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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.

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;

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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-

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. 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.

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.

‘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.” 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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Beyond 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 foregoing 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.  

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. 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.

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

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.

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-


6 Aaron and Reischauer, Countdown to Reform.
surgery 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 not 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 increasingly 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. 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-
continue to increase, though by how much remains highly uncertain. 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. 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. 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. 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. 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.

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.


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.

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


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.
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 sur-
vived into the eighth decade of life.
Those few individuals who successfully
avoided the myriad causes of adult
mortality – principally, infectious dis-
eases, trauma, and cardiovascular fail-
ure – were expected to face a steady at-
trition of their most human qualities:

memory, reasoning, judgment, abstraction,
and language. In the popular mind,
and even among scientists and philoso-
phers, the idea that great age inevitably
brought about an inability to think clearly
was widely accepted. But intensive re-
search into the pathology and biochem-
istry of the aging brain during the last
few decades has revealed that specific
diseases cause major impairment of cog-
nition late in life and that the process
of aging per se results only in relatively
subtle changes in certain mental func-
tions.

This reinterpretation of the nature of
the aging mind has profound implica-
tions on both the personal and societal
levels. In contrast to the assumption in-
herent 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 be-
come 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 multi-
ple small strokes (multi-infarct demen-
tia), 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-
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. 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

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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. Overall, the aged brain tolerates relative-
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 β-protein (Aβ), 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 Aβ) 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 Aβ 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 Aβ 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 Aβ 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 Aβ. In addition to the fact that APP molecules give rise to the Aβ 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 Aβ 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 Aβ protein in cerebral blood vessels. In 1990, scientists discovered that a mutation in the APP gene that changes a single amino acid within the Aβ region of APP was responsible for this rare disorder, demonstrating for the first time that
mutations in APP could cause Aβ 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 Aβ 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 Aβ region. Tellingly, geneticists did not find any AD-causing mutations away from the Aβ 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 Aβ fragment.

As these genetic findings were emerging, a major biochemical discovery was made: all cells normally produce the Aβ peptide throughout life. Thus, Aβ 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 Aβ in their brains.

Putting together these two key observations – that healthy cells continually make Aβ 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 Aβ production. Scientists could now study many details of the production and metabolic fate of Aβ in simple cell models. They could also use such cells to screen large libraries of drug-like molecules and pinpoint compounds that lower Aβ 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 Aβ protein in brain regions important for memory and cognition. The practical outcome has been to encourage scientists to find ways to lower Aβ levels in humans.

Still, there are many unanswered questions about the Aβ hypothesis. What causes the imbalance in Aβ 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 Aβ levels? Does the Aβ peptide begin to aggregate inside the neuron before the Aβ 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 Aβ 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,

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 Aβ plaques with Aβ antibodies. This has been accomplished in two ways: either actively vaccinating the mice with synthetic Aβ so that they gradually generate their own Aβ antibodies, or passively administering laboratory-made Aβ 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 Aβ 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 Aβ 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 Aβ-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 Aβ levels, and a special brain imaging procedure like the emerging ‘amyloid scans’ that employ an injected chemical agent to visualize one’s cerebral Aβ burden. Such a multi-component 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 workforce 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.
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-


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.\(^9\)


\(^9\) 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.

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.

Depending on these reforms, Social Security remains mostly pay-as-you-go in its...
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.\(^\text{10}\) Were the new monies spent

\(^{10}\) Peter Diamond, “Social Security, the Government Budget and National Savings,” un-
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.

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**Figure 2**
Social Security’s Revenues and Outlays

![Graph showing Social Security’s Revenues and Outlays](image)

Source: Social Security Administration.

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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.\(^\text{11}\) 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.\(^\text{12}\) 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\(^\text{13}\) – 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 the indefinite future.

\(^{11}\) 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.

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. 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 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 prevent the trust fund from disappearing. Without raising taxes, we would eventually exhaust the trust fund.

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 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. But the most important weakness of this and other traditional measures of Social Security’s finances is


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


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. 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, 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-

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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.


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. 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

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. 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.

The Congressional Budget Office estimates that every dollar transferred from future to current generations reduces private savings by zero to fifty cents. 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. 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


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. 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 over-spending 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.” 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.

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

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. 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.

Legally, however, debt held by the public is a legal liability that the government must honor unless it declares bankruptcy. 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


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.