Aging, Health, and Longevity in the 21st Century

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Still In Search of Methuselah

In 1825, British actuary Benjamin Gompertz (1825) made an important observation about human mortality; he suggested that a law of geometric progression characterizes the rise in death rates as we grow older. With the risk of death doubling about every 7-to-8 years following puberty—a characteristic of human mortality that appears never to have changed—it is easy to understand why Gompertz and many other scientists since then have concluded that some biological "force" limits how long we are capable of living. The question for most scientists now is not whether there is a limit to life, but rather, have we approached it?

When Social Security was created in the United States in 1935, actuaries responsible for making official government forecasts believed that a limit to life existed, the rise in life expectancy observed since the turn of the century soon would decelerate, and the number of people drawing from this newly created age entitlement program would plateau at 20 million by the end of the century (for a summary of these forecasts, see Olshansky, 1988). Driving these assumptions was a belief in the presence of a biological limit to life (influenced by a passage in the Old Testament suggesting that three score and ten is the normal duration of life), and that humans were approaching that limit.

Other population scientists have shared this belief in limits (although not necessarily its religious origins), leading them to predict that life expectancy at birth for men and women combined would never exceed approximately 85 years (Bourgeois-Pichat 1978; Fries 1980). It is worth noting that none of the scientists involved in forecasting human longevity ever have suggested that the gender gap in life expectancy would be eliminated. This means that the claim that there are "broken limits to life expectancy" because females in Japan may have a life expectancy above 85 years (Oeppen & Vaupel, 2002), is spurious.

In 1990, my colleagues and I set out to reverse engineer an answer to the question of how long humans could live by estimating the magnitude of the reduction in death rates required to raise life expectancy at birth to much higher levels (Olshansky, Carnes, & Cassel, 1990). We illustrated that as life expectancy at birth rises up to and beyond 80 years, it becomes increasingly more difficult to raise it further because of a phenomenon known as entropy in the life table (Keyfitz, 1977). The mathematical explanation for this phenomenon was that for populations where most everyone born is expected to live to older ages, a life saved or extended by any means at middle and older ages yields shrinking gains in life expectancy because the risk of death always rises exponentially, or nearly so, until very old ages. This means that a one-year gain in life expectancy at birth from 80 to 81 years, for example, is far more difficult to achieve (requiring much larger reductions in death rates) than the same one-year gain from 70 to 71 years (Olshansky, Carnes, & Désesquelles, 2001).

As life expectancy at birth for a population (men and women combined) approaches about 85 years, the magnitude of the reduction in death rates required to yield just one more year of life expectancy becomes prohibitive, but not impossible, to achieve. We demonstrated that although death rates are likely to decline significantly at older ages in the coming decades for some subgroups of the population, such an achievement (as important as it would be) would yield smaller fractional improvements in life expectancy as we approach a life expectancy at birth (for the entire population) of about 85 years. It would be incorrect, however, to associate with this view the conclusion that declines in death rates at older ages are no longer possible, or that investing in the elderly is not worthwhile (Vaupel, 1997).

The medical explanation for diminishing returns in life expectancy is known as competing causes (i.e., if the risk of death from one cause is

reduced, another lethal disease rises to the occasion). A biological explanation for diminishing returns that has emerged links the age-specific rise in mortality to the onset and length of the reproductive window of a species—known as the biodemography of aging (which originally appeared as a new scientific discipline in the following publications: Carnes & Olshansky, 1993; Carnes, Olshansky, & Grahn, 1996; Wachter & Finch, 1997). Taken together, the demographic, medical, and biological evidence supports the existence of a "soft" limit to the human lifespan (for men and women combined) in the range of 85-to-90 years unless some intervention comes online to alter the basic biological process of aging—a conclusion that has not changed in the last twenty years (Olshansky et al., 1990).

Our estimate of the upper limit to life expectancy met with considerable resistance from mathematical demographers who claimed that historical trends in life expectancy imply that a "limit" to longevity is not in sight, if in fact it even exists (Tuljapurkar, Li, & Boe, 2000; Vaupel et al., 1998; Wilmoth, 1997). Indeed, proponents of this mathematical approach to the question of upper limits to life insist that if they cannot find evidence for a limit in observed past demographic trends in life expectancy or mortality, then such limits either have not been approached or they do not exist. In a letter to *Science* as part of the debate about how long humans can live, one mathematical demographer went so far as to claim that there are "no demographic or biological reasons why death rates cannot decline to zero" (Wilmoth, 2001, p. 1611). For the uninitiated to this debate about human longevity, this is a backhanded way of saying that immortality is possible.

As the debate about the future of human longevity progressed, evidence for a limit to longevity began emerging in the biological and medical literatures (for a summary, see Carnes, Olshansky, & Grahn, 2003). In spite of this evidence, some mathematical demographers still adhere to the admonition that if *their* data do not reveal a limit, then it does not exist (Christensen, Doblhammer, Rau, & Vaupel, 2009; Vaupel, 2010). Perhaps those who maintain this view should consider the possibility that the evidence for limits to life first appear outside of their scientific discipline.

The presence of a limit as described here should not be thought of as an immutable force. Rather, it is a "soft" limit imposed by a biology that arose through evolutionary processes for other purposes, and which essentially ignore events that occur in later regions of the lifespan. It should be emphasized that longevity determination in humans (just like the fastest speed at which humans can run one mile) cannot be a direct product of Mendelian predetermination (Hamilton, 1966; Kirkwood & Holliday, 1979; Medawar, 1952), but it certainly can be a secondary effect.

Figure 1

What Determines Human Longevity?

In his 19th-century book *Human Longevity and the Amount of Life Upon the Globe*, Florens (1855, p. 1) asked, "What is the natural, usual, and normal duration of the life of man?" In a commissioned painting entitled "The Bridge of Life," which portrayed the force of mortality throughout the human lifespan (Figure 1), statistician Carl Pearson (1897) illustrated a crumbling abrupt end to the bridge with an elderly man approaching an abyss—implying that there is a biological limit to the duration of life that imposes its force at later ages. Throughout history, the greatest thinkers of every era not only speculated about the duration of life and whether there was a limit (for summaries of this history, see Austad, 1997; Hayflick, 1994), but many also devised what they believed were methods of modifying how long people

are capable of living (Gruman, 1966). Questions of this sort are no longer esoteric. How long we live as individuals and populations has important public policy implications (Olshansky, Goldman, Zheng, & Rowe, 2009), and some believe we are on the verge of scientific breakthroughs that could extend life even further (Butler et al., 2008; Miller, 2009; Sierra, Hadley, Suzman, & Hodes, 2009).

In order to understand why there are constraints on the duration of life, it is important to recognize and appreciate the evolutionary theory of senescence. This theory has been discussed many times in the literature (for a summary, see Kirkwood & Austad, 2000), so here I will present only a very brief summary.

At the heart of this theory are links between the force of extrinsic mortality (i.e., non-aging related causes of death such as predation and communicable diseases that kill early in life) and the timing of reproduction. In turn, there is a fundamental biological link between reproduction and length of life. According to evolution theory, the force of natural selection— the ability of selection to influence the distribution and frequency of alleles (which are various forms of the DNA sequence of specific genes) in subsequent generations—begins to decline rapidly once reproduction commences at puberty, approaching negligible levels at the end of the reproductive window (at menopause) (Charlesworth, 1994).

The force of selection is governed by the intensity and timing of extrinsic mortality. For example, species that face high extrinsic mortality early in life (such as mice that frequently end up on the dinner plates of many other animals) must reproduce early while animals that face low extrinsic mortality (such as humans) experience the luxury of slower growth and development and, importantly, delayed reproduction. Medawar (1952) suggested that natural selection operates not just on individuals, but also on all the genes in our bodies. As the force of selection wanes following the onset of puberty, harmful alleles accumulate in the gene pool because under normal living conditions there is no penalty for detriments to health emerging in older regions of the lifespan to which only a few members of the species normally survive (Hamilton, 1966; Kirkwood, 1977; Williams 1957).

Why do aging and death occur when they do? Using the poetic words of Medawar (1952, p. 13), aging is revealed "only by the most unnatural experiment of prolonging an animal's life by sheltering it from the hazards of its ordinary existence." In other words, aging becomes evident only when life is lived with regularity into the post-reproductive region of the lifespan where harmful alleles have an opportunity to be expressed, as is now the case for most people in the developed world. Empirical tests of these hypotheses have shown that the age trajectory of death is, in fact, a species-specific phenomenon that, as predicted from evolution theory, is calibrated to the onset and length of a species' reproductive window (Carnes et al., 1996; Rose, 1984). This means that whales, elephants, and humans—species that face low extrinsic mortality pressures and therefore experience an extended period of growth and development, a significant delay in puberty, and a longer time window within which reproduction can occur—live longer than species that reproduce early and for shorter time periods.

It is important to remember that the reproductive window is a genetically determined and fixed attribute that is established as part of every species' life history strategy that was in turn molded by the environment within which the species evolved. The rate at which we senesce, and its correlate, duration of life, are therefore inadvertent byproducts of genetically fixed programs for the early life events of growth, development, and reproduction. As such, a unitary programmed aging process is unlikely even to exist, and there can be neither death genes nor longevity genes that evolved under the direct force of natural selection. From this perspective, how long we live is under genetic control only indirectly, and duration of life should most appropriately be thought of as a product of evolutionary neglect, not evolutionary intent. This is what is meant by the concept of a "soft" limit to life.

Can We Control Our Aging Destiny?

A persistent concept that has emerged from the major world religions and has appeared repeatedly in legends from almost every culture dating back to antiquity is that humanity is in control of its own aging destiny (Gruman, 1966). Familiar examples include an immortal Adam in the Garden of Eden before his fall from grace, and biblical patriarchs like Methuselah who was said to have lived 969 years. The most common historical explanation for the loss of

immortality, the lack of perfect health, and the steady decline in human longevity since the time of the patriarchs has been that each new generation has adopted increasingly more decadent lifestyles.

Roger Bacon, an influential English philosopher and scientist of the 13th century was the first to popularize this view (Burke, 1998). He also believed, however, that the trend toward shorter lifespans could be reversed by invoking the "secret arts" of the past—namely, the adoption of more restrained lifestyles and the ingestion of foods and other substances believed to have life-extending properties. Thus, the perspective that aging and diseases are amenable to modification through changes in lifestyles, which is the basis for contemporary medical and epidemiologic views of chronic degenerative diseases, has its origins in thinking that extends back in time at least one thousand years, and perhaps as far back as the golden mean in Greek philosophy.

These persistent beliefs about aging and disease that have been passed down through time have spawned two other positions that continue to have a significant philosophical and practical influence on contemporary scientific views of mortality. The most important of these is the belief that aging and disease are unnatural and are, therefore, somehow avoidable—a view now popularized by the modern anti-aging industry that leads people to believe that aging and disease are our faults (Weiner, 2010). Naturally, purveyors of anti-aging "medicines" claim they have the cure for aging, and they're willing to sell it to us for a hefty price, as long as the cash-only transaction takes place outside of the vigilant eye of the FDA and the insurance industry (Weintraub, 2010). The second viewpoint is that the health and longevity consequences associated with perfection can be reclaimed through human actions.

These beliefs and the quest for the resulting longer lives also have become a central part of the paradigm of modern medicine and the effort of epidemiologists to understand how risk factors alter death rates. On the surface, this philosophy of personal empowerment is seductive. After all, people want to believe that they have some control of their own aging destiny. An evolutionary perspective suggests, however, that aging and many of the diseases that accompany it are not deviant departures from perfection, or even the consequences of moderately decadent lifestyles. Instead, they are primarily the consequences of operating our living machines beyond their biological warranty period (e.g., beyond the end of the reproductive window, and for some species, into a region of the lifespan where grandparents can contribute to reproductive fitness of offspring) (Olshansky, Carnes, & Grahn, 1998). Automobile owners are not surprised when their cars begin to break down after using them for several years; we should not be surprised that the same fate awaits our bodies when used for extended periods of time.

Thus, the romantic philosophy that people are empowered to control their own aging destinies becomes, in modern times, an ideology of personal blame. In effect, we are inappropriately held responsible for many of the diseases and disorders that we experience as we age, and more importantly, are led to believe that aging and the diseases that accompany it are largely avoidable. An evolutionary view leads to the realization that even though aging, disease, and death are not programmed into our genes, once the engine of life is switched on, our destiny as an aging animal is written in stone. Our bodies fail over time not because they were designed to fall victim to aging and disease at a predetermined age (Hayflick, 2000), or because of the acquisition of decadent lifestyles, but because they were not designed for extended use.

Is the Promise of Radical Life Extension a Mirage?[1]

A mirage is a false image that the human eye detects because of the unique way light rays are bent, often appearing as a body of water visible on the horizon. The brain interprets the image with stark reality, enough to fool even the most astute observer. People who see a mirage can detect what looks like movement and even pinpoint its exact location on the road ahead, but as each spot where the mirage appeared on the horizon is approached, it disappears—only to reappear at the same distance as before. And so it goes, repeatedly throughout the journey until the viewer finally realizes that the image was never real in the first place. I suggest that historical and contemporary claims about radical life extension are a recurring mirage artfully constructed by alchemists and prophets for centuries past and continuing today. Some prophets of immortality may have constructed this mirage with the goal of lining their pockets, but for many, the radical life extension mirage is real.

To gain an understanding of the immortality mirage, consider the following exchange that took place between a modern proponent of immortality and this author.

"We Will Be Able to Live to 1,000" by Aubrey de Grey (http://news.bbc.co.uk/2/hi/uk_news/4003063.stm)

Ageing is a physical phenomenon happening to our bodies, so at some point in the future, as medicine becomes more and more powerful, we will inevitably be able to address ageing just as effectively as we address many diseases today.

I claim that we are close to that point because of the SENS (Strategies for Engineered Negligible Senescence) project to prevent and cure ageing.

It is not just an idea: it's a very detailed plan to repair all the types of molecular and cellular damage that happen to us over time.

And each method to do this is either already working in a preliminary form (in clinical trials) or is based on technologies that already exist and just need to be combined. This means that all parts of the project should be fully working in mice within just 10 years and we might take only another 10 years to get them all working in humans.

When we get these therapies, we will no longer all get frail and decrepit and dependent as we get older, and eventually succumb to the innumerable ghastly progressive diseases of old age.

We will still die, of course—from crossing the road carelessly, being bitten by snakes, catching a new flu variant etcetera—but not in the drawn-out way in which most of us die at present.

So, will this happen in time for some people alive today? Probably. Since these therapies repair accumulated damage, they are applicable to people in middle age or older who have a fair amount of that damage.

I think the first person to live to 1,000 might be 60 already.

It is very complicated, because ageing is. There are seven major types of molecular and cellular damage that eventually become bad for us—including cells being lost without replacement and mutations in our chromosomes.

Each of these things is potentially fixable by technology that either already exists or is in active development.

'Youthful not frail'. The length of life will be much more variable than now, when most people die at a narrow range of ages (65 to 90 or so), because people won't be getting frailer as time passes. The average age will be in the region of a few thousand years. These numbers are guesses, of course, but they're guided by the rate at which the young die these days.

If you are a reasonably risk-aware teenager today in an affluent, non-violent neighbourhood, you have a risk of dying in the next year of well under one in 1,000, which means that if you stayed that way forever you would have a 50/50 chance of living to over 1,000.

And remember, none of that time would be lived in frailty and debility and dependence—you would be youthful, both physically and mentally, right up to the day you mis-time the speed of that oncoming lorry.

Should we cure ageing? Curing ageing will change society in innumerable ways. Some people are so scared of this that they think we should accept ageing as it is.

I think that is diabolical—it says we should deny people the right to life.

The right to choose to live or to die is the most fundamental right there is; conversely, the duty to give others that opportunity to the best of our ability is the most fundamental duty there is.

There is no difference between saving lives and extending lives, because in both cases we are giving people the chance of more life. To say that we shouldn't cure ageing is ageism, saying that old people are unworthy of medical care.

Playing God? People also say we will get terribly bored but I say we will have the resources to improve everyone's ability to get the most out of life.

People with a good education and the time to use it never get bored today and can't imagine ever running out of new things they'd like to do.

And finally some people are worried that it would mean playing God and going against nature. But it's unnatural for us to accept the world as we find it.

Ever since we invented fire and the wheel, we've been demonstrating both our ability and our inherent desire to fix things that we don't like about ourselves and our environment.

We would be going against that most fundamental aspect of what it is to be human if we decided that something so horrible as everyone getting frail and decrepit and dependent was something we should live with forever.

If changing our world is playing God, it is just one more way in which God made us in His image.

"Don't Fall for the Cult of Immortality"
by S. Jay Olshansky. (http://news.bbc.co.uk/2/hi/uk_news/4059549.stm)

Some 1,700 years ago the famous Chinese alchemist, Ko Hung, became the prophet of his day by resurrecting an even more ancient but always popular cult, Hsien, devoted to the idea that physical immortality is within our grasp.

Ko Hung believed that animals could be changed from one species to another (the origin of evolutionary thought), that lead could be transformed into gold (the origin of alchemy), and that mortal humans can achieve physical immortality by adopting dietary practices not far different from today's ever-popular life-extending practice of caloric restriction. He found arrogant and dogmatic the prevailing attitude that death was inevitable and immortality impossible.

Ko Hung died at the age of 60 in 343 AD, which was a ripe old age for his time, but Hsien apparently didn't work well for him.

The famous 13th Century English philosopher and scientist, Roger Bacon, also believed there was no fixed limit to life and that physical immortality could be achieved by adopting the "Secret Arts of The Past." Let's refer to Bacon's theory as SATP.

According to Bacon, declines in the human lifespan occurred since the time of the ancient patriarchs because of the acquisition of increasingly more decadent and unhealthy lifestyles. All that was needed to reacquire physical immortality, or at least much longer lives, was to adopt SATP—which at the time was a lifestyle based on moderation and the ingestion of substances such as gold, pearl, and coral—all thought to replenish the innate moisture or vital substance alleged to be associated with aging and death.

Bacon died in 1292 in Oxford at the age of 78, which was a ripe old age for his time, but SATP apparently didn't work well for him either.

Physical immortality is seductive. The ancient Hindus sought it, the Greek physician Galen from the 2nd Century AD and the Arabic philosopher/physician Avicenna from the 11th Century AD believed in it.

Alexander the Great roamed the world searching for it, Ponce de Leon discovered Florida in his quest for the fountain of youth, and countless stories of immortality have permeated the literature, including the image of Shangra-La portrayed in James Hilton's book *Lost Horizon,* or in the quest for the holy grail in the movie "Indiana Jones and the Last Crusade."

What do the ancient purveyors of physical immortality all have in common? They are all dead.

Prophets of immortality. I was doing a BBC radio interview in 2001 following a scientific session I had organised on the question of how long humans can live, and sitting next to me was a young scientist, with obviously no sense of history, who was asked the question: "how long will it be before we find the cure for ageing?"

Without hesitation he said that with enough effort and financial resources, the first major breakthrough will occur in the next 5-10 years.

My guess is that when all of the prophets of immortality have been asked this question throughout history, the answer is always the same.

The modern notion of physical immortality once again being dangled before us is based on a premise of "scientific" bridges to the future that I read in a recently published book entitled *Fantastic Voyage* by the techno-guru Ray Kurzweil and physician Terry Grossman.

They claim unabashedly that the science of radical life extension is already here, and that all we have to do is "live long enough to live forever."

What Kurzweil and others are now doing is weaving once again the seductive web of immortality, tantalising us with the tale that we all so desperately want to hear, and have heard for thousands of years—live life without frailty and debility and dependence and be forever youthful, both physically and mentally.

The seduction will no doubt last longer than its proponents.

'False promises'. To be fair, the science of ageing has progressed by leaps and bounds in recent decades, and I have little doubt that gerontologists will eventually find a way to avoid, or more likely delay, the unpleasantries of extended life that some say are about to disappear, but which as anyone with their eyes open realises is occurring with increasing frequency.

There is no need to exaggerate or overstate the case by promising that we are all about to live hundreds or even thousands of years.

The fact is that nothing in gerontology even comes close to fulfilling the promise of dramatically extended lifespan, in spite of bold claims to the contrary that by now should sound familiar.

What is needed now is not exaggeration or false promises, but rather, a scientific pathway to improved physical health and mental functioning.

If we happen to live longer as a result, then we should consider that a bonus.

Proponents of the immortality mirage often portray themselves as futurists who are just ahead of the curve. They claim that life expectancy soon will rise dramatically to thousands or even billions of years, physical immortality is on the horizon, and some people alive today will drink from the equivalent of a yet-to-be-discovered fountain of youth (de Grey & Rae, 2007; Kurzweil & Grossman, 2004).

One case for immortality is based on the premise that information technology will increase at an exponential rate, eventually leading to dramatic new technological advances (such as nanobots) that will wipe out all diseases and aging itself (Kurzweil & Grossman, 2004; 2010. Proponents of this view rely on the promise that "regenerative medicine" will engineer new or refurbished body parts that will lead to increases in life expectancy that occur at a faster pace than the passage of time itself (de Grey et al., 2002). Under futurist scenarios, physical immortality and eternal youth will be achieved for all of humanity in this century; people over age 85 will become indistinguishable (physically and mentally) from people at young and middle ages; old age as we know it today will cease to exist; and the world will become populated only by those who are physically healthy and mentally vibrant.

This mirage is extraordinarily appealing to the popular news media because it feeds on a deeply rooted fear of death and the long-held desire by every generation to believe that "our science" will lead us down the path toward immortality, or at least much longer and healthier lives. This is not an uncommon theme; Gruman (1966) documented the immortality mirage dating back several thousand years, noting that this vision of immortality and the belief that we could intervene in aging itself was previously known as prolongevity.

As appealing as this mirage may be, there is no scientific evidence to suggest it is real. For example, at the foundation of Kurzweil's vision of bridges to immortality is the view that it will be information technology (IT) that will drive radical life extension (Kurzweil & Grossman, 2010). Although there is no doubt that IT has been advancing exponentially in recent decades, there is no evidence provided by Kurzweil that links IT to how long people live. In fact, to the contrary, during the time when IT was rising exponentially in the latter part of the 20th century, human longevity was increasing arithmetically in some parts of the world, and actually declining in others (Olshansky et al., 2005; United Nations, 2004).

The concept of indefinite repairs to the human body is also appealing, but so far there has been no science provided to support de Grey and Rae's (2007) assertions that such technological developments are forthcoming, nor is there any scientific evidence provided to defend assertions that the first billion-year-old person is already alive, or that there is a 50 percent chance of regenerative medicine yielding immortality by 2040. This version of the radical life extension mirage is propped up by exaggerated claims, made-up life expectancy estimates, and fabricated time lines linked to how much money is thrown at transforming the mirage into reality.

Past proponents of the immortality mirage are all dead, having died at about the same ages as everyone else (Olshansky, 2010), and there is reason to believe the same fate awaits those who see and promote the same mirage today. It is unfortunate but perhaps inevitable that the news media is also hungry to believe the mirage is real (Weiner, 2010).

Will Treating Disease Extend the Period of Old Age?

The traditional medical approach to ameliorating modern chronic diseases has been to attack them individually, as if they were independent of one another. This approach follows from humanity's long-term experience with communicable diseases, where medical care was sought for one condition at a time as it arose. The application of this strategy to communicable diseases was a resounding success; it helped to deliver a rapid increase in life expectancy during the 20th century in today's developed nations. This public health triumph is known as the first longevity revolution (Butler, 2008). Although some communicable diseases have chronic effects on health (such as malaria and HIV), and others remain difficult to treat (including tuberculosis and most viral diseases), public health efforts to combat these diseases have made it possible for people in today's developed nations to live long enough to

experience chronic degenerative and neoplastic diseases that are now the dominant causes of morbidity and death.

The price humanity has to pay for our extended lives is a new and much more complicated relationship with disease. While prior successful efforts to combat communicable disease enabled many people to live full lives into extreme old age, comparable achievements made against chronic fatal diseases today will not have the same effect on either length or quality of life. The reason is simple: saving the life of a young person can add decades to lifespan, but saving the life of a person who has lived seven decades or more yields an incrementally smaller increase in lifespan (Olshansky et al., 1990).

The health status of the survivors is even more complicated. Remember: competing causes in aging bodies means that those saved from dying from one condition eventually will face an elevated risk of dying from something else. For some, life extension through disease reduction will add healthy months and years, but others will be exposed to highly disabling conditions (such as Alzheimer's disease, diabetes, and osteoarthritis) for a longer portion of their lifespans than would otherwise be the case. In fact, the disease-specific model may very well lead to an extension of the period of old age (Butler et al., 2008). In spite of considerable time and resources invested in the analysis, prevention, or cure of single diseases, nearly all of the diseases and disorders experienced by middle-aged and older people still show a near exponential rise in the final third of life. The reason is that the risk of death from diseases might decline through behavioral modification and advances in medical technology, but aging marches on unaltered by these interventions.

In this regard, it is important to acknowledge the fundamental differences between disease and aging (Carnes, Staats, & Sonntag, 2008 Hayflick, 2004). Although age-associated changes in the body produce an increased risk of disease, the reverse is not true. Reducing the risk of disease has no influence on biological aging. Thus, if a population is saved with increasing efficiency by advances in technology that extend life by reducing the risk of disease, those saved will live into increasingly older regions of the lifespan where aging continues to take its toll on body and mind. Life extension achieved in this way could extend old age by exposing survivors to the high-risk conditions of frailty that are common, and currently largely immutable, near the end of life—the very outcome that medicine and public health practitioners are trying to avoid.

Decelerated Aging: A New Paradigm for Health Promotion and Disease Prevention in the 21st Century

Given the complicated relationships that have emerged between rising life expectancy, health, and disease in long-lived populations, scientists and geriatric physicians suggest that the primary goal of medical technology should not be the exclusive pursuit of life extension, but rather, the lengthening of the period of youthful vigor (Butler et al., 2008; Rae et al., 2010). Although efforts to combat disease should continue, one way to protect against an unwanted prolongation of old age and simultaneously extend the period of healthy life is to pursue the means to modify the key risk factor that underlies almost everything that goes wrong with us as we grow older—aging itself. We have referred to this shift in approach to public health as the Pursuit of the Longevity Dividend (Butler et al.; Olshansky, Perry, Miller, & Butler, 2006). The logic behind the Longevity Dividend is as follows:

1) Population aging is a demographic certainty, producing significant increases in the number of very old people in the coming decades even if life expectancy remains constant;

2) Attacking one disease at a time as if they are independent of each other ignores the presence and importance of their common risk factor—aging. This means life expectancy increases will decelerate, even if death rates at older ages decline;

3) Reducing the risk of disease does not influence the pace of biological aging;

4) Life extension achieved through disease reduction may produce short-term gains in length and quality of life for some, but for others (perhaps many) this could lead to an extension of old age;

5) The time has arrived for a supplementary health promotion and disease prevention paradigm based on a concerted effort to slow biological aging;

6) Successful efforts to slow aging simultaneously would postpone all fatal and non-fatal disabling diseases, producing gains in health and longevity equivalent to cures for major fatal diseases;

7) The extension of healthy life will yield economic and health dividends for most current and all future generations—referred to as the "Longevity Dividend;" and

8) The decision to pursue the scientific means to slow aging does not require that we know, in advance, which of the current ideas about mechanisms affecting the rate of aging are most likely to produce effective interventions.

Medical institutes and public health professionals across the globe are dedicated to combating the causes and consequences of heart disease, cancer, stroke, and myriad other fatal and disabling conditions that plague humanity, and many people are alive today because of their heroic efforts. These battles need to continue. However, the line of reasoning supporting an attack on aging requires both a population- and biologically based view that forces us to step back for a moment in order to examine the foundation of our beliefs about what happens to our bodies as we grow older.

The underlying premise of the Longevity Dividend is controversial, in part because our modern world is entrenched in a one-disease-at-a-time model applied to bodies that no longer operate that way. Although it is acknowledged that positive changes in behavioral risk factors can alter the expression of disease, and medical technology can continue to manufacture survival time, the case for the Longevity Dividend requires us to appreciate the profound and currently immutable influence of biological aging on the age-dependent expression of disease. Our battle with death inevitably will fail (a view that mirage makers reject), but proponents of the Longevity Dividend contend that death is not where the battle lines should be drawn. The successful pursuit of an extension of youthful vigor at all ages, no matter how much longer we live as a result, is the goal, and the only way to get from here to there is to recognize that the root of almost all that goes wrong with body and mind with the passage of time is due to biological aging. The time has arrived to recognize radical life extension as a distracting mirage, and get on with the business of extending healthy life by initiating a new paradigm that is focused on slowing the biological processes of aging.

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Endnote

1. The idea of referring to this view of immortality as a mirage originated from Dr. Leonard Hayflick, who in his time has seen more than his fair share of exaggerated claims about radical life extension (Olshansky & Hayflick, in press).

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