, vol. 43, ed. A. K. Cho, T. F. Blaschke, P. A. Insel, and H. H. Loh (Palo Alto, Calif.: Annual Reviews, 2003), 545 – 584.

the novel idea of immunizing patients with the very peptide that builds up in their brains has led to evidence in mice that one can efficiently clear Aß plaques with Aß antibodies. This has been accomplished in two ways: either actively vaccinating the mice with synthetic Aß so that they gradually generate their own Aß antibodies, or passively administering laboratory-made Aß antibodies to them. When the active vaccination approach was initially tried in AD patients, some 6 percent developed inflammatory cell infiltrates in the brain, or meningoencephalitis, and the trial stopped. The apparent reason for the inflammation: some patients had generated specialized T-lymphocytes directed against the tail end of the Aß peptide. Modified active vaccines comprising the front end only have now been designed but not yet tested in humans. In the meantime, a phase 2 trial of passive antibody administration is underway in AD patients, with initial results hoped for by late 2006.

In addition to the above approaches to the Aß part of the AD equation, there are strategies that attempt to target other key steps in the disease cascade. These include oxidative injury to neurons, the build-up of tau as tangles, local inflammatory changes, or a potential imbalance of certain metals such as copper and zinc in the AD brain. The use of cell culture and mouse models has assisted in the development of each of these potential therapies, followed in some cases by the initiation of clinical trials. At this writing, unequivocal evidence of successful slowing of the disease has not emerged, but hope runs high.

The advent of therapeutic agents that slow and perhaps even prevent AD could have profound effects on the aged human population, both on the individual and the societal levels. A vaccination strategy for a noninfectious disease in late life is unprecedented. Were a safe vaccine or another Aß-lowering therapeutic such as a γ-secretase modulator approved, healthy people might avoid the onset of Alzheimer-type cognitive loss by undergoing the therapy in late middle age or perhaps even earlier. Such an approach would have to include a formal, semiquantitative assessment of an individual's likelihood of developing AD. Components of such a risk assessment may encompass a neurological examination that includes cognitive testing, a detailed family history, a blood screen for genetic mutations known to predispose to AD or other dementias, a blood test for plasma Aß levels, and a special brain imaging procedure like the emerging 'amyloid scans' that employ an injected chemical agent to visualize one's cerebral Aß burden. Such a multicomponent assessment could assign individuals a rough probability of developing AD and perhaps other dementias, and those in moderate- or high-risk categories could then be offered one of the preventative agents envisioned above.

While such a combined diagnostic/therapeutic paradigm seems achievable with time, it raises difficult new questions. How can we administer such a relatively complex protocol to very large numbers of aging individuals? How will we pay for it? Will only relatively well-off individuals in developed nations have access to it, at least for the foreseeable future? And how will we handle the ethical challenges posed by widespread testing for the genetic risk of a major, brain-destroying disease?

And there are other social implications to ponder should a successful therapy for Alzheimer's disease emerge from current research. The prospect of many more people retaining most of

their cognitive functions into late life should accelerate the current trend toward longer careers, potentially displacing younger workers. And because improvements in the physical health of octogenarians will likely accompany the prevention of Alzheimer's disease, and later other dementias, we will need to expand the availability of activities such as driving, entertainment, tourism, and financial services. Healthy elders themselves will presumably provide much of the labor required to deliver these services, but younger members of the work force should also benefit from these new opportunities.

Medical questions also abound. Could widespread access to effective therapy for late-life cognitive failure actually increase longevity? Certainly, the average life expectancy at birth would rise modestly, at least in developed societies, but will resolving dementia have a direct and measurable impact on the maximal age that humans achieve? Will many more people live to 90 or 100 with their mentation largely intact and then succumb fairly rapidly to other causes of mortality? And will other, currently infrequent forms of cerebral deterioration take the place of Alzheimer's disease as the primary cause of dementia, just as Alzheimer's emerged strongly after the eradication of neurosyphilis and the more recent decline in strokes?

The looming prospect of solving Alzheimer's disease should be incorporated into the thinking of politicians, economists, and all those concerned about planning the future of our societies. While we will no doubt experience numerous fits and starts along the way, it appears increasingly likely that a world with less Alzheimer's disease lies ahead.

Caleb E. Finch

Aging, inflammation & the body electric

In a famous photograph of Walt Whitman taken in the 1860s (see inside back cover), the great American bard looks wizened – his hair white, his face weathered. He looks, in short, like an old man. In fact, he was only in his forties.

During the Civil War, Whitman spent hours each day in hospital wards attending to desperately sick soldiers, which exposed him to dysenteries and horribly infected wounds. As a result, a bad infection in one hand climbed up into his shoulder, and he became beset by chronic headaches and fevers.

One hundred and fifty years after Whitman sang "the body electric," we can find in Whitman's fate some clues to the nature of aging. For much of his adult life, he complained of chronic headaches, fevers, and weakness. At the age of 55, he suffered a stroke that paralyzed his left side. Other strokes followed, though without noticeably impairing his memory. Whitman eventually lived to the age of 72, exceeding his generation's life expectancy by about thirty years. Yet shortly before his death, one of his doctors noted, "His apparent age was greater than his real years."

A postmortem by experts in gross morbid anatomy showed that Whitman had long suffered from both meningitis and tuberculosis. Tuberculous meningitis may have contributed to his strokes and would have been consistent with his other reported ailments. Both infections inflame arteries at the base of the brain, which, in turn, increases the risk of infarcts and strokes that selectively damage deep brain centers in a 'TB zone,' but usually spare higher cognitive functions. Although tuberculous meningitis is a rare disease, the 'Whitman case' points us to more general principles in aging.

Inflammation is increasingly recognized as fundamental to aging. As modern medicine has brought infectious diseases like tuberculosis and meningitis under control, successive generations have had to carry less of the inflammatory burden of such diseases – which may help account for recent improve-

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ments in human longevity. Changes of the inflammatory burden may also anticipate limits ahead. Aging, of course, is an immensely complex process governed by multiple gene-environment interactions. No single factor governs aging – biogerontology is a graveyard of single-cause hypotheses.

In the past century and a half, human life spans have increased remarkably, with one year added to the life span for every three to four years of calendar time. Before the Industrial Revolution, the average life span was about thirty-five to forty years. Even if one survived the hazards of childhood to reach maturity, the remaining life span was still shorter than today's. Currently, life expectancy in most developed countries has doubled to about eighty years and continues to climb. The result is a major shift in population age structure, from broad-based pyramids with younger groups in the majority, to skyscraper-shaped structures, which are arising everywhere because of the steadily growing rates of survival at younger ages and survival to increasingly older ages. In fact, centenarians are the fastest expanding age group. Sitting on the topmast is Jean Calment (1875 – 1997) who tested cognitively normal at 119. Her 122-year longevity may yet be superseded.

The decline in childhood mortality rates is yet another remarkable change that has occurred in recent generations. Not so long ago, infant and childhood mortality rates were very high almost everywhere; rates of 30 percent were very common in Europe and North America until the Industrial Revolution, when early-age mortality began to decrease. This is significant because childhood mortality trends are the strongest predictors of later mortality. In following birth cohorts over their life spans, Eileen Crimmins and I discovered that the survivors in birth cohorts with high early-age mortality rates showed much higher mortality rates at all later ages than survivors in birth cohorts with lower early-age mortality rates.[1] Evidence from Sweden, which has kept remarkably complete records of mortality since 1750, makes a very strong case for this.

We hypothesized that this outcome was the result of chronic infections and inflammations accelerating aging processes. For example, rheumatic fever, the result of streptococcus infections, killed many children in the 'bad old days' before antibiotics. But the disease continued to affect even its survivors, who rarely lived beyond middle age, because the bacterial colonization of the valves had weakened their hearts. TB was another common scourge often acquired in childhood that shortened adult life spans. Thus, it appears that early infections have a strong connection to adult longevity.

This 'cohort morbidity' has gradually decreased during the past two centuries. Its decline began long before people understood the germ theory of infectious transmission and very long before modern medicine discovered vaccination and antibiotics. By Whitman's time, and even a century before in Sweden, societies were improving public health and personal hygiene. The stink of human waste and rotting garbage was becoming less acceptable. Governments were increasingly expected to provide clean public water, covered sewers, and sanitation squads to clean up after horses. Improving transport was also giving broader access to better food year round.

1 Caleb E. Finch and Eileen M. Crimmins, "Inflammatory Exposure and Historical Changes in Human Life Spans," *Science* 305 (5691) (September 17, 2004): 1736 – 1739.

Still, it is fair to say that we do not fully understand the precise relation of the improvements in education, hygiene, and nutrition to the advance in health during the Industrial Revolution.

We do know, though, that the legacy of bad conditions can persist for several generations. Low–birth weight babies not only grow up with a higher risk of heart disease and hypertension, but also tend to produce relatively small progeny of their own. Impaired fetal growth affects the pelvic blood vessels, which may never develop optimally even with good nutrition after birth. Maternal infections may be at least as important as nutrition in impairing fetal growth: for example, women with HIV or TB, even with good diets, tend to have smaller babies. Smoking can also have transgenerational effects. If your grandmother smoked during pregnancy, your risk of asthma is two times greater because the egg from which you came was fully formed (and, as a result, exposed to carcinogens) while your mother was still a fetus. We may find still other infections and environmental inflammogens with persistent effects; such factors will likely slow or limit future average increases in life span.

When Walt Whitman sang of the "body electric," he coined a metaphor that turns out to be more literally true than he may have imagined. Our aerobic metabolism is continuously producing free radicals, 'chemical sparks' that attack invading microbes with their highly reactive, unpaired electrons.[2] When microbes enter our bodies, macrophages, one of the most ancient immune cells, are rapidly activated during the 'acute phase response.' Enzymes in these macrophages convert oxygen to the radical superoxide and fuse with carbon, chlorine, hydrogen, and nitrogen to form other radicals. As the free radicals from activated macrophages diffuse from their cell source to attack an infection, however, they also inflict local 'bystander' oxidative damage on other cells and molecules.

During systemic infections, the liver also shifts gears to secrete inflammatory proteins, e.g., C-reactive protein (CRP), cytokines such as interleukin-6 (IL-6), and complement system proteins. CRP is an ancient protein that binds to certain classes of bacteria and enhances their uptake and digestion by macrophages. IL-6 and other cytokines cause fever and also mediate the next phase of host defense, instructive immunity, which emerges days later with specifically targeted antibodies or cell defenses through B-cells and T-cells.

While free radicals play a vital role in the body's defense as well as in the normal and essential signaling between cells, they can also cause slow yet cumulative damage to irreplaceable molecules and cells. Such multiple effects, or 'pleiotropies,' underlie a basic principle in aging called 'antagonistic pleiotropy': some mechanisms that evolved to mete immediate benefits to the young have delayed consequences that slowly emerge during aging. Another example is glucose. While an essential fuel, glucose also spontaneously reacts with the amino groups of proteins and nucleic acids. The resulting oxidative modifications, or advanced glycation end-products (AGE), can cross-link proteins and cause DNA mutations. We know that chronic hyperglycemia, as in diabetes, accelerates AGE formation. Moreover, in diabetic neuropathology, AGEs also

2 In chemical terms, 'free' refers to the availability of the unpaired electron to form a stable chemical bond; the taking of electrons from another atom by free radicals is called oxidation.

cause cell death by synergizing with free radicals.

Because many chronic diseases of aging develop through inflammatory mechanisms, inflammation and aging are now merging fields of research. The case is strongest for vascular disease as an inflammatory process. Because atheromas, the raised fatty plaques in the arteries that can contribute to vascular disease, are actually hotter than surrounding vessels, the idea that the sparks within are driving aging is not just a metaphor. Atheromas are loaded with inflammatory cells and proteins – cytokines, complement factors, etc. In fact, the foam cell of atheromas, which accumulates lipids, is an activated macrophage.

Moreover, in brains with Alzheimer's disease, senile plaques harbor many of the same inflammatory processes found in vascular atheroma. Senile plaques are surrounded by the brain's special macrophage, the microglial cell. Besides the cytokines and complement factors also found in atheromas, Alzheimer plaques contain the beta-amyloid protein, which is induced by hemorrhage and inflammation.

Inflammation seems also to play a key role in the etiology of heart attacks. Elevated blood CRP and IL-6, when combined with elevated LDL, result in a fivefold or higher risk of a future heart attack. In the Honolulu Asia Aging Study, elevated blood CRP also predicted a threefold higher risk of later developing Alzheimer's disease and vascular dementia. Tellingly, most of the known risk factors for heart attacks are also risk indicators for Alzheimer's disease and vascular dementia.

Vascular disease typically begins decades before symptoms arise. At autopsy, soldiers killed in Korea and Vietnam often had advanced fatty plaques in their arteries. Even fetuses often have minute versions of atheromas. While these 'prodromal' atheromas do not block arteries they may seed further growth. Ultrasound imaging has also shown that obesity and diabetes can accelerate the growth of atheromas even in childhood. Over the course of a lifetime, the aorta and most other arteries progressively accumulate fatty deposits. Some spontaneously regress, while others balloon into full-blown intrusive plaques. In general, atheromas with many macrophages and large lipid cores are the most unstable and likely to form the thromboses (clots) that block blood flow. Besides these focal lesions, many vessels also develop thicker and more rigid walls during aging through the accumulation of AGEs. Thickening in the carotid arteries that feed the brain is a predictor of stroke.

There is growing evidence that various common infections increase the risk of heart attacks and strokes. The number of antibodies to different infections correlates with blood CRP and risk of heart attack. Hepatitis C virus (HCV) alone, for example, may heighten the risk of coronary disease up to five times. When present in a donor for cardiac transplant, HCV tripled the mortality risk from accelerated coronary disease in the grafted heart. Other common infections have modest, but potentially important, effects over the long haul. *Helicobacter pylori*, which leads to ulcers and a high risk of gut cancer, also increases the risk of heart attack by about 20 percent. Besides the blood vessels, the heart valves are vulnerable to microbial attack, as in rheumatic fever.

Even oral infections play a role in vascular disease. Our gums and teeth harbor an amazing diversity of bacteria as dense biofilms, which resist scrubbing

and flossing. Tooth loss due to periodontal disease was once common: before 1900, few adults reached age 60 with any teeth. The great improvements in oral hygiene that now help us keep our bites into old age are also thought to have reduced vascular disease by lightening the load of systemic pathogens. However, it is hard to prove a particular bacteria or virus carried elsewhere in the body, particularly in low-grade infections, is a specific cause of vascular disease. The classic requirement of Koch's postulates that the condition be transmissible with the same result may not apply to heart disease and other multifactorial conditions.

Anti-inflammatory drugs, which reduce the risk of heart attacks, give further evidence of the importance of inflammation in vascular disease. Aspirin is best known for helping prevent heart attacks. Statins also demonstrate powerful anti-inflammatory capabilities, for example, lowering blood CRP. Further, since oxidative damage is a shared mechanism in the progression of vascular disease, cancer, and Alzheimer's disease, most drugs that retard vascular disease by acting on shared inflammatory processes also help prevent other chronic diseases. For example, aspirin reduces the risk of GI-tract cancer, though how it does so is still unclear. It is also uncertain how much of the oxidative damage is the 'prime mover' or a secondary effect.

Nonetheless, anti-inflammatory drugs, in reducing the activity of macrophages and other inflammatory processes, appear to lower the risk of various chronic diseases. This correlation points strongly to a connection between infections and vascular disease, one that provides modern evidence for the critical role that reducing infections and inflammation has played in the historical improvements of both childhood mortality and adult longevity rates. Animal models have supported these associations by showing that inflammation or chronic infection accelerates vascular disease, cancer, and Alzheimer's-like changes. These modern developments also support the importance of inflammation in the historical improvements in mortality across the life span.

Besides common infections, obesity is also a risk factor in vascular disease, cancer, and possibly Alzheimer's disease. Again, obesity and diabetes are characterized by increased inflammation, including higher blood CRP. Conversely, animal studies consistently show health and longevity benefits in proportion to the level of caloric restriction. In laboratory rodents, reducing caloric intake by 10 to 40 percent delays many chronic diseases, lowers free radical production and oxidative damage, and reduces blood CRP. Moreover, in mice engineered with genes that cause cancer, vascular disease, or Alzheimer's disease, caloric restriction slowed all these conditions. In these same models, obesity and diabetes accelerated many aging changes slowed by caloric restriction. Scientists have discovered that the mechanisms at work in caloric restriction include lower blood glucose and AGE formation.

The benefits of caloric restriction to lab animals, however, may be something of an artifact of their confinement. Benign lab environments do not demand the activity required in the real world for the relentless search for food and avoidance of predators. Primates in captivity tend to obesity and diabetes, making them good models of our modern couch-potato lifestyle. In humans, obesity and diabetes may be successfully treated by diet and exercise.

Would caloric restriction benefit current human aging? One widely used measure is body mass index (BMI), which adjusts body weight for height. A BMI below twenty represents extreme leanness; a BMI above thirty, obesity. A BMI below twenty may be unhealthy because of anorexia, smoking, or wasting diseases. On the other hand, a BMI above thirty increases the risk of diabetes, hypertension, and vascular disease. Most studies agree that a BMI somewhere between these extremes has little influence on mortality risk.

So would caloric restriction benefit the majority in the mid-BMI range? John O. Holloszy's study of eighteen volunteers showed that a 20 percent caloric restriction for three or more years improved risk indicators of vascular disease. Several groups are accumulating personal data on caloric restriction, and efforts are underway to find drugs that mimic caloric restriction. But one cannot forget that Jean Calment was also known for her hearty appetite for food and wine.

The recent increase in human longevity contradicts old beliefs that life spans are fixed. The scourge of heart attacks has diminished remarkably in the past thirty years, in part because of the gradual decrease in smoking. There is growing recognition that lifestyle choices strongly influence health at later ages.

Aging is very plastic, whichever genes an individual has inherited. This plasticity implies that in past centuries earlier signs of aging accompanied the shorter life spans. To Walt Whitman, even 50 was old. In a February 12, 1867, letter, he wrote, "[I] am now in good spirits . . . I don't feel a bit 'pegged out' – only getting old – most 50, you know . . . . " It is frustrating that we do not have reliable general markers for rates of individual aging that we can apply to earlier times. Graying hair certainly is not a good indicator of one's state of health or future longevity: Whitman's hair had begun to turn gray by 30, when he was in robust health. Even today, there is no consensus on which biomarkers of aging can predict the remaining life span of an individual.

Genetic vulnerabilities undoubtedly account for some of the individual differences in mortality risk – but they are not the whole story. The longevity of highly inbred laboratory rodents within the same colony, for example, differs widely. Individual rats of the same sex living in the same cage also vary widely in learning ability, reproduction, tumor incidence, and molecular damage to mitochondrial genes as they age. Within the same colony, life spans may range 50 percent about the mean, from twenty to forty months. Flies and worms also show wide individual variations in cell damage during their month-long life spans. Humans show a similar range, when calculated in proportion to life span.

From observing human twins and laboratory animals, geneticists have concluded that the heritable component of longevity is relatively modest, between 10 and 35 percent. Studies of human twins make clear the limits of genetics in aging. Menopause in identical twin pairs is typically separated by two years but can be up to twelve years apart. Chance variations in the numbers of egg cells formed in the ovary before birth may be responsible for these differences in menopause. However, after 80, the percentage of the remaining life span in twin pairs that is attributable to heredity is almost zero. At this point, the more sociable twin is likely to live longer. On average, human sociobehavioral factors also have a stronger influence than ge-

netics on an individual's ability to survive, for example, a hip fracture.

Nonetheless, the genetics and genomics of aging is a thriving, exciting field. Some centenarians carry rare genes that appear to favor longevity, including ones that mediate cholesterol metabolism. Major advances are also being made in the genetics of longevity for many different species. Mutations in metabolic pathways relevant to insulin have increased longevity in yeast cells, flies, worms, and mice. Some of these mutations, however, are less viable under more natural conditions. Dwarf mice, in particular, can live longer in certain conditions; until those conditions were discovered, they were thought to show accelerated signs of aging. What regulates the rate of oxidative damage is also mysterious – for example, why do humans live thirtyfold longer than lab mice, despite identical levels of blood glucose and body temperature?

How did aging evolve? In nature, overall mortality is vastly higher than in our current human societies. Most animal populations are dominated by young adults who do most of the reproduction needed for the perpetuation of the species. Thus natural selection is strongest against genes that impair development of young adults. According to the evolutionary theory of aging, genes that cause dysfunctions later in life are permitted to accumulate in populations because any adverse effects are delayed to minimize impact on reproduction. In effect, it is the 'schedule of reproduction' that determines potential longevity. If a gene mutation arose that caused heart attacks or strokes soon after puberty, this gene would be strongly selected against.

Having considered the recent improvements in longevity, let's look farther back to consider how humans became the longest-lived primate. In nature, life expectancy for monkeys at birth is about fifteen years; for great apes, it is about twenty. When protected in a zoo, primates live longer, but still not as long as humans live. The evolution of longevity in primates may have occurred in two stages; the first consisted of having fewer children per pregnancy. Among animals, great apes are distinctive for giving birth to one child at a time and providing the child parental care almost to puberty. Chimpanzees, for example, may continue to nurse for six years; orangutans, ten years. This extended care requires great apes to live longer than monkeys, whose maternal care ends after three years. For chimpanzees and monkeys in captivity, the duration of parental care is proportionate to life span: chimpanzees live to about forty-five years, monkeys to about thirty years.

Humans, who have further extended the care and training of their young to twenty years or more, also live proportionately longer. Our unusually slow maturation depends on a multigenerational support system not found in other primates. These social support systems were instrumental in evolving our tool-based cultures and effectiveness as hunters. Perhaps multigenerational support also favored survival to later ages and enhanced the evolutionary benefit of elders to their offspring. An edentulous fossil jaw found in the Dmanisi site in the Transcaucasus implied such social support, even of the physically infirm, existed as long as 1.7 million years ago. However, Whitman's paralytic stoke would have soon doomed him to be left behind by some migratory foragers.

Diet is another key difference between the great apes and humans. Anthropologists find that hunter-gatherers around

the world, like most of us, love to eat lots of meat. In this regard, humans differ hugely from the great apes, who are predominantly vegetarians with little regular intake of cholesterol – a substance not produced by plants. While some male chimpanzees avidly hunt and eat small animals, females do not eat meat during pregnancy or nursing. Because high-cholesterol diets accelerate vascular disease, cancer, and Alzheimer's disease in laboratory animals, Craig Stanford and I have hypothesized that the evolution of greater longevity despite this new diet required our human ancestors to evolve 'meat-adaptive genes.'[3]

One potential meat-adaptive gene is the cholesterol carrier, apolipoprotein E, which has two common genetic variants apoE3 and apoE4. The 'good' apoE3 gene lowers the risk of elevated cholesterol and Alzheimer's disease, and increases life span by several years. Carriers of the 'bad' apoE4, on the other hand, show lower frontal lobe metabolism, which could be an early stage of neurodegeneration. ApoE4 is the ancestral gene and may once have been advantageous because its proinflammatory activities may have been protective in an earlier environment. For example, people who have HCV infections but carry apoE4 have less liver damage. This double-edged impact of apoE4 can be described as an 'antagonistic pleiotropy,' bringing advantages earlier in life, but contributing to disadvantages (e.g., Alzheimer's disease) that emerge later. These genes were not actively selected against because so few people survived to advanced ages until recently.

ApoE genes also influence response to injury. ApoE3 minimizes many inflammatory responses; thus, in head trauma, for example, apoE3 carriers show less damage. Conversely, because of premature Alzheimer's changes, the punch-drunk boxer's condition (*dementia pugilistica*) is more common among apoE4 carriers. Although we do not think of head trauma as very common, soccer players who 'head' the ball frequently have higher risks of cognitive losses in middle age. This should put 'soccer parents' on alert for the future brain health of their kids who often emulate the pros in 'heading' the ball. Given the consistent evidence that apoE4 increases the risk for delayed brain dysfunctions, it is not far-fetched to think that athletes in contact sports may soon care to consider apoE4 and other genetic risk factors.

ApoE gene variants may also influence brain development. When engineered into mice, the human apoE4 decreases the complexity of neurons, relative to human apoE3. Thus, the evolution of the apoE3 variant several hundred thousand years ago may have supported greater brain development as well as longevity in our species before we left Africa.

Further increases in human longevity seem likely through improving 'gerotechnology.' New drugs are being developed to reduce obesity, possibly as mimetics of caloric restriction. If we find drugs that give normal humans longevity benefits equal to caloric restriction in rodents, then life expectancy could grow to 110 and possibly beyond 150 (which should give those political or religious organizations that elect their heads for life pause). Other drugs may broadly protect against Alzheimer's disease or arrest it at early stages. We may also achieve cancer prevention someday, despite increasing exposure to carcinogens.

3 Caleb E. Finch and Craig B. Stanford, "Meat Adaptive Genes and the Evolution of Slower Aging in Humans," *Quarterly Review of Biology* 79 (1) (March 2004): 3 – 50.

Many also look to regenerative medicine. One day, we may engineer stem cells to replace cells lost in diseased organs. Current debates about embryo stem cells will sooner or later fade as research discovers ways to reprogram an individual's own skin or marrow cells for new functions. Still there is a long way to go before neuronal stem cells can restore neurological damage from traumatic injury or Alzheimer's disease. We may also find new ways of repairing vascular damage, through engineered cells or circulating microrobots (vaso-rooters?) that are sent on patrol to repair unstable atheromas. Of course, such future regenerative medicine will be very expensive because of the huge cost of development, limiting access to those who can afford it. If the current U.S. political climate is any guide, these costly wonders will not soon be available to those with low incomes.

Beyond these anticipated biomedical advances, we need a broader view of the ecology of human aging. A starting point for modeling future population age structures is a more comprehensive account of aging that includes the load of infections and environmental inflammogens. We must also consider social dynamics such as the change in multigenerational support for the elderly that is sweeping across developed countries. The ecology of human aging must also consider the genetic changes evolved from our great-ape ancestors who were largely vegetarian and had limited multigenerational interactions. We should also expect greater demographic diversity, both of life spans and health during the later years. The fortunate who grew up with little childhood illness and maintain optimum body weight through diet and exercise could live even longer than present cohorts.

However, new risks are appearing. Obesity and diabetes are more prevalent, even among children. Maternal diabetes, moreover, heightens the risk of obesity in children. This fatty trend, if it continues to grow, could reverse historical gains. Air pollution is also on the rise. Small particles from internal combustion engines and various industrial sources inflict cardiovascular inflammatory damage; animal studies have shown that airborne pollutants activate lung macrophages and increase oxidative stress.

Also important to consider is the gap between rich and poor. Not far from the healthiest and most affluent elite in any large city are many who experience the onset of diabetes, hypertension, and other chronic diseases earlier. Epidemiologists associate the poor health of those of lower socioeconomic status with higher exposure to infections and pollution, poor diet, and limited health care. In migrant workers, TB is at least five times more prevalent than in other groups. Moreover, the spread of HIV increases the risk of TB and other infections that shorten life. Thus, poverty both dooms the disadvantaged to higher morbidity and shorter life spans, and increases the reservoirs of infectious agents that can spread to the advantaged. Sooner or later, voters must see that the potential for longer, healthier lives depends on much higher standards of health for the whole population.

Kenneth Clark

The artist grows old

"What is it to grow old?" asked Matthew Arnold, and gave a depressing answer:

> …'Tis not to have our life
> Mellowed and softened as with sunset-glow
> …'Tis not to see the world
> As from a height, with rapt prophetic eyes,
> And heart profoundly stirred.
> …It is to spend long days
> And not once feel that we were ever young;
> …Deep in our hidden heart
> Festers the dull remembrance of a change,
> But no emotion – none!

Arnold was about forty when he wrote these melancholy lines, and his experience of old age was presumably drawn from his father's friends or his fellow civil servants. He wrote in a reaction against the conventional picture of a golden old age which had been current in antiquity from Sophocles to Cicero's *de Senectute*. Everyone remembers Cephalus, Plato's dear old man at the beginning of the *Republic*: 'Old age has a great sense of peace and freedom. When the passions have lost their hold, you have escaped, as Sophocles says, not only from one mad master, but from many!' Arnold was justified in refuting this classical myth of a golden sunset. But all the same, his diagnosis is not entirely correct. 'No emotion – none!' On the contrary, elderly people feel emotion, and tend to weep more than young ones. But is it the kind of emotion that can be expressed in memorable words? A few minutes' reflection shows that it is not. The number of poets who have written memorable verse over the age of seventy is very small indeed, and to write tolerably over the age of sixty-five is exceptional. This decline in the poetic faculty in old age must be distinguished from

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the loss of inspiration that may afflict a poet at any age. But the two are obviously connected. However desirable it may be, in the conduct of life, to be free from passion, the mad masters have been responsible for at least three-quarters of the great poetry in the world. And old age, although it does not put an end to our emotions, dulls the intensity of all our responses. The romantic poets recognised that this was the cause of declining inspiration; and, as we know from Coleridge, it could happen quite early. He was only thirty-two when he wrote that long and moving letter in verse to Sarah Hutchinson from which he later extracted his 'Ode to Dejection':

> I see them all, so excellently fair,
> I see, not feel, how beautiful they are.

And he went on to define more precisely the feeling that he had lost:

> Joy is the strong voice, joy the luminous cloud.
> We in ourselves rejoice.

This is a much more accurate description of the loss that befalls us in old age than Arnold's 'no emotion – none!' Elderly people do not, and perhaps should not, rejoice in themselves. Coleridge read this letter to the Wordsworths on 21 April 1802. At that time William had not lost the faculty of joy: in fact he was at work on the 'Immortality' ode. He was so shocked by Coleridge's pessimism that he added one (or perhaps two) stanzas to the 'Ode' in order to refute it. Alas, a few years later he suffered the same fate. He continued to write poetry; he wrote on high themes, with conscientious skill. 'But emotion – none!' As most of you will know, there was an exception, and I will quote it to prove, if proof were needed, that our feelings do not die, but are buried so deeply in our memories that only some shock, some unforeseeable Open Sesame, can bring them out of bondage. In 1835 Wordsworth heard of the death of James Hogg, the Ettrick Shepherd. He went into the next room. He thought of Chatterton, that marvellous boy; he thought of his lost friends; and in less than an hour he returned with an extempore effusion:

> Nor has the rolling year twice measured
> From sign to sign its stedfast course,
> Since every mortal power of Coleridge
> Had frozen at its marvellous source.
> The rapt one, of the Godlike forehead,
> The heaven eyed creature sleeps in earth.
> And Lamb, the frolic and the gentle,
> Has vanished from his lonely hearth.

A parallel instance can be quoted from Tennyson; he had long been deprived of poetic inspiration, and had just finished writing 'Romney's Remorse', which even the most fervent Tennysonians do not defend, when, crossing to the Isle of Wight in October 1889, he was struck by an exceptionally high tide, which seemed for some reason to symbolise his recent recovery from a serious illness. Open Sesame. When he returned to Faringford he went straight to his room and in twenty minutes emerged with a poem:

> But such a tide as moving seems asleep,
> Too full for sound and foam,
> When that which drew from out the boundless deep
> Turns again home.

He knew what had happened, and knew that it wouldn't happen again. He gave instructions that 'Crossing the Bar' should always be placed last in any collection of his works. Of course, the trouble about these flashes from the depths of an elderly poet's buried life is that they cannot be sustained. To do so requires the kind of concentration that is a physical attribute. 'I can no longer

expect to be revisited by the continuous excitement under which I wrote my other book', said A. E. Housman in his preface to *Last Poems*, 'nor indeed could I well sustain it if it came.'

If, for obvious reasons, elderly writers cannot sing with the same fervour as young ones, are there not other branches of literature in which they can excel? One poet, who himself wrote movingly in old age, tried to put a case for his fellow ancients:

> And yet, though ours be failing frames,
> Gentlemen,
> So were some others' history names
> Who trod their track light-limbed and fast
> As these youth, and not alien
> From enterprise, to their long last,
> Gentlemen.
>
> Sophocles, Plato, Socrates,
> Gentlemen,
> Pythagoras, Thucydides,
> Herodotus and Homer – yea,
> Clement, Augustin, Origen,
> Burnt brightlier towards their setting day,
> Gentlemen.

It is a valiant effort, but I do not find Hardy's roll-call wholly convincing. Sophocles is the classic instance, and we must allow it. But we have no means of knowing whether the late works of Homer and Pythagoras were superior to their early ones. I am ashamed to say that I have not compared the late and early works of Clement and Origen; but I have compared St Augustine's *Confessions* with the *City of God*, and have no hesitation in saying that the *Confessions*, written twenty years earlier, is the more brightly burning of the two. I fear that after the age of seventy, or at most seventy-five, not only is the spring of lyric poetry sealed up in the depths which cannot be tapped, but the ordering, or architectonic faculty, which depends on a vigorous use of memory, with its resulting confluence of ideas, is usually in decline. The most ironic instance is that of Bernard Shaw, who believed that man would become wise if he could live to be over 100 and to prove it wrote a diffuse and unreadable play that lacks all the intellectual vigour of his maturity.

Such are the facts that must be faced if we are to consider the old age of writers and artists. But they do not by any means exhaust the subject. I believe that old, even very old, artists, have added something of immense value to the sum of human experience. There is undoubtedly what I may call, translating from the German, an old-age style, a special character common to nearly all their work; and during the rest of the lecture I shall try to discover what it is.

For some reason which is rather hard to analyse, painters and sculptors do not suffer from the same loss of creative power that afflicts writers. Indeed the very greatest artists – Michelangelo, Titian, Rembrandt, Donatello, Turner and Cézanne – seem to *us* to have produced their most impressive work in the last ten or fifteen years of fairly long lives. I say seem to *us* because this was not formerly the accepted opinion. In the nineteenth century Turner's later paintings were considered the work of a madman, and Rembrandt's *Conspiracy of Claudius Civilis* was called a grotesque masquerade. The lack of polish in Titian's later canvases was excused on the grounds that the painter was over ninety, and John Addington Symonds said of Michelangelo's Capella Paolina, 'the frigidity of old age had fallen on his imagination and faculties – one cannot help regretting that seven years . . . should have been devoted to a work so obviously indicative of decaying faculties.'

That we should now admire these late works so highly, often finding in them

some anticipation of the tastes and feelings of the present day, tells us two things about them – that they are pessimistic and that they are not concerned with the imitation of natural appearances. Contrary to the Sophoclean or Ciceronian myth, it is evident that those who have retained their creative powers into old age take a very poor view of human life, and develop as their only defence a kind of transcendental pessimism. We need only think of the eyes that look out on us from the late self-portraits of Rembrandt to realise how deeply this great lover of life became disenchanted by life. Michelangelo's head becomes, in Daniele da Volterra's portrait bust, an emblem of spiritual suffering as poignant as his own Jeremiah; and when he portrayed himself it was as the flayed skin of St Bartholomew in the *Last Judgement*. Mantegna, a name that can be added to the list of aged painters, looks in his bronze bust more indignantly pessimistic than Michelangelo, but he left in the corner of one of his last pictures, the *S. Sebastian* in the Ca' d'Oro, the emblem of his beliefs, a smoking candle, with a scroll on which are written the words *Nihil nisi divinum stabile est, coetera fumus*.

This at least suggests a belief in God, which has been denied to pessimists since the Enlightenment. 'He was without hope', said Ruskin of Turner, one can imagine how reluctantly. By the time that the author of *Modern Painters* had met his hero, Turner had grown almost completely monosyllabic in conversation, but he continued to pour his feelings about human life into the formless, ungrammatical verses of *The Fallacies of Hope*, and celebrated the salvation of mankind after the Flood with these lines (which, incidentally, are the best he ever wrote):

> The Ark stood firm on Ararat; th' returning Sun
> Exhaled earth's humid bubbles, and emulous of light,
> Reflected her lost forms, each in prismatic guise
> Hope's harbinger, ephemeral as the summer fly
> Which rises, flits, expands and dies.

Turner could express his sense of tragedy only through red clouds and a menacing vortex of sea and sky. His figures, although not insignificant, are ridiculous. But in the great period of figure painting the aged artists chose tragic themes, and treated them in such a way as to bring out their most disturbing possibilities. As far as I know the first artist to develop what I have called the old-age style was Donatello. Already in the St Anthony reliefs in Padua he had moved a long way from the Hadrianic beauty of the David or the Dionysiac rapture of the dancing putti. The scenes are vehemently dramatic, but the character of St Anthony prohibits tragedy. By the time he came to the pulpits of S. Lorenzo – he worked on them till his death at the age of eighty – he was no longer persuaded by the comforting beliefs of humanism, so beautifully expressed by the arcaded aisles beneath which the pulpits are placed. The rough, passionate, hirsute figures who surround Christ in the *Harrowing of Hell* and seem to menace him with their angular gestures, have no interest in reason and decorum. They are like a new race of Langobardi; and Christ himself, as he rises from the tomb in the next panel, is like a shipwrecked sailor, only just able to drag himself ashore. The means by which this fierce new world of the aged imagination is made visible are equally remote from the humanist tradition of decorum. The

scenes are crowded, a reckless perspective is used intermittently in order to heighten emotional effect, and the actual modelling (or rather the carving, for almost the whole surface has been cut in the bronze) is as free and expressive as the stroke of a pen in an impassioned drawing.

As with many works of the old-age style (Titian will provide another example) the S. Lorenzo reliefs are so far outside the humanist norm that an earlier generation of critics questioned their authenticity. And when they were done – in the light-footed youth of Lorenzo de' Medici – Donatello must have felt completely isolated from his contemporaries. Old artists are solitary; like all old people they are bored and irritated by the company of their fellow bipeds and yet find their isolation depressing. They are also suspicious of interference. Vasari describes how, late one night, he was sent by Pope Paul III to Michelangelo in order to obtain from him a certain drawing. Michelangelo, recognising his knock, came to the door carrying a lamp, and Vasari just had time to see that he was working on a marble pietà; but when Michelangelo noticed that his visitor was looking at it, he dropped his lantern, and they remained in the dark, till Michelangelo's servant, Urbino, a feeble candle in his hand, returned with the drawing. Then, as if to excuse himself, Michelangelo said, 'I am so old that often death tugs at my sleeve, and soon I shall fall like this lantern and my light will go out.' The reason, says Vasari, why Michelangelo *dilettassi della solitudine* was his great love of his art. But it would be a mistake to suppose that great artists escape the pains of old age through the joys of creative labour. On the contrary, all old artists who have left us a written record of their experiences, have described how the act of creation has become for them a torture. Michelangelo is, perhaps, not a good example, because he grumbled about every job he undertook; but when he wrote beneath a late drawing of the Pietà 'Dio sa che sangue costa', he was surely thinking of himself as well as of his Redeemer.

At the opposite end of the spectrum of art, Claude Monet, whose skill in rendering a visual experience has never been surpassed, created his own marvellous and unforeseeable late manner, out of infinite pain. He wrote of his water-garden canvases, 'in the night I am constantly haunted by what I am trying to realise. I rise broken with fatigue each morning. The coming of dawn gives me courage, but my anxiety returns as soon as I set foot in my studio … Painting is so difficult and torturing. Last autumn I burned six canvases along with the dead leaves in my garden.' Gone the same way as Christ's torso in the Rondanini Pietà. The aged Degas wrote in almost identical terms.

So the aged artist's pessimism extends from human life to his own creative powers. He can no longer enter sympathetically into what he sees, and he no longer has any confidence in human reason. This, as I have said, is something that we can understand more easily than could our grandfathers. They loved the art of the renaissance because it was based on naturalism, a love of physical beauty and rational order. Berenson, no less than John Addington Symonds, speaks with real hatred of Michelangelo's *Last Judgement* and (as far as I know) never even mentions the frescoes in the Capella Paolina. Yet for those who have the good fortune to see them, these two extraordinary works provide an experience as moving as anything in art, as moving as the storm scenes of *King Lear*, and as rich in layer upon layer of meaning.

Kenneth Clark on aging

As usual Michelangelo had undertaken them reluctantly. 'I cannot refuse anything to Pope Paul; but I am ill-pleased to do them and they will please nobody.' 'The art of fresco', he complained, 'was not work for old men.' But, as he said in the same year, 'one paints with the brain and not with the hands', and having once started on the work, his whole mind and spirit were engaged. The subjects selected for him were the Conversion of Saul and the Crucifixion of St Peter, episodes which had a particular theological and doctrinal importance to Paul III. The Conversion of Saul was the supreme example of grace, and in Rome of the 1540s the doctrine of justification by grace was a topic of heart-searching and earnest discussion. The most learned and devout of the Cardinals, Contarini, Morone and Pole, who had been the associates of Paul III before his elevation, were deeply impressed by the arguments of Luther, and at the centre of their discussions was Michelangelo's dearest friend, Vittoria Colonna. Thus the Conversion of Saul became for Michelangelo almost a personal experience, and he has made Saul's head an idealised self-portrait. There are many representations in art of ecstasy, of suffering, and of enlightenment; but none that equal this portrayal of the painful transition through blindness to spiritual sight. Saul lies on the ground protected by the encircling arms of one of his companions, an ordinary man. The rest of his troop breaks up in confusion. Their world seems to have exploded, as Christendom had just exploded, touched off by the doctrine of faith. The cause of this explosion, the figure of Christ, swoops down from the sky. With one hand he confirms Saul in his new belief; with the other he points to the world beyond Damascus, in which St Paul will preach His Gospel. Michelangelo has put into this drama some of his greatest formal inventions, many of which would have had a special meaning for his contemporaries. For example, the pose of Saul extended on the ground is clearly reminiscent of Raphael's Heliodorus, the would-be despoiler of the Temple. Paul III would have instantly recognised his allusion. He would have thought of the contrast between the avengers of Heliodorus and the divine apparition that redirects Saul; and would also have noticed that Michelangelo's age-old enmity with Raphael had at last been reconciled. Another example: Saul's horse, whose panic leap away from us is, so to say, the most massive fragment of the exploded world, is one of the antique horses of the Quirinal, seen from below, as Michelangelo must have seen it almost every day when he made his way to Vittoria Colonna's apartment. Almost every figure has a resonance of this kind.

But marvellous as it is, the *Conversion of Saul* is a less moving work than the *Crucifixion of St Peter*, and I may add a less complete example of the old-age style. The *Conversion* is still full of energy and the intervention of the heavenly powers gives us reason for hope. The *Crucifixion of St Peter* portrays the human lot as hopelessly and monotonously tragic. Instead of an explosion, with its possibility of a new life, the *Crucifixion of St Peter* is a wheel of life, a *rond des prisonniers*, revolving round the central figure, in and out of the frame. On the left-hand side Roman legionaries, inspired by Trajan's column, move upwards; on the right, conquered and disinherited people move downwards. Their leader, a barbarian giant with head bowed and arms folded in resignation, is one of Michelangelo's noblest inventions, a piece of visionary art that was to inspire Blake's first dated engraving. Two groups are not part of the wheel.

One represents the forces of law and order, who have condemned St Peter to death, and have been ordered to see that the sentence is being carried out. They are led by a captain, who is the pitiless embodiment of action, and closely resembles one of those ideal heads which Michelangelo had drawn twenty-five years earlier for presentation to those handsome young men who so troubled his peace of mind. Balancing these active participants is a group of four women, two of them looking at the Martyrdom, one gazing wildly into space, one looking directly at us. They are like a Sophoclean chorus. Incidentally, technicians tell us that this was the last day's work on the wet plaster of the fresco, and so the last piece of painting ever executed by Michelangelo.

Within the circle of life is an inner circle formed by St Peter's arms, and the men who are raising his cross, and it, too, has an appendage – the young man who, with mindless concentration, digs the hole in which the cross will be placed. He is innocent, the air-force pilot who releases the bomb. The saint himself is one of Michelangelo's most formidable embodiments of faith and will. Unlike Saul, who receives his painful enlightenment with a kind of gratitude, St Peter is not at all resigned to his fate, and glares at us angrily. He will break through the circle of human bondage if he can. It is no accident that Michelangelo has given his body the same form that we find in his magnificent drawings of Prometheus.

Michelangelo's frescoes in the Pauline Chapel exhibit almost every characteristic of the old-age style: its pessimism, its *saeva indignatio*, its feeling of hermetic isolation; and on the formal side its antirealism, and its accumulation of symbolic motives. In one respect, however, they do not entirely conform: in the actual technique or facture. The aged artist usually employs a less circumscribed and rougher style. In fact parts of the frescoes are painted with considerable freedom; but as a whole Michelangelo has maintained the firm outlines of every form, either because the medium seemed to demand it, or because he felt that great truths must, in Blake's words, be bounded by the wiry line of rectitude. This clarity of enunciation (even when the statements are themselves obscure) the old-age style tends to reject. To illustrate this characteristic we must turn to the only artist of equal greatness whose lifespan probably equalled Michelangelo's eighty-nine years – Titian.

The artist grows old

Nobody knows when Titian was born. Renaissance artists were in the habit of lying about their birthdays for financial reasons, and the tradition that Titian was born in 1477 is hardly credible. But when he painted the pictures in which he develops his old-age style, he was certainly over eighty. Three of them, the *Martyrdom of St Lawrence* in the Escorial, the *Crowning with Thorns* in Munich and the *Flaying of Marsyas* in Kromeresz, are reworkings of pictures that Titian had painted earlier, and it is remarkable that he chose to repeat three of the most violent and tragic subjects in the whole of his œuvre. In the later versions of all three the sense of tragedy and its universal application to human life is enhanced by purely pictorial means. The earlier *Crowning with Thorns* is a superb academic exercise, but visitors to the Louvre do not look at it for long. We have all grown too suspicious of rhetoric, and Christ's anguished movement has a chilling effect. Translated into the old-age style it is subordinated to a single passionate cry made through the medium of colour and design. The central theme is no longer the expression of Christ's head, but the cruel geometry of the soldiers' sticks. A

powerful diagonal leads up to a basket full of flames, and we suddenly realise how great a part fire and flame play in Titian's later work. It became an obsession similar to the ageing Leonardo's obsession with destruction by water; and we find it again in the Escorial *St Lawrence*, where the fire that lights up the evil faces of his executioners is, for the saint, a source of ecstasy. I am reminded of some lines by one of the rare poets who continued to write great poetry in advanced age, W. B. Yeats:

> *Saeva indignatio* and the labourer's hire
> The strength that gives our blood and
> state magnanimity of its own desire
> Everything that is not God consumed
> with intellectual fire.

Throughout his life, Titian had been the supreme master of fruitfulness. He had used his skill in the cuisine of painting to render the smooth, full pressure of flesh on skin, or pulp on rind. In the work of his old age these sensual and vegetable images are replaced by fire, flame and smoke. Titian, like Turner, did not put his thoughts into words, but even his earlier paintings leave us in no doubt that he had a powerful and well-stored mind; and in his last pictures he becomes a profound philosopher. The most complete expression of his philosophy is to be found, after considerable search, in the Moravian town of Kromeresz. It represents one of the cruellest myths of antiquity, the Flaying of Marsyas. As with the *St Lawrence*, we know that he had painted a version of the subject in his maturity, but the picture at Kromeresz is one of those left in his studio on his death, and sold by his great-nephew, Tizianello, to the Earl of Arundel. In case the story of Marsyas is not fresh in your minds, let me remind you that he was a satyr who excelled in playing the flute. The flute was out of favour on Olympus because the goddess Athena, having invented it, found that it distorted her features and threw it away. It was picked up by Marsyas, who learned to play the instrument so skilfully that he was emboldened to challenge Apollo to a musical contest. The judge was King Midas, who, as King of Phrygia, decided in favour of Marsyas; but the Muses reversed his decision, and ordered that as a punishment for his insolence, Marsyas should be flayed. It is one of those offsprings of the Greek imagination in which the forces of divine order assert themselves by an act of cruelty and we are left horrified by the price that it seemed worth paying for Olympian harmony and reason. The antique world does not seem to have questioned it, and two groups of statuary, one of them by Myron, were amongst the most frequently copied sculptures of the ancient world. The hanging Marsyas from one of these groups was, in fact, known to Michelangelo and provided a model for those late drawings of a Crucifixion which are amongst his most moving examples of the old-age style. Titian saw the myth in less simple terms. To begin with his Marsyas is hung up by the feet, like a dead animal in a butcher's shop – or like St Peter who would not be crucified in the same position as his Saviour. All the other protagonists crowd round him in a circle, giving the design that uninterrupted fullness which is a mark of the old-age style. Titian understands that this sacrifice is questionable. Midas sits beside the central figure, sunk in reverie, and behind him a satyr who has come to help his tortured sovereign, starts back with pity and astonishment. The flaying goes on as a ritual act, accompanied by the music of Apollo's cithara. He plays as if in ecstasy, and vibrations of sound seem to fill the whole canvas. We are ravished, and yet we feel

that beauty achieved at the expense of life is outrageous. This is a kind of crucifixion, a sacrifice of pure instinct to reason, and if all that reason can achieve is the hideous shedding of blood, why not leave the Dionysiac impulses to follow their own course? An answer is given by another masterpiece of the old-age style, the *Bacchae* of Euripides. The triumph of the irrational produces its own kind of catastrophe, as cruel as the triumph of reason.

This bare description of Titian's imagery suggests a wealth of visual metaphor almost as great as is to be found in Michelangelo's Pauline frescoes. But what I cannot convey in words is the extraordinary freedom with which it is painted. Every stroke of the brush is itself a metamorphosis, in its first dictionary sense, 'the action of changing in form or substance, especially by magic.' Paint is no longer a solid sticky substance, but precious, volatile and alive.

The transformation of paint into an endless series of direct messages from the painter's imagination appears in another great masterpiece of the old-age style, Rembrandt's *Conspiracy of Claudius Civilis*. As with Titian, this is the reworking of an earlier invention, only in this instance Rembrandt has painted over a fragment of the original canvas which, for some unexplained reason, had been rejected by his patrons, the City Fathers of Amsterdam. He has felt free to please himself and in the Cyclopean hero and his grotesque attendants has produced a world so bizarre that one cannot but admire the courage of the seventeenth-century connoisseurs who saved the picture from destruction. But these strange figures have the inevitability of Macbeth's porter or Hamlet's gravedigger. And the freedom with which every form is translated in the colour holds us spellbound in a way that the subject alone would not achieve. Titian's subject is horrifying, Rembrandt's grotesque, yet both arouse in me a similar emotion. For a second I feel that I have had a glimpse of some irrational and absolute truth, that could be revealed only by a great artist in his old age.

> Clouds of affection from our younger eyes
> Conceal the emptiness which age descries.
> The soul's dark cottage, battered and decayed,
> Lets in new light through chinks that time hath made.

The Rembrantesque image of Edmund Waller is irresistible. But it is only partly accurate, because the light that entrances us in these old-age pictures is not the result of exhaustion or decay, but is communicated to us by the indestructible vitality of the painter's hand. Nearly all the painters who have grown greater in old age have retained an astonishing vitality of touch. As their handling has grown freer, so have strokes of the brush developed an independent life. Cézanne, who in middle life painted with the delicacy of a water-colourist, and was almost afraid, as he said, to sully the whiteness of a canvas, ended by attacking it with heavy and passionate strokes. The increased vitality of an aged hand is hard to explain. Does it mean that a long assimilation of life has so filled the painter with a sense of natural energy that it communicates itself involuntarily through his touch? Such would seem to be the implication of the famous words of Hokusai in the preface to his *Hundred Views of Fuji*:

> All I have produced before the age of seventy is not worth taking into account. At seventy-three I learned a little about the real structure of nature, of animals, plants, trees, birds, fishes and insects.

Kenneth Clark on aging

> In consequence when I am eighty, I shall have made still more progress. At ninety I shall penetrate the mystery of things; at a hundred I shall certainly have reached a marvellous stage; and when I am a hundred and ten, everything I do, be it a dot or a line, will be alive. I beg those who live as long as I to see if I do not keep my word. Written at the age of seventy-five by me, once Hokusai, to-day Gwakio Rojin, the old man mad about drawing.

'Everything I do, be it but a dot or a line, will be alive.' Rembrandt could have said the same, and so, before his loss of manual skill, could Leonardo. There is nothing more mysterious than the power of an aged artist to give life to a blot or a scribble; it is as inexplicable as the power of a young poet to give life to a word.

Another reason for the reckless freedom of facture in the old-age style is the feeling of imminent departure. 'I haven't long to wait. I shall say what I like, how I like, and as forcefully as possible.' Maer Grafe put it more vividly in his description of Van Gogh's style: 'He paints as one whose house is beset by burglars, and pushes his furniture and everything he can lay his hands on against the door.' Van Gogh was in his thirties. Cézanne and Monet did not arrive at this state of desperation till their last years. Then they began their furious battle with time, not staining, but scarring the white canvas of eternity. But in contrast to this grandiose impatience is an ultimate feeling of resignation and total understanding. In Rembrandt's *Prodigal Son* in the Hermitage we feel that the whole of humanity has been enfolded in an act of forgiveness, beyond good and evil.

Titian, the sensualist, courtier and libertine, reveals himself in his latest pictures, the master of resignation. In the first version of his *Crowning with Thorns*, Christ's head is twisted in agony, like Laocoon; in the later version he sits motionless with downcast eyes. His last great Pietà in the Venice Academy unites both the elements of the 'old-age style'; Mary Magdalene steps forward from the platform, passionate, enraged, like an actress who can no longer endure her role, but must break out of the scene and appeal to the audience; but the Virgin and St Jerome are resigned.

Incidentally, we may suppose that this sublime work was originally in the same style as the Marsyas, and perhaps for that reason was refused by the monks of the Frari. Palma Giovane, who finished it with skill and understanding, added an inscription, saying that it had been *inchoatum*. We cannot blame him, but if it had come down to us as Titian left it, I think it would have been one of the greatest pictures in the world.

Writers on Titian have long accepted that St Jerome who kneels before the Virgin is an idealised self-portrait, and, as I have said, the Midas in the *Flaying of Marsyas* is almost identical. Twenty years earlier Michelangelo had included his idealised self-portrait, as Nicodemus, in the marble pietà now in the cathedral of Florence. It may have been the piece which Michelangelo was carving when Vasari paid his nocturnal call, and shortly afterwards Michelangelo broke it up; just as Rembrandt cut up his canvases, and Monet burned his. Later Michelangelo was persuaded to sell the pieces to a friend named Bandini, and it was restored by the sculptor Calcagni. It was really *inchoatum* and Calcagni was more ambitious and less sensitive than Palma Giovane. But fortunately he died before completing his work. The figure of Nicodemus remains unrestored, and as one looks at his noble head from different angles and in different lights one finds a whole range of human emotion be-

ginning with unutterable grief, passing through practical solicitude (specially praised by Vasari), and ending with calm and an almost beatific resignation. I do not think that Titian was inspired by this precedent, and indeed it is most unlikely that he had seen the group. Nor do I think that the desire of an aged artist to include himself in his last great work was a piece of egotism. Rather, I would suppose that he has come to think of the great tragic myths of the human imagination as almost his private property. He sees them with a mixture of heartfelt participation and detachment that requires his actual presence in the drama.

Now let me try to summarise the characteristics of the old-age style as they appear, with remarkable consistency, in the work of the greatest painters and sculptors. A sense of isolation, a feeling of holy rage, developing into what I have called transcendental pessimism; a mistrust of reason, a belief in instinct. And in a few rare instances the old-age myth of classical antiquity – the feeling that the crimes and follies of mankind must be accepted with resignation. All this is revealed by the imagery of old men's pictures, and to some extent by the treatment. If we consider old-age art from a more narrowly stylistic point of view, we find a retreat from realism, an impatience with established technique and a craving for complete unity of treatment, as if the picture were an organism in which every member shared in the life of the whole.

I have mentioned a few of the artists in whose late work these characteristics can be found. I could have extended it to almost every great painter who has lived beyond the age of 65 or 70. Indeed I can think of only one exception, Piero della Francesca; and there a physical cause, cataract or partial blindness, prevented him in his old age from painting at all.

Turning back to writers of equal stature, one cannot but be struck by the difference between the two arts.

One of the finest critical essays in English begins with the words, 'It is a mistake of much popular criticism to regard poetry, music and painting – all the various products of art – as but translations into different languages of one and the same fixed quantity of imaginative thought, supplemented by certain technical qualities.' Pater's warning is always in my mind. Nevertheless the elderly great do seem to have a good deal in common, and it is worth speculating on the reasons why they can express themselves so much more movingly in painting and sculpture than in poetry. Perhaps a clue is given by Coleridge's words, 'we in ourselves rejoice' together with the word vitality. The painter is dealing with something outside himself, and is positively drawing strength from what he sees. The act of painting is a physical act, and retains some element of physical satisfaction. No writer enjoys the movement of his pen, still less the click of his typewriter. But in the actual laying on of a touch of colour, or in the stroke of a mallet on a chisel, there is a moment of self-forgetfulness. Harassed public servants – presidents, statesmen and generals – take up painting; they do not (with the exception of Lord Wavell) write poetry. It may seem ridiculous to compare the therapeutic activities of these amateurs to the struggles of Titian or Rembrandt; but I think that they do indicate a fundamental difference between the two arts. A visual experience is vitalising. Although it may almost immediately become a spiritual experience (with all the pain which that involves), it provides a kind of nourishment. Whereas to write great poetry, to draw continuously on one's inner life, is not merely exhausting, it is to keep alight a consum-

ing fire. What in old age feeds this fire? Memories of past emotions; only very occasionally fresh experiences which, if they are strong enough to generate poetry, cannot as Housman said, be endured for any length of time.

Before trying to discover instances of the old-age style in literature and music, I ought perhaps to consider the question of what, in a creative artist, is meant by 'old.' Painters and sculptors tend to live much longer than writers or musicians, and their work shows no sign of old age till their last years. Mr Henry Moore is seventy-three, but neither in himself nor in his carving is there the slightest sign of old age. Matisse became bedridden, but his art remained as fresh as a daisy. Conversely, Beethoven was under fifty when he entered what critics agree to call his last period, and the quartets, written when he was fifty-five, are classic examples of the old-age style in their freedom from established forms and their mixture of remoteness and urgent personal appeal. Like the last works of Michelangelo and Titian, they seem to go beyond our reach, and yet there is an ultimate reconciliation. One should, I suppose, add that Beethoven's isolation may have been increased by his deafness.

But there are other examples of an old-age style in a great artist under fifty for which there is no such simple explanation. How do we explain Shakespeare's last four plays? Critics tend to write of them as if they were the work of an old man, although Shakespeare was in his middle forties when he wrote them. *Pericles*, *Cymbeline* and *The Winter's Tale* do indeed show some of the negative characteristics of the old-age style – the impatience, the recklessness and the bitterness. But these seem to me symptoms of exhaustion rather than of a new direction. Lytton Strachey's notorious judgement that 'Shakespeare in his last years was half enchanted by visions of beauty and half bored to death', although it has been rejected with horror by most scholars of Shakespeare, seems to me substantially true. No man has ever burnt himself up more gloriously. But *The Tempest* does seem to show some characteristics that only an artist who has lived his life could give. Far more than the earlier plays it creates a private world of the imagination. Shakespeare, who had in the past written so immediately for his actors and his audiences, now seems to be writing only for himself. And Prospero's last speech could surely not have been written by a young man, even the young Shakespeare.

I have hesitated to quote the example of Shakespeare in *The Artist Grows Old*; and I would definitely exclude Racine, for, in spite of the enormous change that took place in his life during the twelve years' silence between *Phèdre* and *Esther*, and the considerable difference of style of his last two plays, they do not seem to reflect the liberation of old age. But I have no such hesitation in including a third – I might say *the* third – great European dramatist: Ibsen. His last plays, from *The Master Builder* to *When We Dead Awaken*, are perhaps the most complete illustration in literature of the characteristics of the old-age style as we have seen them more consistently revealed in the visual arts.

First, isolation. In the 1890s Ibsen was the most famous writer in Europe, but after his return to Norway he lived in a solitude of his own making. He was as lonely as Michelangelo, and if anything rather grumpier. Then the flight from realism. Viewed as a realistic drama *The Master Builder* is unconvincing, and in *When We Dead Awaken* all pretence of naturalism is abandoned. Both plays are still based on marvellous and embarrassing psychological insights; but in form

they are allegories of guilt and redemption. They are full, perhaps too full, of symbols; and as usual with the old-age style, these symbols can be interpreted differently and leave us with an uneasy feeling that we can decipher only half the message. They are intensely personal; in fact it can be argued that the hero-villain of each play is Ibsen himself, the man who sacrificed life to art and came to believe that life is the more important. Michelangelo, when asked to design the reverse side of his portrait medal by Leone Leoni, chose as his emblem an old blind pilgrim, led by a mongrel dog, trotting along confidently with tail erect. Ibsen would have agreed. But solitude and physical inaction do not imply a lack of vitality, and during the years in which his last play was being written, Ibsen was constantly falling in love with young girls. Hilde Wangel and Irene were real experiences and few things gave him more satisfaction than to read about the aged Goethe's love affair with Mariana von Wilmer. Only instead of his young ladies inspiring him to write poems to the rising moon, as Goethe did, whether effectively or not it is hard to say, Ibsen saw them as a new kind of Eumenides, playing on his sense of guilt and driving him on to self-destruction.

On the name of Goethe, I must confess that the greatest and most prolific of septuagenarian poets does not illustrate the characteristics of an old-age style, which seems to me so evident in the work of painters and sculptors. It is true that the second part of *Faust* ends with symbolic utterances as mysterious as the last speeches in *When We Dead Awaken*. But Goethe's respect for conformity (what is usually referred to as his wisdom) led to a tone of vague optimism, which his fellow ancients have not usually shared. It is remarkable that Thomas Hardy does not include Goethe in his list. Perhaps he could not bring himself to say (and we sympathise with him) that the second part of *Faust* burned brightlier than the first. The numerous lyrics that Goethe wrote in his last years at the drop of a hat may be better than Longfellow. I cannot tell. What is certain is that they might have been written by any middle-aged poet conscious of his powers, and of his responsibilities to a rather conventional notion of poetry.

In the present century, as opposed to the last, poets have tended to gain in power as they grew older, and a number of them have written movingly in the old-age style – Yeats, Rilke, Thomas Hardy himself. Yeats and Rilke used the freedom of address and the almost impenetrable symbolism of aged painters. Thomas Hardy in such a poem as *Aftermath* spoke more simply, but with a feeling of isolation and imminent departure. But with no disrespect to these fine poets, I think one must allow that they are in a different category to Michelangelo, Titian and Rembrandt. Can we name an aged poet of this stature? Although he arouses no enthusiasm among modern critics, I hope I may be allowed to pronounce the name of Milton. *Samson Agonistes* is, so to say, a double distilled example of old-age writing, because it is undisguisedly modelled on the *Oedipus at Colonus* which Sophocles is supposed to have written after the age of 87. Like the other examples I have quoted, it is deeply personal. Milton was himself blind; his hopes had been shattered, his cause betrayed, and although his relations with the opposite sex were certainly not as simple as those of Samson and Delilah, he felt that his love of women was in some way connected with his failure. *Samson Agonistes* is almost as autobiographical as the last plays by Ibsen. But it differs from them

in that Samson discovers a humility that Ibsen's guilt-ridden characters cannot achieve, and so unlike the questionable victories of Solness and Rubeck, he ends his career with an apotheosis which is also the highest victory of old age.

Samson Agonistes, like *Paradise Regained*, also ends on a note of resignation; and in its actual diction it introduces one more aspect of the old-age style – a stoic austerity which denies any appeal to the emotions made through the sensuous quality of the medium. Michelangelo, Titian, Rembrandt, Donatello, Cézanne, all continued to use their media with an added sense of its material possibilities. But at least two great artists of the seventeenth century voluntarily rejected that charm of colour, light and joy in the use of paint which captivates us in their early work. These are Poussin and Claude. Poussin had equalled the great Venetians in his richness of colour and had sought for subjects that might allow him such sensuous delights. But by the time he had come to paint his second series of the *Seven Sacraments*, he had come to feel, as did Milton in *Paradise Regained*, that to display any pleasure in sensation would be to deprive the subject of its high seriousness. Poussin by the intellectual power of his invention seems to me to have justified his puritanical renunciation. But a poem, which has to hold our attention and keep our faculties warm for a longer time than a picture, may suffer more severely from the exclusion of ornament and graphic invention. The old-age style of Claude was less calculated. In his latest landscapes he did not deliberately exclude the enchantments of light and distance; but he retreated into a remote world of his own creation, where colour is subdued to a near monochrome and events take place in a sort of trance. This gentle, dreamlike departure from reality is very different from the fiery pessimism of Michelangelo and Titian, and is perhaps the least painful expression of growing old.

The fact is that Arnold was not far wrong. The outstanding poet of our own day, Mr T. S. Eliot, has amplified his statement with more subtlety and even greater bitterness:

> Let me disclose the gifts reserved for age
> To set a crown upon your lifetime's effort.
> First, the cold friction of expiring sense
> Without enchantment, offering no promise
> But bitter tastelessness of shadow fruit
> As body and soul begin to fall asunder.
> Second, the conscious impotence of rage
> At human folly, and the laceration
> Of laughter at what ceases to amuse.
> And last, the rending pain of re-enactment
> Of all that you have done, and been; the shame
> Of motives late revealed, and the awareness
> Of things ill done and done to others' harm
> Which once you took for exercise of virtue.

Any elderly person can vouch for the accuracy of those lines. They record the common lot of *homo sapiens*. And the miraculous fact, which I have tried to describe in this lecture, is that many artists and some writers have, with infinite pain, created great works of art out of these miserable conditions. Their rage at human folly has not been impotent, their re-enactment of things done has been a means of re-creating them as part of a life-preserving myth, and they have arrested the moment when the body and soul fall asunder, caught enough of the body to make the moment comprehensible, and seen how its disintegration reveals the soul.

Jagadeesh Gokhale & Kent Smetters

Measuring Social Security's financial outlook within an aging society

The U.S. Social Security program provides an important 'first pillar' of retirement income.[1] Policymakers and the media, therefore, pay considerable attention to the financial viability of the program. Each year, the Social Security trustees release a report that summarizes the financial position of the Social Security program. Among other measures, the report draws attention to the program's 'crossover date' (the year the program's benefit outlays will begin exceeding its tax receipts), the date of 'trust fund exhaustion,' and the present value of the program's financial shortfalls over the next seventy-five years.[2]

These measures have two problems. First, they create a misleading impression of the program's financial outlook. Second, they are biased against potential reforms that could improve the program's finances.

Fortunately, the trustees have recently adopted new accounting measures that deal with both problems. These measures reveal an $11.1 trillion present-value shortfall, which equals about 3.5 percent of the present value of all future taxable payrolls. Unfortunately, because these new measures are buried in the trustees' report, they have received only scant consideration from policymakers and

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2 'Present value' is a number that summarizes a sequence of financial shortfalls by applying

the media. The newer measures should receive greater attention. Indeed, were these new measures taken more seriously, reforming Social Security and Medicare could reemerge as the top policy priority that it deserves to be.

Social Security covers almost the entire U.S. population, providing participants and their spouses with retirement, disability, and other benefits during different stages of life. Social Security is currently the largest single outlay in the U.S. federal budget; many consider it one of the most successful programs in U.S. history. Although Social Security, on average, replaces only about 40 percent of a worker's annual earnings before retirement, it provides an important 'first pillar' of retirement income. Indeed, for poorer retirees, Social Security replaces 90 percent or more of their previous earnings. Social Security is often credited with reducing poverty among the elderly in the United States.[3]

Participation in Social Security is mandatory for most occupations.[4] Social Security is financed by a 12.4 percent payroll tax on covered earnings up to a limit. This limit is currently $94,200, but it increases each year with the economy-wide average wage. Employer and employee split this tax evenly. Participants become 'fully insured' after they have worked in a covered job for forty calendar quarters and earned more than a predetermined wage. Fully insured participants, however, do not acquire a contractual right to specific amounts of benefits.[5] Instead, they earn a noncontractual right to benefits that are governed by the laws in effect when they become eligible to receive benefits. These laws as well as the benefit formula are subject to change by Congress.

Social Security's benefit formula is similar to a private-sector defined-benefit plan's, where a specific formula applied to a retiree's wage history determines his or her benefits.[6] In contrast, voluntary, tax-favored defined-contribution retirement plans – 401(k), 403(b), Keogh, and others – generate retirement income based directly on a person's pre-

a discount factor to future shortfalls and taking their sum. The further in the future that a shortfall occurs, the larger the discount factor applied. This is done to place dollars accruing at different points in time on an equal valuation scale. Discount factors are usually of the form [1/(1+r)]t. Here, r is an annual interest rate that signifies the 'time value of money.' If investing $1 earns interest of 5 cents per year, the value of $1 available today is the same as $1.05 available next year. Similarly, the value of $1 available next year equals $[1/(1.05)] today, which is less than $1: in other words, this amount plus accrued interest will amount to $1 next year. The discount factor applied to dollars accruing after t years is, therefore, [1/(1.05)]t (where r=0.05). The 'present value' of all future financial shortfalls is the sum of those shortfalls taken after each is discounted according to the number of years in the future that it occurs. When calculating the present value of projected shortfalls for government programs, the appropriate interest rate to use is the 'government's interest rate' – the market rate that it must pay lenders to obtain funds. For Social Security, the annual inflation-adjusted interest rate used in recent years by the program's trustees equals 3.1 percent.

3 Gary Engelhardt and Jonathan Gruber, "Social Security and the Evolution of Elderly Poverty," National Bureau of Economic Research, Working Paper 10466, May 2004.

4 A notable exception includes state workers who are covered by state pension programs.

5 See the U.S. Supreme Court case, *Nestor v. Flemming*, 363 U.S. 603 (1960).

6 One major difference is that Social Security bases a retiree's benefit on many more years of earnings throughout his or her lifetime than the number of years most private-sector defined-benefit plans use to determine benefits.

vious contributions and subsequent market investment returns.

Whereas previous contributions 'fully fund' withdrawals from voluntary tax-favored retirement plans, Social Security operated mostly on a 'pay-as-you-go' basis between the 1940s and the early 1980s: payroll tax revenue collected each year was paid out almost immediately as benefits rather than saved, thereby producing rates of return on previous contributions in excess of the risk-adjusted rates of return that those contributions could have earned in financial markets.[7] For those who retired shortly after Social Security began, this financing structure meant that they received more benefits from Social Security in present value than they had paid in payroll taxes. These windfalls occurred each time that Congress expanded Social Security's coverage and benefits, after 1950 until well into the 1970s.[8]

Unfortunately, the windfalls awarded to prior generations of retirees do not come for free: future generations must pay for them by receiving lower rates of return on their payroll taxes compared to the rates they could have earned if they had invested their contributions in government bonds instead. In fact, *all* future generations are worse off.[9]

7 Dean R. Liemer, "Cohort-Specific Measures of Lifetime Net Social Security Transfers," Social Security Administration, Office of Research and Statistics, Working Paper No. 59, February 1994.

8 John Geanakoplos, Olivia S. Mitchell, and Stephen P. Zeldes, "Would a Privatized Social Security System Really Pay a Higher Rate of Return?" in *Framing the Social Security Debate: Values, Politics and Economics*, ed. R. Douglas Arnold, Michael J. Graetz, and Alicia H. Munnell (Washington, D.C.: National Academy of Social Insurance, 1998), 137 – 157.

9 F. Breyer, "On the Intergenerational Pareto Efficiency of Pay-As-You-Go Financed Pension Systems," *Journal of Institutional and Theoretical Economics* 145 (1989): 643 – 658. Assuming that the growth rate of the economy is less than the interest rate, the so-called dynamic efficiency condition, the present value of the gains and losses across all past, current, and future generations is exactly zero.

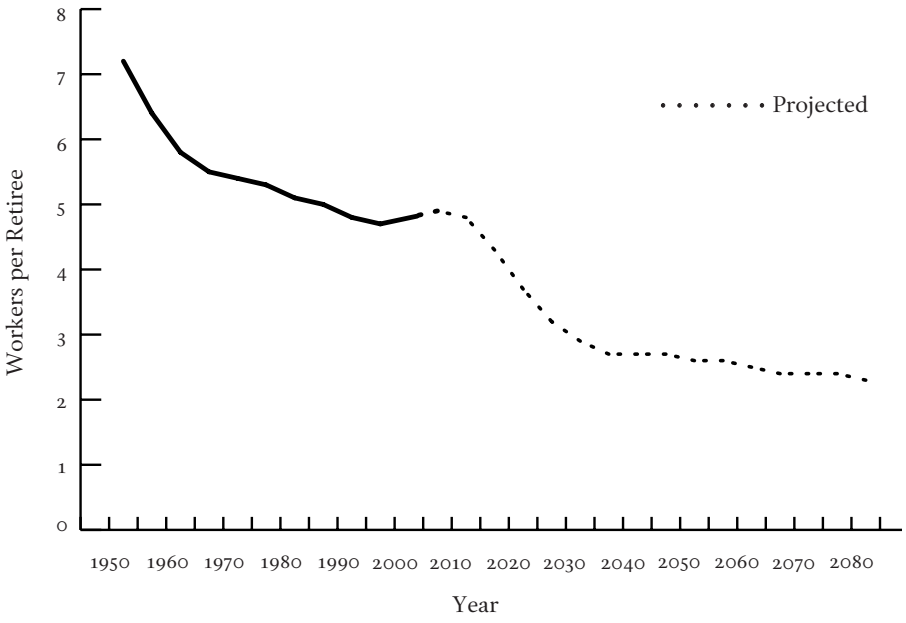
Measuring Social Security's financial outlook

During the early 1980s, the independent Office of the Actuary at the Social Security Administration projected that revenues would fall short of benefit outlays during the early part of the twenty-first century, largely because of the baby boom generation's retirement. Although this generation enlarged the labor force considerably (in part through the greater participation of women in the workforce) and made significant contributions over the past several decades, its members will soon retire, substantially reducing the number of workers available to finance their Social Security and Medicare benefits through payroll and other taxes. As Figure 1 shows, today there are almost five people of working age – between ages 20 and 64 – for each retiree age 65 and older. By 2030, the number of people of working age per retiree will decline to less than three; by 2080, the ratio will fall to about two.

Recognizing these future demographic changes, Congress amended the Social Security Act in 1983 in an attempt to increase the system's cash flow over the next seventy-five years. Those amendments approved payroll tax hikes, subjected the Social Security benefits of those with other income sources to income taxation, and scheduled a gradual increase in the full retirement age from 65 to 67 beginning in 2003. Since 1983, these changes have generated surpluses in the Social Security trust fund, which currently holds $1.7 trillion in Treasury IOUs.

Despite these reforms, Social Security remains mostly pay-as-you-go in its

Figure 1
Workers (Ages 20 – 64) Per Retiree (Age 65 and older)



Source: Social Security Administration.

financing structure. And though $1.7 trillion sounds like a lot, it is insufficient to pay current retirees their scheduled benefits for more than three years. Had the 1983 amendments 'fully funded' the Social Security system instead, the trust fund would hold about $13.7 trillion today. Contributions by past and current generations would have been enough to cover their own benefits, and future generations would not have to shoulder any of the burden.

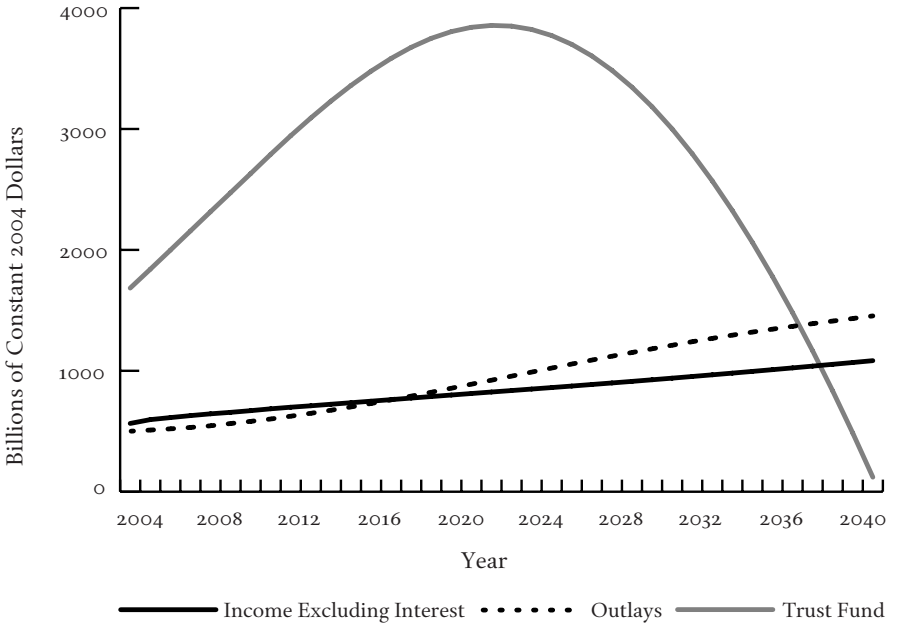
At the time, many thought that the 1983 amendments had resolved Social Security's financial shortfalls for the subsequent seventy-five years. But soon thereafter projected seventy-five-year imbalances began appearing again.

As shown in Figure 2, payroll tax surpluses will probably continue until 2017 – the so-called crossover date – after which projected benefits will exceed revenues. The trust fund will continue increasing because of interest income accruals through 2027, after which it is projected to decline gradually and be exhausted by 2041. The Social Security trustees estimate that the *present value* of benefits, scheduled under current law, over the next seventy-five years will exceed by $4 trillion the *present value* of its payroll tax revenues plus the *current* value of the trust fund's Treasury securities.

In other words, only if the government immediately deposited an additional $4 trillion into the trust fund, by increasing taxes or reducing spending, would it be able to pay current-law benefits over the next seventy-five years. An infusion of money into the trust fund would also increase public and national saving if it were not reborrowed and spent on other government programs – a topic of recent debate.[10] Were the new monies spent

10 Peter Diamond, "Social Security, the Government Budget and National Savings," un-

Figure 2
Social Security's Revenues and Outlays



Source: Social Security Administration.

entirely on other programs, the government's overall capacity to pay future Social Security benefits would not improve even though the value of Treasury securities in the trust fund would increase.

The 'moving window' phenomenon partially explains why the seventy-five-year imbalances reappeared after 1983. In 1983, the projected seventy-five-year window ended in 2057; today it ends in 2079. Simply moving the seventy-five-year window to cover the years 2058 through 2079 – when cash-flow shortfalls are projected to accrue – created most of the recent $4 trillion imbalance. In other words, because the measures of the system's solvency used in 1983 were based on a limited time horizon, policymakers back then failed to include the additional adjustments to taxes and benefits necessary to achieve a sustainable Social Security system. Unfortunately, their failure means that we must make even larger adjustments in the future.

The same limited perspective on the system's financial condition is again hampering reform efforts today. Indeed, the problem of a 'moving window' implies that reforms that make the system solvent over the next seventy-five years will just falter again as the window moves forward into the future. As shown in the first panel in Table 1, the 2005 *Social Security Trustees Report* projects an *additional* $7.1 trillion imbalance in present value (as of 2004) *after* the year 2079.

published mimeo, MIT, March 24, 2003; Sita Nataraj and John Shoven, "Has the Unified Budget Undermined the Federal Government Trust Funds?" mimeo, Stanford University, 2004; and Kent Smetters, "Is the Social Security Trust Fund a Store of Value?" *American Economic Review Papers and Proceedings* 94 (2) (May 2004): 176 – 181.

Table 1
Unfunded OASDI[a] Obligations [present values as of January 1, 2005; dollar amounts in trillions]

Unfunded obligations through 2079[b]	$4.0
Unfunded obligations after 2079[c]	7.1
Equals total unfunded obligations (open-group obligations)	**11.1**
Unfunded obligations attributable to past and current participants (closed-group obligations)[d]	12.0
Unfunded obligations attributable to future participants[e]	-0.9
Equals total unfunded obligations (open-group obligations)	**11.1**

a 'Old age, survivors, and disability insurance' is the official name of Social Security.
b Present value of future costs less future taxes through 2079, reduced by the amount of trust fund assets at the beginning of 2005.
c Present value of future costs less future taxes after 2079.
d This concept is also referred to as the closed-group unfunded obligation. It is equal to the present value of benefits paid to current and past generations less the taxes and the value of the trust fund.
e People age 14 and below in 2005.

Source: 2005 *Social Security Trustees Report*, Table IV.B6 and IV.B7.

Adding the $7.1 trillion imbalance after the year 2079 to the $4 trillion imbalance projected through 2079 produces a present-value imbalance of $11.1 trillion, which is equal to about 3.5 percent of the present value of all future taxable payroll revenue.[11] Barring any reform this year, this $11.1 trillion imbalance will only grow with interest, just like any regular 'debt rollover.' Indeed, according to the trustees, this imbalance will increase by about $600 billion over just a single year if we do not take legislative action.[12] To be sure, the economy will also expand over time and so this $600 billion figure only tells part of the story. Still, even relative to the present value of all future payrolls, Social Security's problems will grow worse over time. And when added to Medicare's shortfalls – about seven times larger than Social Security's[13] – the imbalance grows by almost 2 percent of the present value of all future covered payroll for every five years that we delay fundamental reforms. In other words, for every five years that we do not enact policy reform, we would have to permanently increase taxes by an additional 2 percent of taxable payrolls, or reduce outlays by the same amount. The cost of delaying Social Security reforms is, therefore, enormous.

Whereas *solvency* typically refers to the government's ability to pay benefits over the next seventy-five years, *sustainability* refers to its ability to pay benefits into

11 Social Security's projected shortfalls could also be represented as a share of the present value of future projected GDP. But we think that representation is quite misleading since the government taxes only between 50 and 60 percent of GDP (the payroll tax applies to an even smaller portion) and will likely continue to do so in the future. An even more misleading statistic is to state only the seventy-five-year shortfall in present value relative to GDP.

12 Social Security Trustees, 2005 *Social Security Trustees Report*, Section IV.B.5.a.

13 Jagadeesh Gokhale and Kent Smetters, "Fiscal and Generational Imbalances: An Update," in James M. Poterba, ed., *Tax Policy and the Economy*, vol. 20 (Cambridge, Mass.: MIT Press, forthcoming 2006).

the indefinite future. A Social Security reform that achieves solvency over a limited horizon, but not sustainability, will soon fail to achieve even solvency as the window moves forward to include future years. However, a sustainable reform will also be solvent. Under Social Security's current projections, achieving sustainability is harder than achieving solvency: an additional $7.1 trillion in tax and benefit adjustments is necessary to address the shortfalls accruing after 2079.

The government routinely uses an ad-hoc measure of sustainability that asks whether the system satisfies two conditions.[14] First, is the Social Security system *solvent*? That is, can Social Security afford to pay current-law benefits over the next seventy-five years with current-law tax revenues over the next seventy-five years plus the current trust fund value? Second, is the trust fund projected to be increasing in size toward the end of the seventy-five-year window? Social Security is deemed 'sustainable' if both conditions are met.

This ad-hoc measure of sustainability assumes that the trust fund will continue to increase in size *after* the seventy-fifth year. This assumption is often invalid. For example, the recent reform plan by Peter Diamond and Peter Orszag[15] appears sustainable under this ad-hoc approach. However, under this plan, we must continue to raise payroll tax rates after the seventy-fifth year in order to pay present-law projected benefits and prevent the trust fund from disappearing. Without raising taxes, we would eventually exhaust the trust fund.[16]

Conversely, a reform might not appear sustainable under the ad-hoc measure even though it fully eliminates the current $11.1 trillion present-value imbalance. For example, Model 2 of the President's Commission to Strengthen Social Security[17] is not projected to achieve solvency over the first seventy-five years – the first condition for sustainability under the ad-hoc measure – without general revenue transfers from the U.S. Treasury. However, if we maintained its reform measures beyond the seventy-fifth year, Model 2 would more than eliminate the existing $11.1 trillion imbalance even without general revenue transfers. That is, Model 2's cost savings after the seventy-fifth year would more than offset, in present value, the shortfalls projected during the first seventy-five years.

The traditional ad-hoc measure of sustainability, therefore, has serious shortcomings.[18] But the most important weakness of this and other traditional measures of Social Security's finances is

14 See, for example, President's Commission to Strengthen Social Security, *Strengthening Social Security and Creating Personal Wealth for All Americans* (Washington, D.C.: President's Commission to Strengthen Social Security, 2001), 68 – 71; Council of Economic Advisors, *2004 Economic Report of the President* (Washington, D.C.: Council of Economic Advisors, 2004), 139; Social Security Trustees, *2004 Social Security Trustees Report* (Washington, D.C.: Social Security Trustees, 2004), Section IV.B.5.a.

15 Peter Diamond and Peter Orszag, *Saving Social Security: A Balanced Approach* (Washington, D.C.: Brookings Institution Press, 2004).

16 Ibid. Diamond and Orszag, however, advocate continuing to increase payroll tax rates after the seventy-fifth year.

17 President's Commission to Strengthen Social Security, *Strengthening Social Security*, 68 – 71.

18 Additional criticisms can be found in Howell Jackson, "Accounting for Social Security and Its Reform," *Harvard Journal on Legislation* 41 (1) (Winter 2004): 59 – 225.

that they introduce a bias in policymaking. In particular, reforms that could reduce Social Security's $11.1 trillion imbalance – and improve Social Security's sustainability – often worsen each of the more traditional measures, including the trust fund exhaustion date, the crossover date, and the seventy-five-year imbalance.

Consider the 'actuarially fair carve out.' This reform is very similar to the plan President Bush is now advocating, which allows participants to 'carve out' some of their payroll taxes and deposit them into a personal account that would later augment their traditional benefit, much like 401(k)s and IRAs.[19] Since these participants would be contributing less to the traditional system, their traditional benefit would also be reduced by an 'actuarially fair' amount equal to one dollar in present value for each dollar carved out.

This reform would have no impact on the $11.1 trillion imbalance. Each dollar that the government loses in payroll contributions would be fully offset by a dollar that the government saves in present value of future benefit payments. Furthermore, unless capital markets responded in an uninformed manner (discussed in more detail later), this reform would not affect wages, interest rates, or gross domestic product (GDP) in any year. Neither would this reform change the net lifetime resources available to any household born at any time. In economic terms, this reform would be fully neutral.

Still, under this reform, all three measures traditionally used to judge Social Security's viability – the trust fund exhaustion date, the crossover date when costs exceed income, and the seventy-five-year imbalance – would worsen. We would exhaust the trust fund earlier because of the short-run decline in payroll contributions; similarly, the crossover date would occur sooner. The seventy-five-year imbalance would also appear larger because much of the lost tax revenue would show up inside the seventy-five-year window while a larger portion of the future reduction in benefits would fall beyond the seventy-five-year window.

Now let's modify the example to consider a 'carve out with a haircut.' Under this approach, we would reduce a participant's traditional Social Security benefit by more than a dollar, say $1.10, for every dollar carved out and deposited into a personal account. A worker might be willing to take this 'haircut' on future benefits in order to obtain greater ownership and control over his or her retirement resources.

In this case, we would *reduce* the $11.1 trillion imbalance since the government saves more on benefit payments in present value than it loses in contributions. Still, if policymakers focused only on the traditional measures of Social Security's finances to judge this reform plan,[20] they might reject it even though it would improve Social Security's financial outlook. The improvement in Social Security's financial outlook – as reflected by its reduced present value of unfunded obli-

19 Technically, President Bush's plan is not quite actuarially fair because his benefit-offset rate does not adjust for preretirement mortality; it is also tied to expected Treasury yields instead of actual yields. The first issue is of second-order importance as preretirement mortality will be low in the future. The second issue is easily correctable.

20 Technically, whether the seventy-five-year imbalance would get better or worse would depend on the timing of the haircut. In any case, the seventy-five-year imbalance measure would fail to capture many of the benefit reductions after the seventy-fifth year.

gations – should exert salutary effects on the economy immediately. In particular, private agents' economic decisions would no longer be distorted by the expectation of higher future costs of resolving Social Security's financial problems.

Thus, the traditional measures are not very revealing of the program's true financial status, and worse, they are biased against reforms that could reduce Social Security's $11.1 trillion imbalance. Unfortunately, these measures often influence the design of reform plans. For example, in Model 2 of the President's 2001 Commission to Strengthen Social Security, participants are allowed to carve out 4 percent of payroll, up to a maximum of $1,000 per year (wage indexed over time).[21] The Commission imposed the $1,000 ceiling to prevent the Social Security system from 'losing' too much money over the projected seventy-five-year horizon. Restricted to that horizon, the Commission did not take into account the large cost savings that would begin accruing *after* the seventy-fifth year. If participants were allowed to make even higher contributions to their personal accounts, Model 2 would more easily eliminate the entire $11.1 trillion imbalance.

Beginning with the 2003 *Social Security Trustees Report* and the 2004 *Medicare Report*, two new measures have emerged that provide greater insight into the financial status of both programs. The Social Security Advisory Board's Technical Panel on Assumptions and Methods, which is composed of leading economists and actuaries outside of the Social Security Administration, have also recently endorsed these new measures.[22] Indeed, these measures correspond to the way that economists have thought about Social Security's finances for many years.[23]

The first measure is sometimes called the 'open-group unfunded obligation.' It is the sum of benefits that all *past, present, and future generations*, or 'groups,' have received (and are projected to receive) in present value less the amount of taxes they have paid (and are projected to pay). We can also calculate it as the present value of all projected Social Security benefits minus the present value of all projected payroll taxes and the current value of the trust fund.

The open-group unfunded obligation reveals the extent to which the current Social Security program is unsustainable. That is, it shows Social Security's financial imbalance arising from all generations. Table 1 shows that based on

21 Wage indexing the $1,000 contribution limit means that the limit increases with annual growth in average, economy-wide wages.

22 See "The 2003 Technical Panel on Assumptions and Methods Report" <http://www.ssab.gov/NEW/documents/2003TechnicalPanelRept.pdf>.

23 See, for example, Alan Auerbach, "The U.S. Fiscal Problem: Where We Are, How We Got Here, and Where We Are Going," *National Bureau of Economic Research Macroeconomics Annual*, ed. Stanley Fischer and Julio Rotemberg (Cambridge, Mass.: National Bureau of Economic Research, 1994); Jagadeesh Gokhale and Kent Smetters, *Fiscal and Generational Imbalances: New Budget Measures for New Budget Priorities* (Washington, D.C.: American Enterprise Institute Press, 2003); Alan Auerbach, William Gale, and Peter Orszag, "Sources of the Long-Term Fiscal Gap," *Tax Notes* 103 (2004): 1049 – 1059; Edward Gramlich, "Rules for Assessing Social Security Reform," Remarks to the Retirement Research Consortium Annual Conference, August 12, 2004; Andrew Rettenmaier and Thomas Saving, "The 2004 Medicare and Social Security Trustees Reports," National Center for Policy Analysis, Policy Report No. 266, June 2004.

calculations provided by the independent Office of the Actuary at the Social Security Administration, the trustees estimate the open-group obligations at $11.1 trillion in present value. In other words, in order to make Social Security sustainable, we must reduce scheduled benefits and/or increase taxes so that the sum of cost savings and new revenues total $11.1 trillion in present value.

The second measure is sometimes called the 'closed-group unfunded obligation.' It shows the amount of Social Security's $11.1 trillion imbalance arising from providing benefits to *past and present* generations (those age 15 and older up to those who are deceased as of 2005) in excess of their payroll taxes in present value. Unlike the open-group obligation, this calculation is 'closed' to, or does not include, future generations.

Based on calculations provided again by the Office of the Actuary, the trustees estimate that past and current generations will receive about $12 trillion more in benefits in present value than they will pay in taxes (see Table 1). In contrast, future generations (those age 14 and younger in 2005 as well as the unborn) are projected to receive $0.9 trillion *less* in benefits than they will pay in taxes (see Table 1). The 'overpayment' by future generations, though, is still not enough to pay for the 'overhang' of $12 trillion they are projected to inherit from past and current generations under current law. Either future generations will have to pay an additional $11.1 trillion in present value or generations alive today will have to make this sacrifice, or a combination of both.

The open-group and closed-group measures are robust to the criticisms that apply to traditional measures of Social Security's finances. For example, both measures correctly identify the economic as well as intergenerational neutralities of the 'actuarially fair carve out' discussed earlier. In the case of a 'carve out with a haircut,' the open-group and closed-group measures both improve (they are both smaller), corresponding to a move toward sustainability and smaller burdens on future generations. In contrast, the traditional measures such as the trust fund exhaustion date and crossover date incorrectly show a deterioration of Social Security's finances in both instances.

Although the usefulness of the closed-group measure in determining sustainability is not as widely understood as the open-group measure's, the closed-group measure is vital to comprehending Social Security's impact on the economy.

Some believe that the closed-group measure is mostly meaningful in the context of a 'fully funded' system.[24] Under such a system, each generation would pay for its own benefits, and so the closed-group obligation would be zero.

But the closed-group measure is a very important statistic even in a pay-as-you-go system for two key reasons. First, it indicates the extent to which any reform will reshuffle fiscal burdens across generations. For example, suppose Social Security benefits were increased and this increase were financed on a strict pay-as-you-go basis by raising payroll taxes. This policy change would not have any impact on the open-group measure or

24 Stephen Goss, "Measuring Solvency in the Social Security System," in Olivia S. Mitchell et al., eds., *Prospects for Social Security Reform* (Philadelphia: University of Pennsylvania Press, 1999), 16–36. An equally plausible story is that policymakers allowed Social Security to become mostly pay-as-you-go over time because the burdens being placed on future generations were not easily observable under traditional measures.

the traditional measures. But the closed-group measure would grow larger because this reform would transfer wealth from future generations to current generations. Current generations would gain from this policy change since they would receive more in benefits in present value than they paid in taxes; indeed, current *retirees* would receive additional benefits for free. But future generations would pay for this windfall by receiving a benefit less valuable than the additional taxes they paid in present value. The closed-group measure, which shows the net gain to past and current generations, would become larger, thereby clearly indicating the extent of this intergenerational transfer.

Second, the closed-group measure reveals how much pay-as-you-go financing may 'crowd out' private saving and, hence, increase interest rates, lower wages, and reduce the nation's GDP.[25] Consider again a pay-as-you-go financed increase in benefits. Because this reform transfers resources from future to current generations, it reduces the amount of money today's generations must save for their own retirement. This reform, therefore, could *permanently* reduce the economy's level of capital.[26]

The Congressional Budget Office estimates that every dollar transferred from future to current generations reduces private savings by zero to fifty cents.[27] Although the wide range of this estimate suggests considerable uncertainty, it follows that Social Security may have reduced the U.S. capital stock by as much as $6 trillion and reduced GDP by as much as $1.1 trillion.[28] Nonetheless, the traditional measures as well as the open-group measure do not indicate these large macroeconomic effects. Presumably, any discussion of Social Security reform would want to take into account the impact of a reform on the economy. Although Social Security has had many successes, its potentially large deleterious effect on capital stock and national output deserves more attention in the debate over Social Security reform.

Because the open-group measure extends the traditional seventy-five-year imbalance measure beyond the seventy-fifth year, one might at first be tempted to argue that the open-group measure

25 Martin Feldstein, "Social Security, Induced Retirement, and Aggregate Capital Accumulation," *Journal of Political Economy* 82 (5) (September/October 1974): 905–926. Feldstein is the first to analyze the empirical issue of Social Security financing's impact on private saving.

26 The Ricardian equivalence hypothesis, however, argues that parents might leave a larger bequest in response to a transfer from their children, thereby leaving national saving unchanged. Robert J. Barro, "Are Government Bonds Net Wealth?" *Journal of Political Economy* 82 (6): 1095–1117. Altonji et al.'s empirical tests, however, reject this hypothesis. Joseph G. Altonji, Fumio Hayashi, and Laurence J. Kotlikoff, "Is the Extended Family Altruistically Linked? Direct Tests Using Micro Data," *American Economic Review* 82 (5) (1992): 1177–1198. Consistently, Gokhale et al. trace a large share of the secular decline in U.S. national saving during the last several decades to the fiscal transfers from workers to retirees. Jagadeesh Gokhale, Laurence J. Kotlikoff, and John Sabelhaus, *Understanding the Postwar Decline in U.S. Saving: A Cohort Analysis*, Brooking Papers on Economic Activity, Winter 1996.

27 Congressional Budget Office, "Social Security and Private Saving: A Review of the Literature," Congressional Budget Office Paper, July 1998.

28 The calculated reduction in GDP assumes Cobb-Douglas production with inelastic labor supply, a net-of-depreciation capital share of 0.25, and a current capital-output ratio of 3. The calculation also assumes that the private-saving offset is constant at fifty cents for each dollar of closed-group obligation.

places too much emphasis on Social Security's long-run finances. In other words, one could imagine a hypothetical 'reform' that does nothing to fix Social Security's finances during the first seventy-five years but enacts large reforms after the seventy-fifth year in order to eliminate Social Security's $11.1 trillion imbalance.

This potential criticism, however, is misplaced since it forgets that the $11.1 trillion open-group obligation is in terms of *present value*. Besides adjusting for inflation, the present-value calculation adjusts for the real interest costs that we save from paying obligations sooner rather than later. For example, increasing payroll taxes by one dollar today would reduce the open-group obligation by, of course, one dollar. But if we postponed this one-dollar tax increase (still measured in 2004 inflation-adjusted dollars) in one hundred years we would reduce the $11.1 trillion open-group obligation by only 4.7 cents in today's dollars.[29] Delaying the one-dollar tax increase 150 years would reduce the unfunded obligations by only one cent. Attempting to postpone reforms would just mean enacting unrealistically large reforms later on.

The closed-group obligation measure reflects the amount of projected overspending on past and current generations. Thus, a policy that lets current generations 'off the hook' produces a larger closed-group obligation than a reform that requires current generations to bear more of the costs.

Rather than drawing 'too much' attention to the long run, the open-group and closed-group obligation measures remove the biases, embedded in the traditional measures, against reforms that could improve Social Security's long-run financial outlook. These newer measures focus attention on the true magnitude of the reforms needed to place Social Security on a sustainable path and, hence, reveal the urgent need for action. Social Security's $11.1 trillion open-group unfunded obligation is almost *three times* as large as the amount the seventy-five-year imbalance measure indicates, *despite* the fact that the present-value calculation considerably reduces the weight placed on shortfalls that accrue after the seventy-fifth year.

Robert Myers, who was chief actuary of the Social Security Administration from 1947 to 1979, points out that before 1965 Social Security actuaries routinely relied on measures looking beyond seventy-five years. In 1965, however, Social Security's actuaries and policymakers began focusing on seventy-five-year shortfalls because then, unlike today, extending the financial projections beyond seventy-five years made very little difference to the program's financial outlook. However, Mr. Myers always thought that truncating measures at seventy-five years was never right in theory because of the moving-window problem: "I'm still an 'infinity' guy, because even if you have a seventy-five-year period, every year you do a new valuation you have some slippage."[30] This slippage is especially acute today, with over two-thirds of the $11.1 trillion shortfall lying outside of the seventy-five-year window.

Critics also charge that present-value estimates beyond seventy-five years are sensitive to underlying demographic and

29 This calculation uses an inflation-adjusted interest rate of 3.1 percent, the rate the trustees use to calculate the $11.1 trillion unfunded obligations.

30 Robert Myers, "Oral History Overview," 1995, <www.ssa.gov/history/myersorl.html> (accessed September 28, 2005).

economic assumptions.[31] Of course, uncertainty should only enhance the desire to seek remedies rather than to ignore the expected problem.[32]

Furthermore, different interest rate and productivity assumptions and different demographic projections do not greatly affect the size of the policy changes – either tax increases or benefit cuts – needed to reduce Social Security's imbalance.[33] Although changes in these underlying assumptions will alter the present value of the imbalance, the present value of Social Security's tax base and future benefits also move almost proportionally and in the same direction. As a result, the increases in tax rates or cuts in benefit rates required to eliminate Social Security's current fiscal imbalance exhibit much smaller sensitivity to parametric changes in economic and demographic assumptions.

President Bush's plan for personal accounts would create additional government debt while simultaneously reducing Social Security's unfunded future outlays. Government debt would increase as households could divert some of their payroll taxes to their personal accounts, thereby reducing government revenue. Future Social Security outlays would also decline however, under the President's actuarially fair carve out because the government could reduce benefit payments by one dollar in present value for each dollar placed into a personal account.

From an economic perspective, one dollar of government debt is not very different than one dollar of federal unfunded obligations. Both represent a dollar the government owes. Hence, real interest rates should not rise in response to the President's plan because investors should be indifferent between the two under reasonable circumstances.[34]

Legally, however, debt held by the public is a legal *liability* that the government must honor unless it declares bankruptcy.[35] Social Security and Medicare benefits, on the other hand, are only *obligations* of the government, which an act of Congress can alter. In practice, therefore, capital market participants may be discounting future Social Security benefits at a higher rate than the yield on Treasury securities because the capital market participants think that the government might pay only a portion of its present-law Social Security obligations in the future. Replacing a dollar in present value of future Social Security benefits with a dollar of explicit debt, therefore, could negatively affect how investors perceive the outlook of the federal government's finances.

However, the government is not necessarily more likely to pay explicit debt liabilities in real terms than Social Security obligations. Indeed, the opposite is also conceivable: most explicit debt

31 See, for example, Congressional Budget Office, "Measures of the U.S. Government's Fiscal Position Under Current Law," Congressional Budget Office Paper, August 2004.

32 This fact holds under any standard preference toward risk that shows a prudence motive.

33 Gokhale and Smetters, *Fiscal and Generational Imbalances*.

34 Technically speaking, the new government debt must have the same stochastic properties as Social Security benefits, including sensitivity to inflation and changes in the average wage in the economy.

35 Of course, in practice, the government can use inflation to reduce the real value of nominally denominated debt. The government would have to declare bankruptcy, however, to avoid paying off inflation-protected instruments.

is not protected against inflation. So faster inflation compounded over time could easily erode the value of the government's payments to bondholders. In contrast, the Social Security benefits of retirees and others, once determined, are fully protected against inflation, and will likely remain so well into the future. Moreover, even if policymakers believed that market participants discount future Social Security benefits by, say, 10 percent above the government's discount rate then policymakers could offer a 'carve out with a 10 percent haircut' to avoid disrupting capital markets.

The Social Security program provides an important source of income for most of the nation's retirees, but the program's long-term viability is in serious doubt unless a fundamental reform is undertaken – either by increasing taxes or by reducing the growth rate of benefits. Unfortunately, the traditional accounting measures used by policymakers and the media convey very little about the true magnitude of the financial problem facing Social Security. Those measures are also biased against reforms that could reduce Social Security's imbalance.

Fortunately, the Social Security trustees have begun to include new measures of Social Security's financial outlook, beginning with their 2003 report and continuing with the 2004 and 2005 reports – measures that fully convey the dimensions of Social Security's financial hole. The independent panel of experts appointed by the Social Security Advisory Board has endorsed these measures but, unfortunately, policymakers and the media are not paying sufficient attention to these new measures. We argue that these measures deserve much more careful consideration.

Lisa F. Berkman & M. Maria Glymour

How society shapes aging: the centrality of variability

We have all known people who grow old suddenly or seem much older than their chronological age. Conversely, we see people who appear vibrant and seem resilient to the challenges they face late in life. What role does society play in shaping these distinct outcomes of aging? The kinds of difficulties each of us are liable to face as we grow older are determined by our opportunities for social interaction and intimacy, by our economic and educational experiences, and by our exposure to severe social and physical stresses. We believe that a great deal of what superficially may seem like random variability in health outcomes is in fact patterned by the kinds of social and economic experiences that people confront throughout their lives. Such variability is key to understanding aging.

Social conditions help determine several health outcomes as we age. Variations in life expectancy and disability, for example, reflect differences in past social investments in the health and well-being of different groups within society. Perhaps more surprising, aging at the biological level – as indicated by measures of metabolic function, glucose metabolism, blood pressure regulation, and pulmonary function – is also correlated with social conditions.

One of the most common indicators of health and aging used in epidemiology and demography is life expectancy, a summary estimate of how long we can expect people to live in the future. Life-expectancy measures are derived from current death rates for men and women in specific age groups. The assumption is that age-specific death rates today will be applicable in the future. Because death rates historically have changed rather quickly, though, projections are not always very accurate.

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Measures of life expectancy, however, are valuable when comparing countries or populations over time – especially when used to look at historical or current, not future, patterns. In the United States in 2002, life expectancy was 77.3 years at birth and 18.2 years at age 65. The latter number means that if a person survived to age 65, he or she could expect to live, on average, another 18.2 years. These summary statistics, however, hide large differences in life expectancy across gender, racial, and ethnic groups. For example, life expectancies for white women were 80.3 years at birth and 19.5 years at age 65, but for African American women those same numbers were 75.6 and 18.0. Life expectancies for black men were 68.8 years at birth and 14.6 years at age 65. For white men, they were 75.1 years at birth and 16.1 years at age 65. Thus, even a preliminary unpacking of average life expectancies of white women and African American men reveals a difference of 11.5 years at birth and about 5 years at age 65.

Equally startling are advances in life expectancy that have occurred over the last century and those predicted based on data from the last decade or two. Life expectancy in the United States increased over twenty years between 1900 and 1950, from 47.3 to 68.2, and nearly another ten years by 2000, to 76.7. If one looks at the long-term increases in life expectancy, the majority of 'added' years in the first half of the century occurred because of improvements in infant and childhood mortality, maternal mortality, and control of infectious diseases – the results of major public-health efforts at the turn of the century.[1]

However, over the last several decades we have also achieved substantial gains in life expectancy for older people. Between 1980 and 2000, life expectancy for 65-year-olds rose 1.6 years. In these decades, increases were greatest for men, who gained almost two years. These added years narrowed the gender gap slightly, but women can still expect to live three years longer than men.

Living longer may be problematic, though, if the elderly spend many of those extra years disabled or suffering from chronic conditions. Ideally, we want to increase 'active' or 'disability-free' life expectancy. Scientific opinion on whether increased life expectancy would translate into increased old-age disability has evolved over time.

In the early 1980s, investigators such as James Fries anticipated "compression of morbidity" to accompany increases in life expectancy.[2] Early evidence contradicted this notion: Health surveys indicated that people were living longer, but with substantial levels of disability for many of those years. Better treatments for many diseases were saving lives, but also leaving survivors with serious disabilities. However, more positive signs have emerged over the past few decades: Eileen Crimmins reported that disability-free life expectancy improved more during the 1980s than the 1970s,[3] while some evidence suggest rates of cognitive impairment have been decreasing over the last decade or so.[4]

1 D. Cutler and G. Miller, "The Role of Public Health Improvements in Health Advances: The Twentieth-Century United States," *Demography* 42 (1) (2005): 1 – 22.

2 J. Fries, "Aging, Natural Death and the Compression of Morbidity," *New England Journal of Medicine* 303 (1980): 130 – 135.

3 E. M. Crimmins, Y. Saito, and D. Ingegneri, "Trends in Disability-Free Life Expectancy in the United States, 1970 – 1990," *Population and Development Review* 23 (3) (1997): 555 – 572.

4 V. Freedman et al., "Resolving Inconsistencies in Trends in Old-Age Disability: Report

Unfortunately, researchers have found marked social inequalities in disability-free life expectancy. Although data are limited, recent studies show that African Americans, especially those living in urban poverty areas, have a strikingly shorter active life expectancy than whites or African Americans living in nonpoverty areas. In a study of life expectancy from age 16, Arline Geronimus discovered that white women living in the United States have an active life expectancy of fifty-two years while African American women – even in nonpoverty urban areas such as Queens, New York, and the better parts of Detroit and Chicago – have one of forty-three. Black women in Harlem, central Detroit, or the south side of Chicago, on the other hand, have the lowest active life expectancy, only thirty-nine years. Even white women living in poverty areas generally have active life expectancies better than those of African American women, ranging from forty-two years in Appalachian Kentucky to forty-nine years in west North Carolina and south central Louisiana. Thus, both poverty and race are factors in the variations in active life expectancy.[5]

Similar variations in life expectancy have been observed between occupational grades in other countries. Both disability-free and total life expectancy increased in France between 1980 and 1991, with larger improvements in disability-free life expectancy. Though the gains were relatively uniform across socioeconomic groups, substantial class disparities had existed in 1980. Thus, class disparities were preserved. In 1991, manual laborers in France still lived 5.4 years less than managers and spent 1.4 more years with a disability.[6]

Three important findings emerge from these studies that are generally applicable to the United States and to other industrialized countries. First, in recent decades, both mortality and disability rates have dropped for older men and women. Second, these gains have affected men and women across virtually all social classes and racial and ethnic groups, though the socioeconomically advantaged have experienced slightly larger absolute gains. Third, while population health as a whole has improved, inequality has persisted across the socioeconomic strata. The gap between rich and poor has not narrowed and, depending upon which indicators are used, may actually have increased in both absolute and relative terms.

The good news is that greater longevity does not mean that we also have to endure many more years of severe disability. Even though the incidence of many diseases has not declined dramatically in the last decades, the consequences – mortality and disability – have decreased.[7] Despite this positive result, differences in outcomes among people based on their social and economic positions are large and persistent. In the next section, we explore the extent to which social and economic investments made

from a Technical Working Group," *Demography* 41 (3) (2004): 417 – 411.

5 A. T. Geronimus et al., "Inequality in Life Expectancy, Functional Status, and Active Life Expectancy Across Selected Black and White Populations in the United States," *Demography* 38 (2) (2001): 227 – 251.

6 E. Cambois, J. Robine, and M. Hayward, "Social Inequalities in Disability-Free Life Expectancy in the French Male Population, 1980 – 1991," *Demography* 38 (4) (2001): 513 – 524.

7 J. M. Robine, P. Mormiche, and C. Sermet, "Examination of the Causes and Mechanisms of the Increase in Disability-Free Life Expectancy," *Journal of Aging and Health* 10 (2) (1998): 171 – 191.

at the national level have had a major impact on the health of the current generation of older men and women. We speculate that as much as explicit, turn-of-the-century investments in public health helped to control infectious diseases and curb infant and maternal mortality, other social and economic investments initiated in the first half of the twentieth century may have also broadly influenced the successful aging of current senior citizens.

The trajectory of health or ill health that the elderly undergo does not begin at age 65: experiences accumulated throughout life set the stage for well-being in old age. To understand the health of today's elderly, it is important to examine the experiences of these cohorts before entering old age. The majority of the elderly in America today were born between the two world wars; those who celebrate their eightieth birthday in 2005 were born during the presidency of 'Silent Cal.' During this time, women gained greater formal political power and rights, while in the South, lynchings of blacks, though declining, still constituted an important form of social control.[8] Southern blacks responded to changing economic and social conditions by migrating north in record numbers.[9] Some older people today may have been immigrants in the large waves of migration that came from Europe, crowding into urban tenements. Others may have been midwestern children during the Dust Bowl years, when bad weather and economic downturns deprived millions of their farms, their primary livelihood.

This cohort entered adulthood as the nation entered World War II. After the war, many members of this generation experienced prosperity, but many also faced continued poverty and racial discrimination. The government responded to many of these social conditions with investments in public education, workplace regulation, federal housing initiatives, and employment and income insurance programs. Further economic progress and extensions of civil rights reflected both secular developments and government intervention.

Improving health and longer lives may have been among the most dramatic and unexpected consequences of these social policies. Medicare and Medicaid were obvious investments in medical care, but investments in other social goods conferred inadvertent health benefits that may have played an even more important role in how older men and women age today.

So which social changes were most relevant to the health of the aging in the twentieth century? Educational attainment, working conditions, minimum income and income stability, and housing conditions all changed profoundly between 1910 and 1980. Civil rights laws in the second half of the century also changed, extending rights and opportunities to many who had been denied them earlier. However, because so little research has directly addressed the health consequences of social policies, we are not in a position to give compelling evidence about which social or economic policies had the greatest effect on health.

Still, what we can surmise is suggestive. For example, we expect work-relat-

8 E. M. Beck and S. E. Tolnay, "The Killing Fields of the Deep South – The Market for Cotton and the Lynching of Blacks, 1882 – 1930," *American Sociological Review* 55 (4) (1990): 526 – 539.

9 S. E. Tolnay, "The African American 'Great Migration' and Beyond," *Annual Review of Sociology* 29 (2003): 209 – 232.

ed policies to have profound health effects. During the twentieth century, both public and private efforts to improve occupational health and safety directly reduced fatalities: between 1933 and 1997, deaths from unintentional work-related injuries declined by 90 percent.[10] Improvements in working conditions probably also affected long-standing chronic conditions and risk of disability. But a number of labor protection policies introduced or universalized in the twentieth century – such as workmen's compensation, unemployment, and minimum wage standards – also likely influenced health outcomes in workers. Although implemented primarily to buffer workers from poverty, these policies may have increased chances of survival and freedom from disability in old age.

Similarly, we might examine the health impacts of housing policies that regulated physical hazards in homes or promoted homeownership by increasing mortgage availability. Some policies may have even been harmful, e.g., home loan programs that served to stabilize racial segregation in neighborhood housing.

We believe that the variations in life expectancy and health in old age observed in current cohorts of the elderly may reflect the consequences of differential social investments. Thus, research on social and economic policies implemented decades ago is important. Policies impact living conditions, which in turn may have profound effects on health and the aging process.

10 "Improvements in Workplace Safety – United States, 1900 – 1999," *Morbidity and Mortality Weekly Report* 48 (22) (1999): 461 – 469. This decline was presumably also attributable, at least partially, to changes in the predominant types of jobs held by workers.

To illustrate how we might explore such associations, we will focus here on one set of policies, relating to educational attainment. Our goal is to show how significant changes in these policies, enacted decades ago, may continue to reverberate in the health patterns of today's elderly.

Educational attainment, in general, is a potent predictor of good health. Epidemiological studies suggest more education correlates with better cognitive and physical function, as well as decreased risks of death, dementia, and a number of diseases – including cardiovascular disease, the major cause of death in the United States. The reasons for this are undoubtedly complex and have often been thought to reflect the intrinsic values, health, and intelligence of individuals who continue their education, not the benefits of education per se. However, there is good reason to believe that there may be a more causal link between education and health. On average, education translates into higher incomes and therefore higher standards of living. People with higher levels of education also tend to have health-promoting behaviors like consuming less tobacco and alcohol and getting more physical activity.

Educational policies changed drastically in the twentieth century in the United States and many other countries. Lawrence Katz and Claudia Goldin have described the first half of the twentieth century as the "second transformation of U.S. education,"[11] when high school completion, previously a rare achievement, became the norm. Changes in average educational attainment partially

11 C. Goldin and L. F. Katz, "Human Capital and Social Capital: The Rise of Secondary Schooling in America, 1910 – 1940," *Journal of Interdisciplinary History* 29 (1999): 683 – 723.

reflected greater demand for schooling. However, extensive and explicit government intervention also increased both the demand for and supply of schooling.

In the United States, state and local governments have historically determined educational policy, and the first half of the twentieth century was a time of exceptionally rapid changes in schooling policies and standards. For example, in 1918, six states required students to complete six or fewer years of schooling before leaving school, and seven more required only seven years of schooling. By 1939, the lowest state schooling requirement was seven years, and all but four states required eight or more years.

Still, compulsory schooling laws varied widely across states and over time. For instance, in 1918, North Carolina mandated enrollment at age 8 and permitted exit at age 14. By 1939, the enrollment age dropped to age 7, effectively adding one year to the length of compulsory schooling. Florida during this time switched from requiring schooling from ages 8 to 14 (six years) to requiring schooling from ages 7 to 16 (nine years). Similar increases occurred all across the United States during this period.

By virtue of extensive social investments, significant improvements in the quality of schooling – including extensions of the school year, construction of new buildings, grade separation, increasing teacher qualifications, and standardization of curriculum – were also achieved during the first half of the twentieth century. The expansion of high school education was probably quite costly to the communities that accomplished it: direct costs of offering high school were roughly twice that of elementary school, and indirect costs, in terms of transportation and lost adolescent labor, were also likely substantial.

On top of local action to build high schools and enroll students, and state changes in compulsory schooling laws, the G.I. Bill (formally the 1944 Serviceman's Readjustment Act) represented a tremendous federal investment in expanding educational opportunities. The G.I. Bill provided unemployment benefits, job search assistance, and loan guarantees for small businesses or home purchases; however, it is best known for promoting education via subsidies for tuition and living expenses – at a cost of over $14 billion to the government. Ultimately, because nearly 8 million World War II veterans received benefits under the aegis of the G.I. Bill, participation in World War II probably raised college completion rates by 5 to 6 percentage points, a substantial increase over prewar college completion rates.[12] Millions more Korean War veterans also benefited from subsequent G.I. Bills.

Social investments such as schooling laws and the G.I. Bill did not affect everyone in the country equally. In states where school segregation was legal, investments in measures such as longer school-term lengths were unequal. Similarly, compulsory schooling laws did not dramatically help young African Americans because many states rarely enforced them for blacks. Even 'race-neutral' policies such as the G.I. Bill, benefited southern blacks very little.[13] In the postwar period African Ameri-

12 J. Bound and S. Turner, "Going to War and Going to College: Did World War II and the G.I. Bill Increase Educational Attainment for Returning Veterans?" *Journal of Labor Economics* 20 (4) (2002): 784 – 815.

13 S. Turner and J. Bound, "Closing the Gap or Widening the Divide: The Effects of the G.I. Bill and World War II on the Educational Outcomes of Black Americans," *Journal of Economic History* 63 (1) (2003): 145 – 177.

can G.I.s were much less likely than white veterans to have completed high school and to be eligible for college tuition benefits. Southern colleges serving blacks also had few seats, turning away many veterans because of inadequate resources.[14] The G.I. Bill, one of the most important federal educational policies in the history of the United States, probably reduced educational inequalities among white men. For blacks, however, it may have stabilized or exacerbated inequalities.

Research explicitly evaluating the effects of twentieth-century social policies on health is relatively limited, in part because of the enormous challenge of finding compelling study designs to investigate these questions. Recently, however, economists have exploited the temporal variations in changes to state schooling requirements – in what amounts to a 'natural' experiment – to examine the effects of education on adult earnings.[15] This work is now being extended to explore the consequences of compulsory schooling on health.[16]

We often think of natural experiments as arising out of natural disasters such as earthquakes or hurricanes, but social scientists have employed this concept very effectively to explore the effects of numerous social 'exposures,' which are normally difficult to randomize in a traditional experiment. Because states have often enacted or extended compulsory schooling laws differently or at different times, we can view this situation as a 'natural' experiment. Except here, the 'treatments' are assigned to individuals by states rather than by a scientist.

In the case of compulsory schooling regulations, individuals receive varying levels of exposure depending on their states' compulsory schooling requirements. While other factors at the state level may occur simultaneously with changes in compulsory school laws, nothing innately individual determines exposure. Therefore, we can sometimes make stronger causal inferences from natural experiments than we can from many observational studies.

There are two important limitations to such a research design. First, when state-level factors determine exposure, it is difficult to disentangle correlated state characteristics and identify which characteristic is at work. Second, for statistical reasons, the results of these studies tend to have wide bounds of uncertainty. Nonetheless, natural experiments represent one of the most promising approaches to understanding how social policies and socioeconomic conditions influence health, and to strengthening our confidence in the results of observational studies.

Initial findings are striking. Early work suggests that changes in compulsory schooling laws impacted both mortality and cognitive function. Adriana Lleras-Muney compared changes in state schooling laws between 1915 and 1939 with census information on the education and mortality rates of people who were age 14 – roughly the prime age to drop out – during these years. Lleras-

14 D. H. Onkst, "'First a Negro . . . Incidentally a Veteran': Black World War II Veterans and the G.I. Bill of Rights in the Deep South, 1944 – 1948," *Journal of Social History* (Spring 1998): 517 – 543.

15 D. Acemoglu and J. D. Angrist, "How Large Are the Social Returns to Education? Evidence from Compulsory Schooling Laws," National Bureau of Economic Research, Working Paper 7444 (1999).

16 J. Angrist and A. Krueger, "Does Compulsory School Attendance Affect Schooling and Earnings?" *The Quarterly Journal of Economics* 106 (4) (1991): 979 – 1014.

Muney found that one additional year of compulsory schooling (or schooling required for a work permit) translated into a 5 percent increase in the average completed years of education for people born in that state.[17] Each additional year of schooling induced by the laws was also associated with a 3 to 6 percentage point reduction in the risk of death over ten years.[18]

Recently completed analyses of the effects of compulsory schooling laws on men and women who were born between 1900 and 1947 confirmed that individuals born in states with high levels of mandatory schooling completed more schooling. They also performed better on cognitive tests taken many decades after finishing school, even after adjusting for demographic characteristics such as race and parental education.[19] We are now extending this work to examine whether other state-level characteristics that changed contemporaneously with schooling policies, such as economic development or the spread of kindergarten enrollment, might account for this relation, and whether schooling appears to have the same effect on other health outcomes.

While we are actively working to identify the most influential policies, these results, in the meantime, highlight the general importance of social-environmental conditions during childhood in determining risk of later cognitive impairment. Despite the common perception that cognitive impairment late in life is a function of one's genetic endowment, these results suggest that state policies, perhaps related to schooling, can help protect the elderly regardless of their individual genetic backgrounds. We can and do change policies such as schooling requirements or resources in response to evolving social demands. If we can show that compulsory schooling plays a salutary role in growing old gracefully, then we can change public policy in order to promote healthier aging for future cohorts.

Individual characteristics undoubtedly influence how we do in old age. Our genes shape how we age – but so do government policies. Indeed, the example of educational attainment indicates that critical childhood experiences can shape outcomes in late life. Mark Hayward has referred to this phenomenon as "the long arm of childhood."[20] Important questions remain. First, what are the crucial biological, psychological, or social mediators between early life experiences and adult health? Second, we know, for example, that education protects individuals from a range of physical and psychological stressors. But are there windows of time in the human life course during which social exposures are most powerful and after which they are ineffective? To answer these questions, gaining further insight into the biological pathways linking social experiences to aging processes is crucial.

17 A. Lleras-Muney, "Were Compulsory Attendance and Child Labor Laws Effective? An Analysis from 1915 to 1939," *Journal of Law & Economics* 45 (2) (2002): 401 – 435.

18 A. Lleras-Muney, "The Relationship Between Education and Adult Mortality in the US," *Review of Economic Studies* 72 (1) (2005): 189 – 221.

19 M. M. Glymour, "Identifying Social Determinants of Old Age Cognitive Function" (dissertation, Harvard School of Public Health, 2004).

20 M. Hayward and B. Gorman, "The Long Arm of Childhood: The Influence of Early-Life Social Conditions on Men's Mortality," *Demography* 41 (1) (2004): 87 – 107.

Here, we explore the ways in which both stressful and fulfilling social experiences actually shape the biology of aging.

Scientists used to believe that each individual was born with an intrinsic 'biological clock' that determined his or her rate of aging. Extensive research focused on identifying indicators of an individual's 'biological' age. Telomere length emerged as a powerful marker of aging. Telomeres are DNA-protein complexes that cap chromosomal ends, promoting chromosomal stability; telomerase is a cellular enzyme that protects telomeres. With age, telomeres shorten in humans, and in vitro cells become senescent when telomeres shorten sufficiently. Indeed, investigations have linked telomere shortening to higher mortality rates in the elderly and to cardiovascular disease.[21] New evidence indicates that both internal and external environmental conditions influence telomere length, potentially producing some of the health variability we observe among older people.

A recent study by psychologist Elissa Epel and biochemist Elizabeth Blackburn examined the effect of social stressors on telomeres, providing us with incredible insight into how social stressors might accelerate aging. The researchers assessed perceptions of stress in a group of almost sixty healthy mothers between the ages of 20 and 50 who had either a seriously chronically ill child or a healthy child.[22] Caregiving, especially for intimate partners and close family, had been known to increase risk of mortality and a host of mental and physical problems.[23] But the biological basis for the heightened risk was unclear. This study discovered that mothers who had been taking care of a chronically ill child for a long time had shortened telomeres, decreased telomerase activity, and higher levels of oxidative stress than mothers who had not been in that caregiving role for long. Furthermore, perceived stress correlated with telomere shortening in all the mothers. In terms of telomere length, women with the highest levels of perceived stress were comparable to women a decade older. Thus, we can see how social stressors influence aging at a cellular level.

Several theories are compatible with this finding. Researchers have long speculated that social and psychological stressors, much like physical activity, have generalized consequences for health. These stressors influence a wide array of diseases through a set of physiological pathways that are so strongly related to chronological age they are considered markers of the aging process. We have hypothesized that stresses presented by social disadvantage and social isolation, as well as major life events such as bereavement and loss, affect health by accelerating the rate of aging.[24] By eroding individual capacity for resilience,

21 R. M. Cawthon et al., "Association Between Telomere Length in Blood and Mortality in People Aged 60 Years or Older," *Lancet* 361 (9355) (2003): 393 – 395, and S. Brouilette et al., "White Cell Telomere Length and Risk of Premature Myocardial Infarction," *Arteriosclerosis Thrombosis and Vascular Biology* 23 (5) (2003): 842 – 846.

22 E. Epel et al., "Stress and Body Shape: Stress-Induced Cortisol Secretion is Consistently Greater Among Women with Central Fat," *Psychosomatic Medicine* 62 (2000): 623 – 632.

23 S. Lee et al., "Caregiving and Risk of Coronary Heart Disease in U.S. Women: A Prospective Study," *American Journal of Preventive Medicine* 24 (2) (2003): 113 – 119.

24 L. Berkman, "The Changing and Heterogeneous Nature of Aging and Longevity: A Social and Biomedical Perspective," *American Review of Gerontology & Geriatrics* (8) (1988): 37 – 68.

these stressors presumably leave people more vulnerable to a host of other genetic or environmental risks. Encounters with these other risks determine the specific disease developed. That is, the social stress 'leans' on the aging body, accelerating the onset of disease, but does not necessarily determine which specific disease occurs.

Two related theories support this framework. 'Weathering,' a concept elaborated by Geronimus, explains African Americans' more rapid deterioration in health and different experiences of reproductive health in terms of their prolonged exposure to discrimination and stress.[25] Other work has focused on 'allostatic load,' or the cost of adapting to the heightened physiological responses that result from repeated or chronic environmental challenges – the wear and tear on the body.[26] In studies of humans, Teresa Seeman and Bruce McEwen initially identified a number of biological indicators of allostatic load. These biomarkers include those related to the sympathetic-adrenomedullary system, the hypothalamic-pituitary-adrenal axis, and the cardiovascular metabolic and immune systems. Longitudinal studies have shown that socioeconomic disadvantage and social isolation predict allostatic load, which itself predicts mortality.

Accumulating evidence indicates that social as well as biological factors determine how people age. Paying attention to the variability in aging produced by exposure to quite different social experiences may provide us with critical clues as to how we can improve the health and well-being of older men and women. We believe that the investments society makes for its citizens all along the life cycle from early childhood to old age accumulate to affect the health of the older population, for good or ill. The degree to which those social investments are unequally distributed will create health inequalities difficult to erase as people age.

25 E. Breeze et al., "Do Socioeconomic Disadvantages Persist Into Old Age? Self-Reported Morbidity in a 29-Year Follow-Up of the Whitehall Study," *American Journal of Public Health* 91 (2) (2001): 277 – 283.

26 B. S. McEwen, "Sex, Stress and the Hippocampus: Allostasis, Allostatic Load and the Aging Process," *Neurobiology of Aging* 23 (5) (2002): 921 – 939.

Poem by Charles Wright

Last Supper

I seem to have come to the end of something, but don't know what,
Full moon blood orange just over the top of the redbud tree.
Maundy Thursday tomorrow,
                then Good Friday, then Easter in full drag,
Dogwood blossoms like little crosses
All down the street,
                lilies and jonquils bowing their mitred heads.

Perhaps it's a sentimentality about such fey things,
But I don't think so. One knows
There is no end to the other world,
                no matter where it is.
In the event, a reliquary evening for sure,
The bones in their tiny boxes, rosettes under glass.

Or maybe it's just the way the snow fell
                a couple of days ago,
So white on the white snowdrops.
As our fathers were bold to tell us,
                it's either eat or be eaten.
Spring in its starched bib,
Winter's cutlery in its hands. Cold grace. Slice and fork.

Charles Wright, a Fellow of the American Academy since 2002, is Souder Family Professor of English at the University of Virginia in Charlottesville and the author of many books of poetry, including "Country Music: Selected Early Poems" (1982), which won the National Book Award, and "Black Zodiac" (1997), which received the Pulitzer Prize. He has also published two works of criticism, "Halflife" (1983) and "Quarter Notes" (1995). In 1999 he was elected a Chancellor of the Academy of American Poets. This poem will appear in "Scar Tissue" in the spring of 2006.

Fiction by Ree Davis

I kneel before you

Light streaming into the room wakes me. I curl around Jaichin's body and close my eyes. Jaichin pushes me away.

"Get up," he says, his face buried in the pillow. "Get up, Xiao-li. It's your time."

The morning is my responsibility. The others always sleep late. I sit up on the mattress. This room is small, but the apartment is large with seven rooms. Our one window faces east, so we always get morning sun. It does not bother Jaichin, but the morning light reminds me of home. I miss my mother most in the morning, but now I push her from my mind.

I stand up and pull on my pants and t-shirt. Jaichin's long, lean body lies motionless on the bed. He wears only underpants. I would love to wake him and make love, but he needs the sleep. He will be hung over from last night. I leave, without disturbing him.

I move across the hall to the toilet, not looking at the door to her room. I do not want to think about her yet, so I go into the toilet and rinse my face in the sink. My eyes are swollen with sleep. Everyone was up late. I cannot wait until they decide it is time for her to go.

I shuffle into the kitchen, pausing to listen at her door. There is no sound. I get a bowl of rice and sit to eat. I drink the tea that remains in my cup from yesterday. I sit longer than I should, delaying the start of the day. I wash my dishes and look out the window into the narrow space behind our building. It is lined with windows to other kitchens just like ours. Everything is filthy and worn; no one cares about this place. After putting the dishes away, I leave the kitchen, stopping in the toilet to get a bucket and cloth. I cross the hall to her room.

I open the door and peer in before entering. Around her, the room is empty except for some old construction materials stacked by the door. She is in the center of the room, just as they left her. Her head hangs down on her chest; she is still asleep. Her wrists are tied together behind the chair. Her ankles are bound and tied to the chair's legs. Her head is covered with her blouse; I tied it there myself last week. I cannot see her face.

"I kneel before you" won first runner-up in the "South China Morning Post"/Radio Television Hong Kong's 2003 short fiction competition. After spending three years in China, Davis lives in North Carolina, where she is an architect and a writer. She is finishing her first novel, "A Terrible Energy."

I do as I have been instructed, cleaning the blood and urine that has collected on the floor at her feet during the night. She stirs; I step back. I know she cannot hurt me; she is bound too tightly. Her cloaked head faces mine. Can she see through the fabric? I look away. I hear her mutter through the tape covering her mouth. I ignore her muffled words and finish cleaning. As I stand to take the bucket from the room, she mutters again and a new pool of urine forms on the floor. I put down the bucket, soak the cloth in the puddle at her feet, and squeeze it over her. She struggles beneath the stream.

I clean the floor again and leave the room to dump the contents down the toilet and rinse the rag. When I am through, I return and sit on the floor behind her. As with every morning, my mind wanders and I think of my mother. I came to this house six months ago, soon after I started seeing Jaichin. Though I am much younger than he, we fell passionately in love. My father claimed Jaichin was a gangster and our affair brought shame to the family. He banished me from my home. That was the last time I saw my mother. She knelt on the floor behind him, sobbing not to lose her only daughter. Now, I can only take comfort in my memories of her. When I was not in school, I spent every moment with her. In the evening, after she put me to bed, she bathed my father's mother in our kitchen. I was quite young, but I remember watching them from the nook where I slept. My mother would help my grandmother undress. Then, my mother would wash her from a bucket of warm soapy water. In silence, my mother lifted each withered limb and stroked it clean. The ritual continued until my grandmother died.

My thoughts are interrupted when the woman shifts in her chair. She rolls her head, trying to face me. She is crying. This makes me angry.

"It is your own fault. You stole from Mr. Wong." I am annoyed. "Your fault. Not mine. Your tears mean nothing."

She tries to move the chair with her body, but she is too weak. She has been here for four weeks. Wong, Jaichin, and Xin, Jaichin's younger brother, brought her here one night. I came to the door of the room, but it was locked. I asked Jaichin what was happening. He said to go to our room, where I waited for him. Through the walls, I could hear her crying to be left alone, to go home, to go back to her baby. They were beating her, calling her a whore and an addict. I was scared, but did nothing.

Before that night, everything was fine between Jaichin and me. I would do anything for him, but now, he spends most of his time with her. Jaichin said she used to buy drugs from them, but stopped when she had a baby. She stole from them, so they had to make an example of her. After seeing her, I wondered if they also brought her here because she was beautiful.

My thoughts are interrupted when she starts banging the chair legs against the floor. I am afraid she will wake everyone, so I get up. I face her cloaked head, "Stop. Now."

I can see tears have soaked the blouse. This makes me angrier. She is naked from the waist up. Her skin is bruised and marked from these weeks with us, but her breasts are still beautiful. My face flushes. Her beauty, which shines through her beaten flesh, angers me more. Jaichin has been touching this skin instead of mine. Jaichin comes to our bed smelling of her. Jaichin does not want to touch me anymore.

Suddenly, the door bangs open and Jaichin enters. "Stupid, useless girl. Can't you keep her quiet?"

I shrink at his rage. He picks up a pipe leaning near the door and crosses the room. In one graceful swing, he smashes the pipe against her head. Her torso slumps forward. He hands the pipe to me and leaves the room, slamming the door.

I am stunned and stand motionless with the cold pipe in my hands. Deep red begins to seep through the fabric covering her face. Her head hangs loose from her neck. I think she is dead. Hot tears race down my cheeks. I pace back and forth, clutching the pipe, scared by the thin stream of blood seeping down her bare breasts and by what Jaichin may have done.

It seems hours pass before I have the nerve to put the pipe down and untie the blouse. I remove the fabric, revealing her face. It is now slack with death. Her jaw falls loose from the blow. The tape hangs useless from her mouth. I kneel before her. I must compose myself; this is no time for self-pity.

I get up, go to the kitchen, and get scissors. When I return, I kneel to cut the bindings from her wrists. Her thin fingers are bruised and broken. The nails are ringed in blood and dirt. The bindings are tight. I must push the scissors into the flesh of her wrists to cut them. They have been there for so long, I must peel them off. Once discarded, the bindings form a broken ring on the floor. Her hands are stuck together with blood; I pull them apart and bring them around to place in her lap. Her waist is bound to the chair, so she does not fall. I kneel again to undo the bindings at her ankles and see bruises up the length of her legs. These are from the second week, when the men stopped beating her and began raping her. I listened from the other room as she struggled beneath them; my face grew hot with jealousy. I wanted Jaichin to stop, but for the wrong reasons. During the mornings of that week, I spat on her. Now, I see how fragile she is. Her arms and legs are pale and thin. Again, I have to push the scissors into the flesh of her ankles to cut the bindings. Again, dried blood binds her limbs together. I will have to clean her to be able to remove the other bindings, so I get the bucket and rag. As I move across the hall, I notice the other rooms are quiet: no one else is awake.

I return, closing the door and locking it. I soak the rag in warm water and clean the blood from her torso. I gently soak the cords that encircled her before I unwrap them. When she is finally free, I pull her from the chair and lay her body on the floor. I cut her tattered and soiled skirt, pulling it off from under her body. She is naked, but covered with blood and filth. I change the water in the bucket several times, flushing the red liquid down the toilet and refilling it with fresh water. I rinse her blouse clean too. Each time after I return to her, I close and lock the door.

When she is completely clean, I put away the bucket, return to the room, and lock the door. I sit beside her. Her body is a map of the last four weeks. The yellowish purple areas of her face, arms, and hands mark week one, when every day she pleaded to be returned to her child, but instead was beaten unconscious. Dark purple areas on her wrists, ankles, and thighs mark week two, when they gagged her and repeatedly raped her. Burns on her face, breasts, legs, and feet were formed in week three. Red welts extend from these areas, indicating infection had set in. Some marks are chemical burns from caustic cleaning fluid. These were my contribution to her torment. I felt excluded, so I gave them ideas for torture and was proud to be allowed to participate. It felt good to hurt her then;

she was the woman who stole Jaichin from me. This last week, week four, they had grown tired of her, so she was beaten just to keep her unconscious. Despite the shroud of violence she wears, I know she was still beautiful until Jaichin's last blow. My hands tremble as I tie her shattered jaw closed with the blouse that has served as her mask. I close her eyelids.

As I sit beside her, I see the marks of childbirth on the skin of her stomach and the edges of her breasts. I see the holes in her earlobes that once held earrings, and the white bands on her fingers where there once were rings. There is a scar on one knee, perhaps from a childhood fall. I hold her hand in mine, as my mother held my grandmother's hand after preparing her body the day she died. I want to cry for this woman, but know I do not have the right, so we sit together in silence, she and I, for hours.

The room is peaceful until I hear the men waking in the rooms beyond. It is not long before they begin pounding on the door.

Dialogue between Daniel Bell & Wolf Lepenies

On society & sociology past & present

Translated by Howard Eiland

"That's no way to start a newspaper article!"* How many times have I heard Daniel Bell say that? For several years he and I worked together with a Japanese colleague – the literary critic and author of No plays, Masakazu Yamazaki – to edit *Correspondence*, a magazine funded by the Japanese Suntory Foundation and first published by the American Academy, and then by the Council on Foreign Relations in New York. Although there were no official positions on the staff, Masakazu and I gladly followed Daniel Bell's lead on a host of matters, ranging from choice of themes for particular issues, to the makeup of pages, and the selection of vignettes to illustrate articles. There was no question who was in fact the primary editor.

I was not the only one who thought that Daniel Bell was a great journalist. Bell's retirement in 1958, after ten years with the magazine *Fortune* (with the exception of a year and a half in Paris), astounded the newspaper magnate Henry Luce.

"But why?" he wanted to know. "You're body and soul a journalist! What reason could you possibly have to return to academic life?"

Daniel Bell, a Fellow of the American Academy since 1964, is Henry Ford II Professor of Social Sciences Emeritus at Harvard University. Bell's publications include "The End of Ideology" (1960), "The Reforming of General Education" (1965), which won the gold medal of The American Council of Education, and "The Cultural Contradictions of Capitalism" (1976). He has written or edited eighteen books, a number of these in Japanese. He was for twenty years a councillor of the Suntory Foundation in Japan and for ten years Scholar-in-Residence at the American Academy.

Wolf Lepenies, a Foreign Honorary Member of the American Academy since 1992, was Rector of the Wissenschaftskolleg zu Berlin (1986 – 2001) and is now a Permanent Fellow there and professor of sociology at Freie Universität Berlin. Among other prizes, he received the Alexander von Humboldt Prize for French-German Scientific Cooperation, the Karl Vossler Prize, and the Joseph Breitbach Prize. His publications include "Die drei Kulturen" (1985), "Benimm und Erkenntnis" (1997), and "Sainte-Beuve: au seuil de la modernité" (2002). His latest book, "The Seduction of Culture in German History," will be published by Princeton University Press in the spring of 2006.

* Wolf Lepenies met with Daniel Bell on December 4, 2004, in Cambridge, Massachusetts. The German version of the interview was published in the newspaper *Die Welt* on January 12, 2005.

"Three reasons," answered Bell. "June, July, and August."

Of course, the lure of summer vacations was not the only thing that led Bell, who during his time at *Fortune* was already teaching at Columbia University, to return to an academic career. Born in 1919 in New York, Daniel Bell early on developed a sociological eye for things, an intuitive grasp of the fundamental changes taking place in the structure of society. Today the titles of his books – *The End of Ideology*, *The Cultural Contradictions of Capitalism*, and *The Coming of Post-Industrial Society* – have become bywords in discussions of modernity.

Bell taught at Columbia as a professor of sociology until 1969. He then switched to Harvard, where he remained until his retirement in 1990. He'd come a long way from 'the poor man's Harvard' – that is, City College in New York, where he studied from 1935 to 1938. As the son of poor Eastern European Jewish immigrants, he was able to enroll at City College tuition-free. Soon he was actively involved with the 'New York Jewish intellectuals,' not all of whom were religious, but who all shared a certain ethos. They saw themselves as deracinated cosmopolitans, but at the same time as part of a widely dispersed intellectual family.

Bell describes himself as "decidedly religious." "My upbringing was very Jewish," he recalls, "and my native tongue was Yiddish. I attended a Jewish school (*kheder*), where neither teachers nor students spoke English. We translated from Hebrew into Yiddish." Still, religion for Bell is "not so much about God, as about the sacred. It's not a matter of ritual or orthodoxy. Religion for me is the holy and the tradition, something that sets limits and that you can't go beyond. I was particularly impressed by the fact that rabbis are not priests but teachers. What remains after the destruction of the temple? The sacred writings."

Our conversation took place in Cambridge, where Bell lives with his wife Pearl (younger sister of literary critic Alfred Kazin) near Harvard Yard. I hadn't seen him in over a year. His house now has a small addition, so that Pearl, who had a serious accident a few years ago, could move back in and receive round-the-clock treatment from two nurses.

The house is not only, as one would expect, full of books. Bell is also an art connoisseur and has collected Japanese and German Expressionist prints. He once described himself as a liberal in politics, a socialist in economics, and a conservative in culture. Does this self-characterization still hold? "Yes, certainly. I had recourse to this tripartite division because I don't regard society as a holistic system. You can be a radical in one area and conservative in another. I'm a liberal in politics because I believe in individual achievement and reward, in the idea of a just meritocracy. In economics, I'm a socialist, because community participation is important to me; everyone is entitled to a decent share of the available resources. And in art and culture I'm conservative, because I uphold values and traditions."

Bell's description of himself as a socialist makes him smile. He remembers a time when there were socialists everywhere at City College; many Stalinists were so argumentative that New York at the time was known as the most interesting city in the Soviet Union. The socialists at City College were abundantly self-conscious, returning manuscripts with the comment: "Tolstoy did it better." And in the midst of a political debate, one might hear someone say, entirely in earnest: "I know what Trotsky should do, and so do you. But does *Trot-*

sky know?" Daniel Bell was a socialist like the others, but, unlike most, he was never doctrinaire. How does he explain that?

"I was lucky. I became a socialist in reaction to the Depression. I saw people living in hovels and starving. Capitalism seemed to be on its last legs – so you became a socialist. Then I met Rudolf Rocker, who though born a Christian had learned Yiddish in order to edit the *Free Workers' Voice* (*Fraye Arbeter Shtime*), a Yiddish-language newspaper printed in Hebrew letters. Rocker gave me anarchist writings, and I read about the sailors in Kronstadt who, in 1921, went to Trotsky and demanded food supplies and the free elections they'd been promised. And Trotsky, the organizer of the Red Army, cried 'Insurrection!' and had the sailors shot. 'Kronstadt' then became a code word for withdrawal from the Communist Party. Some people had their 'Kronstadt' during the mass purges in the 1930s, others during the Hungarian uprising, and still others during the Prague spring. My Kronstadt was Kronstadt."

Like many of his college friends, Daniel Bell grew up on the Lower East Side of New York – in a milieu marked by persistent poverty if not by overwhelming misery. His generation did not come to socialism through dramatic conversion experiences but rather grew into it, as youngsters grow into the clothes of older siblings. Schoolchildren were already trade unionists and agitated from soapboxes for a more just society. In this spirit, the young Bell, speaking on soapboxes, memorized passages from Upton Sinclair's novel *The Jungle*. His reward – the astonished exclamations of passersby on the New York sidewalk, "How eloquent he is!"

At eighty-six, Daniel Bell has lost none of his youthful eloquence. According to the definition once devised in a City College cafeteria, a New York intellectual is someone who, after two minutes' preparation, can talk uninterruptedly on any subject whatsoever for at least a quarter hour. Bell needs no two-minute preparation, and one can listen to him for hours. He personifies a kind of American intellectual who, unlike his European colleagues, has never had any illusions about belonging to a 'socially free-floating intelligentsia.' Nor has he ever taken refuge in a concept of 'the inner life.' "With Roosevelt and the New Deal, there came into being a type known as the 'policy intellectual.' That was what I wanted to be: someone who understands something of the details of politics, and is interested in its everyday working. Friends of mine would say: the intellectual has to be critical. That was not enough for me. For me, the most important function of the intellectual was to take responsibility."

It was because of this 'ethic of responsibility' that Bell became a member of four government commissions and, in 1965, cofounded the journal *The Public Interest*, with his old friend Irving Kristol. Bell resigned in 1972, and was replaced by Nathan Glazer. Kristol became the intellectual forerunner of the neoconservatism that brought Richard Nixon and Ronald Reagan to the White House.

Bell likes to quote Irving Kristol's definition of the neoconservative as "a liberal mugged by reality." But he does not by any means number himself among the neoconservatives. "In 1972, the *New York Times* invited Kristol and me to contribute guest columns. They wanted us to explain why he supported Nixon and I supported McGovern. We wrote our commentaries, but at the last minute the *Times* could not print them. We still have the articles. Unlike Kristol, I didn't trust Nixon for a minute. But my

friendship with Irving Kristol was never compromised by our differing political views. Friendship has always been more important to me than ideology."

Bell's political differences with the neoconservatives – those who provide the ideological lining to U.S. policy at present – became clear in the course of our conversation: "I don't trust a politics geared to securing American hegemony. Cheney, Rumsfeld, and the rest – I don't trust them. They're obsessed with geopolitics." Still, Bell refuses to describe American hegemony as 'imperialism.' "There's a big difference between hegemonic and imperial. Hegemony, and, above all, military hegemony, the role of superpower – this role has fallen to the Americans. I once cited André Malraux, who had it right: An imperial style is something denied to Americans. Nixon aimed for it – without success. And Bush, too, with his 'Mission accomplished!' – his advisors came up with that. Our society is much too bourgeois to be able to cultivate an imperial style."

In speaking of members of the Bush administration, Bell frequently uses one adjective: "smart." Unlike most intellectuals, he has not believed that those who hold the reins in Washington today are blockheads. As a sociologist, he is careful not to underestimate the reelected president. "What drives George W. Bush is his faith. He's a born-again Christian and must be taken seriously as such. Compared to, say, Ronald Reagan, who in these things was very clever, Bush is no operator. I sometimes wish he *were* – then there might be some chance of changing him. But, no, he really believes in what he says. It's on this score that so many people misjudge him."

For over forty years, Daniel Bell has bemoaned the lack of *civitas* in the life of the modern democracies, the unwillingness of most citizens to make sacrifices for the community. He also predicted thirty years ago a resurgence of religious conviction in the world; he believes European society is only superficially secularized. And he has cited the observation of his friend Irving Kristol, that societies in the West are unprepared for major catastrophes. 9/11 was proof of that.

Asked if he was worried about anything in particular these days, Bell had this to say: "Optimism is a philosophy, pessimism a character trait. My character trait is pessimism. Jews continually have had cause for anxiety; that's part of our history. I'm a pessimist – there's always something that's got me worried. At the moment, it's the Israeli-Palestinian conflict, to which the American government is paying insufficient attention."

With this, our conversation came to an end – though not our evening together. We went to dinner at the Kebab Factory, a popular Indian restaurant in Cambridge. The table was small but the noise level high. Bell requested that the music be turned down a little. He wanted to sing me a song he'd written in his youth at City College. The song was called "The Old Bolshevik." He started up: "When I was a lad in Nineteen Six, / I joined a group of Bolsheviks." There were at least six choruses, and before long nearly everyone in the Kebab Factory had stopped eating. I thought of the passersby in New York who had once listened in amazement to the little soapbox orator; like them, one wanted to exclaim: "How eloquent he is!"

Jeri Laber

on torture

It has been well over a year since we first learned about the torture by American soldiers in Iraq's Abu Ghraib prison. Yet only low-level offenders have been tried and punished. Americans have been forced to confront difficult questions: Why were these abuses committed? Who is ultimately responsible?

As a human rights activist for the past thirty years, I have learned a lot about torture. My colleagues and I at Human Rights Watch have documented the use of torture in many parts of the world and have pressured offending governments to change their practices.

Torture is a gruesome subject. My friends and acquaintances, on the whole, have always shied away from discussing it with me because it was too upsetting. It was also too remote. Why concern themselves with gross abuses happening in far-flung parts of the world, about which there was little an ordinary American citizen could do? And anyway, don't those people – the torturers – have a distinct 'mentality' that enables them to torture? Nothing like that would ever happen here!

No wonder Americans were shocked and transfixed by the spectacle of American soldiers, ordinary men and women from towns across the United States, subjecting Iraqi detainees to painful and humiliating treatment – and recording the abuses on their camcorders with boastful glee. Who are these people? Who are their superiors? And who in our great democracy gave them permission to act as they did – with a sense of impunity, without any shame?

"Are you surprised?" friends asked me. My answer was yes . . . and no. On the one hand, I was surprised because such behavior goes against the basic principles of American democracy, against our belief in the rights of the individual and the sanctity of his or her person. I believe in those principles. They have made me proud to be an American.

Yet I am not surprised. Since September 11, our government has systematically chipped away at the guarantees that have kept our democracy sound – by refusing to apply the Geneva Conventions to detainees in Guantánamo, by using the U.S. Patriot Act to undermine our civil liberties at home, by saying that torture may be justified under certain circumstances because of our war against terrorism. In direct violation of international law and the Convention Against Torture, senior officials in the U.S. government stated that the presi-

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dent has the authority to set aside such laws in wartime. The administration has engaged in a deliberate policy of permitting illegal, coercive interrogation techniques. It has then tried to cover up or ignore reports of torture and other abuse by U.S. soldiers, in Afghanistan, in Guantánamo, and in Iraq.

There is no such thing as 'lite' torture. Once the rules are bent, or lifted, despicable acts like those at Abu Ghraib become possible.

It was a 1975 article about torture that started me on my human rights career. Though it may seem surprising today, the use of torture in more than 150 countries around the world was not widely reported back then, and the facts were not known to even the most thoughtful and concerned people. Shocked by what I read and convinced that I had to do something about it, I became part of the nascent human rights movement in the United States. In 1978, I became a founder and then the long-term executive director of Helsinki Watch, which grew to be Human Rights Watch, the largest human rights organization based in the United States.

I will never forget my first interview with a torture victim. In 1977, I met with an Iranian poet and professor who had been imprisoned and tortured in 1973 by the Shah's secret police, the dreaded SAVAK. He described beatings that tore apart the soles of his feet, threats to rape his wife and daughter, and a mock execution in which he thought he was about to die. He depicted torture chambers with iron beds to which prisoners were tied and "roasted." He spoke of whips and electric prods that shocked the chest and genitals. He described how torturers hung their victims upside down and raped them.

In the years that followed, I went on to interview hundreds of torture victims. In doing so, I learned to close off part of my feelings and not allow myself to fully imagine the experiences they described. Only then could I stay measured and disciplined in dealing with their terrible tales.

On my desk each day were dozens of grotesque torture descriptions. I had to pick those that I thought would be most effective for an article or a report, discarding others that were either not graphic enough or too ghastly for the average reader. It was a strange experience indeed to matter-of-factly edit a report, inserting the appropriate commas between sequential words like "beheaded, mutilated, and raped," while trying to ignore the import of the words themselves.

Most torture victims never fully recover from the experience. The most devastated are usually those who broke under torture and incriminated others. They live with intense pain – physical, emotional, and spiritual.

Other torture victims have amazed me with their strength to resist and rebound. There is a remarkable similarity in what they have to say:

"I could stand the pain because there was a part of me they couldn't touch."

"They tortured my body, but not my soul."

I have given a lot of thought to the psychology of the torturers, trying to understand how 'ordinary' people could commit such atrocities. Torture victims often describe their torturers as "family men" who left home each morning to do a job like everyone else. Occasionally, I also read reports by a few repentant torturers who had the courage to confess what they did and deplore their own actions.

Not every torturer is born a monster. Many are ambitious young people, recruited as soldiers into elite forces and

Note by Jeri Laber

specially trained for the dirty work in which they then feel trapped. Others, like those Americans in Iraq, are untrained and inexperienced, led to believe that they are serving a good purpose by 'softening up' prisoners for interrogation. They believe that their work is necessary in order to get information that will protect their country. But in the process of torturing other human beings, they destroy their own humanity and become overwhelmed with feelings of power that enable them to commit monstrous deeds.

Particularly instructive are the findings of Stanley Milgram, a Yale psychologist who, in 1974, attempted to prove that ordinary New Haven residents would follow orders to the point of administering life-threatening shocks to an innocent victim in the course of a supposed scientific experiment. While Milgram's research on obedience to authority may have been flawed, the results demonstrate the complexity of human behavior: people are not necessarily bad or good; the circumstances of the moment may influence their actions. Torturers, or potential torturers, may exist in every society: it is society's responsibility to pass laws to protect its citizens – sometimes from themselves.

Torture is destructive to all involved – to the victims, to the torturers, to those who ignore or deny what is happening. Lectures about what is right and what is wrong are not enough to stop it. Society needs principles, of course, but it also needs laws to protect its citizens. It needs strong institutions to enforce those laws and to punish all transgressors, including senior officials responsible for a high-level policy of abuse.

It is inexcusable for any government – and especially the government of the United States with its high moral precepts – to make exceptions to the laws against torture. The U.S. government should change its policy and conduct a serious, independent investigation of the deplorable events that have so tarnished our country's reputation and self-respect.

Robert F. Nagel

on the decline of federalism

In the last century, federalism dramatically declined in significance to the American public. One reason for this deterioration was the development of new individual rights, particularly as a result of the civil rights movement, when state sovereignty was closely associated with opposition to racial integration and, more distantly, with slavery itself. The expansion of government at the national level also reduced the importance of states. If states sometimes did fulfill their vaunted function as laboratories of democracy, as was true with welfare reform, ensuing reforms enacted at the national level soon eclipsed the states' contribution.

Most people have not mourned this loss of state authority. In fact, only a few years ago, sophisticated observers became alarmed at what they perceived as the Supreme Court's attempts to engage in a radical, even revolutionary, effort to revive the role of states in the federal system. Over the past three years, however, the clamor has begun to subside as the Court has issued several rulings that once again expand the scope of Congress's power and limit state immunity from that power. These recent decisions, together with a long-running series of cases that constrict state regulatory authority through the aggressive creation of individual rights, make it clear that federalism is not nearly as high a priority for the justices as their occasional states' rights rhetoric might suggest.

Yet a lack of public or scholarly interest in states is not the same as a lack of interest in decentralization. In fact, since at least the 1950s, considerable academic and journalistic concern has been expressed about the loss of political life at the local level. On both the right and left, many have called for the reinvigoration of neighborhood councils, school boards, and the various private clubs and associations that grow up around local governmental institutions. What makes this kind of decentralization appealing, of course, is that it provides the opportunity for direct, personal dealings among citizens and for decision making that is highly sensitive to local conditions.

These communitarian values have also attracted the justices of the Supreme Court. For instance, they have approved local financing of public education, de-

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spite the unequal funding patterns that result, because local taxation means local control. They have also urged district judges in school desegregation lawsuits to return authority over educational policy to elected school boards as soon and as fully as possible. Many other decisions – for example, favoring the autonomy of the Boy Scouts, the prerogatives of parents in shaping family life, or the participation of religious schools in voucher programs – acknowledge the importance of decentralized community life.

Political and cultural life at the national level, then, is naturally appreciated because it is visible, important, and dramatic, while political and cultural life at the local level is naturally appreciated because it is intimate and responsive. Associational life at the state level, poor gray thing, cannot regain the appeal it lost during the long history of racial conflict because it seems to be neither especially significant nor highly personal.

But what if the intermediate organizations constituting (and also surrounding) state governments enable local institutions and associations to remain healthy? In that case, the very structures that almost everyone neglects would be vital to the kind of social and political life that almost everyone values.

There are some commonsense reasons to think that federalism promotes local community life. As Theda Skocpol shows in her recent book, *Diminished Democracy*, local groups can affect national policies through state-level organizations. Moreover, leadership that circulates up and down through a federated system can become relatively sophisticated while staying connected to local communities. In fact, small associations and governments, while easy to romanticize, have many disadvantages if isolated. Without intermediate connections, they tend to be homogeneous, provincial, ingrown, and oppressive. Localism in America is so attractive precisely because we have had very few truly isolated communities; the norm, largely taken for granted, has been a federated political system that ensures various ties to wider communities and thus helps local institutions and associations to function in healthy ways.

This possibility finds some support in the fact that local associational life began its precipitous decline in the 1960s along with the waning of federalism. Skocpol observes that as the range of national undertakings widened during the civil rights revolution, organizations centralized, abandoning the membership-based structure previously so common and altering their lobbying tactics. Consequently, a national leadership class, cut off from any roots in local communities, emerged, replacing the rich associational life of federated organizations with direct mailings and single-issue advocacy.

But the damage to state-level organizations must have been more complex than a mere quantitative increase of power in Washington. The expansion of the power of the national government began, after all, long before the 1960s. What changed during the civil rights era was qualitative. It was during this period that the federal government began to determine not just industrial or commercial policies but moral issues of immediate significance in people's lives.

During the 1960s federal courts and bureaucracies began to control sensitive aspects of public education, including such issues as attendance policies and the place of religious subject matter and observances. They also began to have significant influence over mental health policies, police practices, contraception, defamation rules, standards for public decency, and so on. This extraordinary

set of initiatives was followed within a decade by the nationalization of policies on abortion, gender relationships, and family life. The extension of national power to such vital, and often intimate, issues came in the form, of course, of expanded individual rights.

The civil rights revolution, therefore, did far more than nationalize a new range of public policy issues. In some of the most crucial areas of human life, it severed individuals from their state and local governments. These governments, after all, were the institutions that rights were held against. In part, the states were disabled from regulating, and in part, they were actually delegitimized – seen as potential threats to the individual's happiness and freedom. State institutions became less important and, at the same time, vaguely sinister.

As a result, political discourse at the state and local level became more bland and insignificant, and less self-assured. Moreover, the repeated validation by national institutions of individuals' claims to autonomy and self-realization helped to unleash what the postwar prosperity had made possible: limitless demands for happiness and freedom from responsibility. This utopianism fueled dissatisfaction with constraints of all kinds, most especially those imposed as part of the traditional regulatory functions of state and local governments.

At the same time that the moral status of states was receding, reformist impulses found their outlet in Washington, D.C. Political control over some of the most interesting and significant issues had moved from local and regional arenas to the national stage. Of course, ambitious professionals could not be content to devote their energies as a part of federated organizations. Suddenly, there was the possibility of imposing reform – relating to the most significant areas of life – nationwide! Even if states had not been stripped of important regulatory authority and delegitimized, they would still have had the defect of limited jurisdiction. No longer would the antiquated system of federalism frustrate the utopian desire for uniform progress.

It is, then, only a small step from Skocpol's data to the hypothesis that what happened beginning in the 1960s was the eclipse of the moral status of state and local governments. When participation at intermediate levels of organizational life became less important and less morally attractive, local associational life suffered as a consequence.

If the decline of state governments is in fact linked to the deterioration of local associational life, reinvigorating the moral status of state governments may help to remedy the depletion at the local level. But is that solution practical? Maybe not, but we should approach the issue with the recognition that many of the alternative reforms suggested are not especially likely to be effective either. Modern communitarians tend to exhort people to join and participate; their concrete proposals often involve peripheral matters like national holidays and shaming rituals.

Nevertheless, we must acknowledge that the system of federalism in the United States is in many ways past the point of no return; indeed, the dynamics of centralization are now self-reinforcing. Strong identification with or attachment to a particular state, much less political loyalty, is now largely a thing of the past. It is also true that potent political, intellectual, and cultural trends underlie the moral eclipse of state governments.

We should consider a number of countervailing factors, however. First, the federated structure of American government has, needless to say, deep historical

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roots. Second, state governments, while often neglected and sometimes scorned, are still ongoing operations. They perform fundamental functions like taxation and moral regulation, and they provide important services such as education, transportation, and police protection. These functions, moreover, are carried out as a consequence of familiar political processes and institutions, and they are carried out by officials who utilize all of the signs and rituals that attend governmental status. Third, given the constitutional functions that states play in the operation of the national government, the quasi-sovereign, constitutive nature of state power is visible to all and difficult to eliminate.

In short, even if powerful and cumulating forces are working against any broad revival of state power, state governments remain significant institutions. The pivotal issue, therefore, is how much invigoration of status and power at the state level is necessary to help revive local associational life. If such a revival would require radical changes, such as a significant rollback of New Deal regulatory power or the dismantling of the technology that underlies the nationalization of political discourse, the widespread tendency to ignore federalism is understandable. But some degree of reinvigoration of community life may be realistic without wholesale changes in state power and prestige and even in the face of a continued decline in state power as a quantitative matter.

Some moderate qualitative shifts in the public attitude toward state governments are within the realm of possibility. After all, what changed with the civil rights campaigns of the 1960s was the way Americans perceived state governments. It is not entirely unthinkable that state governments might gradually regain some of the moral status and sense of connection that they lost, especially if a significant public reaction against the excesses of the rights revolution were to develop.

Another realistic change would be for scholars who value local associational life to give more attention to the relationships between that life and intermediate organizations at the state level. Without an accurate understanding of the preconditions for vigorous local associations, no reform will be effective. One of those preconditions may be state governments that are not discredited in the eyes of their citizens and not disconnected from organizational life at the local level. And, as for the Supreme Court, it does not need to lead a federalism revolution, but it might at least take into account more fully its own role in isolating and, therefore, diminishing local community life.

Letters to the Editor of *Dædalus*

On compromised work

November 22, 2005

To the Editor:

I write in response to the article "Compromised work" by Howard Gardner in the Summer 2005 issue of *Dædalus*. I read some months ago the much longer research paper that Gardner summarizes by Paula Marshall, his colleague on The GoodWork® Project.

My reaction to his comments about Hill & Barlow is the same as it was to Marshall's long report. I felt she did an excellent job of putting the history of Hill & Barlow's last few years in the broad context of the trends in law firms that have been going on now for several decades. Boston was the last major city to succumb to the drift towards ever larger and less personal organizations that look more like businesses than they do professions. Gardner/Marshall were also right in saying that the leadership of Hill & Barlow failed to see the changes coming when they should have and did too little in response once they did. But they don't get at the underlying reason why all this happened.

In the past, the great strength of law firms (and, indeed, of most professions) was their 'culture.' We understood this to mean the values a firm held and the degree to which they lived by them. Hill & Barlow was renowned for its culture. The firm lived, for example, by the old fashioned creed of "all for one, and one for all." And, while not always perfect in execution, Hill & Barlow had the reputation of coming closer to balancing personal and professional needs than any firm in town at that time. In this respect, other firms followed Hill & Barlow's lead.

In my view, the collapse of Hill & Barlow was the direct result of its culture, a culture so strong that it made it impossible for the firm to take drastic actions solely for business purposes that were in contradiction to their values. Even when information was put before them that should have raised alarms, most partners paid little or no attention. They couldn't believe that it would not turn around 'next year.'

For example, they shied away from separating unproductive partners on a large scale. Hill & Barlow just 'didn't do that' to its partners. And even though they tinkered around the edges from time to time, the firm could never bring itself to seriously alter its policy and procedures for approving associates for partnership. At Hill & Barlow, associates could be confident of being made partner as long as they were bright and did basically good legal work without regard to their overall fit into the department's profile or their capacity to attract new clients. In one year in the 1990s, the firm elected to partnership all seven associates who were eligible, an extraordinarily high number.

Gardner/Marshall lay special blame on Real Estate for the firm's collapse without providing support for that prop-

osition. They couldn't put forward any evidence because there isn't any. By the 1990s, Real Estate was by a substantial measure the strongest department in the firm. Sometime in 2000, the Real Estate partners became fully aware of how weak the finances of the firm were, and they had no confidence that there was a plan in place that could be implemented quickly enough to salvage the firm. Some members began considering options to move to other firms. If the senior partner in the department had not begun working to ensure that this extremely well-knit group could stay together, even if it meant outside Hill & Barlow, many of the department's younger partners would have walked immediately. In the end, the result would have been the same for the firm and, perhaps, a much worse financial result for all the remaining partners individually.

As executive director of the firm from 1990 until my retirement in 2000, I watched all the early trends unfold and was not surprised when the firm collapsed. (I was not interviewed for either of these articles.) This is not an instance in which blame should be placed anywhere. This was an instance in which an institution was condemned to die because of the very culture that had once made it great. Perhaps the end could have been managed less drastically, but apparently there were pressing financial reasons that led the partners to vote unanimously to close the doors when they did. So, the firm vanished with a bang instead of a whimper.

Hill & Barlow died because it refused, perhaps blindly, to sacrifice its classical values. It wasn't because of "compromised work." Some might call it the result of stupidity and unwillingness to face facts. But, perhaps, it was something of an heroic end for a grand institution that could no longer exist by its own standards in today's changed conditions. I like to think of it that way.

Simone Reagor

Simone Reagor was executive director of Hill & Barlow from 1990 to 2000. She received her doctorate in history from Oxford University. She is currently a management consultant, principally for Education Development Center, Inc., in Newton, Massachusetts.

November 28, 2005

Howard Gardner and Paula Marshall respond:

We appreciate Simone Reagor's comments on the sad demise of Hill and Barlow (H&B). However, our detailed study of this institution, carried out principally by Paula (see www.goodworkproject.org) and reported in Howard's synthesizing piece, yielded a different story and pointed to a different moral.

First, our sources of information. For the study, we contacted fifteen key, long-term partners, each of whom had been nominated by a knowledgeable partner or observer, and secured in-depth interviews from twelve partners. We also had comments from informed observers of the legal scene in Boston. Revealingly, the only group that resisted participation in this retrospective inquiry were individuals from the Real Estate (RE) group. When a draft of the paper was completed, we sent it to all interviewees, and secured comments from several of them; these comments were incorporated in the paper that was posted in September 2004. Far from being an idiosyncratic interpretation by outsiders, our study represented an emerging consensus among the chief participants, except for members of the RE group.

Second, what actually happened. We agree that the culture of H&B had been special – indeed exemplary – for decades, and its demise cannot simply be attributed to the greed of the RE group. We disagree, however, on what happened over the last thirty years and what could have been done about it. Cultures cannot be taken for granted; they need to be recognized as such, nurtured as much as possible, and – when survival is at stake – adjusted and revitalized so that the core values can be maintained. Some law firms have been able to do just this. Indeed, the Boston firm most often compared with H&B – Ropes and Gray – appears to have weathered the legal storms. One of their methods has been a long-term, self-renewing management team, which, among other things, determines salaries for the partners.

As detailed in Paula's paper, H&B is better described as a confluence of missteps, miscalculations, and missed opportunities, followed by a series of poor management practices and decisions. For sure, some members of the firm did try valiantly to save it and to preserve its core values. But they were not the majority, and the practices that had been in the DNA of partners for decades had begun to unravel by the 1980s. By any criterion except that of a completely marketized 'bottom-line' profession, the RE group did not have to leave the firm. They were making a very good living! Our interviews documented grave disappointment at the RE group and the secretive and preemptive way in which it operated – the diametric opposite of the picture intimated by Reagor. Moreover, in retrospect, many of the partners whom we interviewed came to believe that the firm could and should have been reconstituted and saved. They regretted the disappearance forever of a precious institution and the loss of jobs and benefits for dozens of long-term employees and retirees.

Finally, the issue of "compromised work." This is a term coined by Howard to delineate work that is not, strictly speaking, illegal but that undermines the core values of a profession. It is a question of judgment whether to apply the same term to the sins of commission by Jayson Blair at the *New York Times* and by the accountants of Arthur Andersen with those of the legal community at H&B. In view of the avowed high standards of the legal profession in general, and the fabled reputation of H&B, we conclude that an indeterminate number of the partner/managers did not live up to pivotal responsibilities: in particular, placing service to the profession above personal gain; ensuring that core professional and institutional cultural values are preserved and passed on to young associates; assuming responsibility for the welfare of paraprofessionals; participating in the management process (as faculty do in university governance), making adjustments in management when necessary; and, finally (if colloquially), not abandoning the ship. Instead, in Reagor's telling phrase "most partners paid little or no attention."

Simone Reagor may continue to believe in the romantic picture of H&B. We sought to document a principal conclusion of the GoodWork project: a healthy dosage of realism is essential if key professions and institutions are to survive in the current American climate.

Poetry for nonpoets

August 29, 2005

To the Editor:

May I explain my submission? The poem that appears in each issue of *Dædalus*

is often written by a recognized composer, sometimes with several books of poems to his or her name.

There is a class of members of the Academy who never read published poems anymore. I would like to capture their attention.

At the head of the page where my submission appears perhaps you could substitute "Jingle" as an indicator that the contribution is not a serious poem. This might get the attention of the class of members at which I am aiming.

Jingle

Alarming

The one-l lama,
He's a priest.
The two-l llama,
He's a beast.
And I will bet
A silk pajama
There isn't any
Three-l lllama.

– Ogden Nash

Ogden Nash once said to me
My poem is lovely as a tree.
I said to him, It's rather trendy
But outdone by my daughter Wendy.
Yes, she can count a little higher
A three-ell lama is a fire.
Though as a rule she's not a sinner
She eight-ell lama for her dinner;
And once when I was in Peru
I eight-ell lama in a stew!

– Ronald N. Bracewell, AO

Ronald N. Bracewell, AO*, a Fellow of the American Academy since 2002, is L. M. Terman Professor of Electrical Engineering Emeritus at Stanford University. In 1998 he was named Officer of the Order of Australia (*AO*).*

Inside back cover: Old before his time, the poet in middle age – Walt Whitman, c. 1860. See Caleb E. Finch on *Aging, inflammation & the body electric,* pages 68 – 76: "One hundred and fifty years after Whitman sang 'the body electric,' we can find in Whitman's fate some clues to the nature of aging." Photograph © Corbis.

coming up in Dædalus:

on the humanities	Patricia Meyer Spacks, Steven Marcus, Andrew Delbanco, Pauline Yu, Gerald Early, Anthony Grafton, Thomas Crow, Jack Balkin & Sanford Levinson, and Dagfinn Follesdal & Michael L. Friedman
on body in mind	Antonio & Hanna Damasio, Jerry Fodor, Carol Gilligan, Gerald Edelman, Jorie Graham, Raymond Dolan, Arne Öhman, Mark Johnson, Jacques d'Amboise, and William E. Connolly
on identity	Akeel Bilgrami, Wendy Doniger, Amartya Sen, Stephen Greenblatt, Kwame Anthony Appiah, Sydney Shoemaker, Joseph Koerner, Susan Greenfield, David A. Hollinger, Claudio Lomnitz, Carol Rovane, Todd E. Feinberg, Ian Hacking, and Courtney Jung
on nonviolence & violence	William H. McNeill, Adam Michnik, Jonathan Schell, James Carroll, Breyten Breytenbach, Mark Juergensmeyer, Steven LeBlanc, James Blight, Cindy Ness, Neil L. Whitehead, and Mia Bloom
on sex	Joan Roughgarden, Terry Castle, Steven Marcus, Claudia Goldin, Brian Charlesworth, Elizabeth Benedict, Wendy Doniger, Lawrence Cohen, Anne Fausto-Sterling, Catharine MacKinnon, Tim Birkhead, and Margo Jefferson
on capitalism & democracy	Joyce Appleby, John C. Bogle, Lucian Bebchuk, Robert W. Fogel, Jerry Z. Muller, Peter Bernstein, Richard Epstein, Benjamin M. Friedman, John Dunn, and Robin Blackburn
on life	Anthony Kenny, Thomas Laqueur, Shai Lavi, Lorraine Daston, Paul Rabinow, Michael S. Gazzaniga, Robert George, Robert J. Richards, and others

plus poetry by Peg Boyers, Kevin Carrizo di Camillo, John Kinsella, Charles Simic, Lawrence Dugan &c.; fiction by Adam Braver, Dorian Gossy &c.; and notes by Michael Cook, Norbert Schwarz, Joel F. Handler, William B. Quandt, William Galston, Richard Morris, Robert J. Sharer &c.

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