The Likelihood and Consequences of "Living to 100"

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Abstract

There is a common belief that it would be a universal good to discover how to slow or stop the aging process in humans. It guides the research of many biogerontologists, the course of some health policy leaders and the hopes of a substantial fraction of humanity.

Yet, the outcome of achieving this goal is rarely addressed despite the fact that it would have profound consequences that would affect virtually every human institution.

In this essay, I discuss the impact on human life if a means were found to slow our aging process, thus permitting a life expectancy suggested by the title of this conference, “Living to 100.” It is my belief that most of the consequences would not benefit either the individual or society.

Introduction

The belief that we have found, or will soon find, a means for arresting or slowing the aging process has been a part of human thought throughout recorded history. The oldest written record—from 1600 B.C.—is an Egyptian papyrus called, “The Book for Transforming an Old Man into a Youth of Twenty.” The ointment described for this purpose was “…found to be effective many times.”

One would have thought that after 3,600 years of unfulfilled promises, the public would have learned how to recognize the promoters of anti-aging snake oil—but they have not. Today, at least in the United States, this second oldest profession is a booming multibillion dollar industry. This industry has received indirect support even from some legitimate scientists who, in the last 50 years, have announced that human longevity will soon increase dramatically (Hayflick 2002; Underwood, Bartlett and Hall et al. 2009). All have been wrong.

For millennia, the belief that the discovery of a method for increasing human longevity has always been 20 years in the future.

Today, the conviction that aging will soon be slowed or stopped is more popular than it has been for decades because we think we now understand so much about biological processes. This view is simply the most recent iteration of the identical reasoning that has been promoted for centuries. One of the most popular recent pronouncements came from the leaders of the Human Genome Project, who proclaimed that their success in sequencing human DNA will, among other accomplishments, soon result in our ability to intervene in the process of aging. One leader, Dr. Francis Collins, now director of the National Institutes of Health, told the Washington Post in 2000 that within 30 years, we will know all the genes involved in the human aging process (McCarthy 2000). In the subsequent 10 years, we have not identified a single gene involved in the human aging process.

As it has been for millennia, the discovery of a method for increasing human longevity is still 20 years in the future. Dr. Collins’ failed prognostication will be realized when he understands there are no genes that carry instructions to make us age.

The Human Genome Project leaders made their *ex cathedra* pronouncements because, like many others, they fail to understand the distinction between two fundamental phenomena—aging and longevity determination. This distinction will be discussed subsequently.

Nevertheless, and in spite of these misunderstandings, we have succeeded in increasing human life expectancy but not life span. The increase, most of which has occurred in the last century in developed countries, was not caused by discovering how to intervene in either the aging or longevity determining processes. It occurred entirely because of our implementation of better public health policies and the resolution of most deaths caused by infectious diseases that mainly impacted the young.

It has always been the elimination of causes of death appearing on death certificates that has increased life expectancy and not interventions in the aging or longevity determining processes. We are reaching the end in respect to the remaining number of major causes of death that might be postponed or eliminated. Other than deaths attributable to accidents that will never be entirely eliminated, the resolution of deaths caused by the three leading causes (cardiovascular disease, stroke and cancer) can only increase life expectancy to about 93 years (Anderson 1999; Hayflick 2007).

The rate of increase in human longevity for the entire U.S. population from 1900 to 2005 has dropped steadily. The rate of increase for the 25-year period from 1900 to 1925 was 5.6 months per year. From 1925 to 1950, it was 4.4 months per year. From 1950 to 1975, it dropped to 2.6 months per year, and, from 1975 to 2005, it dropped further to 2.2 months per year. All of the increase in life expectancy that has ever occurred can be attributed to the resolution of causes of death attributable to infectious disease, pathology, famine, and violence or accidents both from manmade or natural causes.

The only remaining ways in which life expectancy might be increased to 100 is to either intervene in the biology of aging or the biology of longevity determinants. However, the consequences of these interventions are fraught with unintended consequences.

Definitions are Critical

To discuss further the likelihood of “Living to 100” by manipulating fundamental biological processes, it is essential to distinguish between these three phenomena—aging, age-associated diseases and longevity determination. The failure of biogerontologists to reach a consensus on the definition of these key terms and others in the field has been, and still is, a major impediment to the correct interpretation of experimental results, the proper allocation of research resources and the establishment of the nation’s health policy (Hayflick 2007; Hayflick 2004a; Hayflick 1995; Hayflick 2003).

In this essay, I will define three key terms as I see them in order to increase the likelihood that readers will understand the concepts to be discussed.

There are four aspects of the finitude of life (aging, longevity determinants, age-associated disease and death). Although there is controversy about the definition of death as it occurs in cells, organs and the whole animal, it will not be defined because it is not a consideration in this essay.

Aging

The obvious premise must be accepted that the survival of a species depends on a sufficient number of members living long enough to reproduce and, if necessary, to raise progeny to independence. In those animals that age, that phenotype becomes manifest only after reproductive maturation.

Age changes can occur in only two fundamental ways: either by a purposeful program driven by genes or by stochastic or random events.

It is a cornerstone of modern biology that a purposeful genetic program drives all biological processes that occur from life’s beginning until reproductive maturation. Once reproductive maturation is reached, thought is divided in respect to whether the aging process results from a continuation of the genetic program or whether it occurs by the accumulation of dysfunctional molecules. Yet, there is no direct evidence that genes drive age changes—a claim made by some because of the failure to distinguish age changes from longevity determinants.

The aging phenotype is expressed after reproductive maturation in animals that reach a fixed size in adulthood and is driven by random events that produce an excess of dysfunctional molecules that are beyond the ability of repair processes to correct them. The accumulation of dysfunctional molecules leads to dysfunction at higher levels of organization (cells, tissues and organs) until it becomes expressed in the whole organism.

No gene that codes for a universal biomarker of aging has been found. Analogously, inanimate objects also require no instructions to age. Evidence for the belief that aging is a random or stochastic process is that 1) everything in the universe

changes or ages in space-time without being driven by a purposeful program, 2) there is no direct evidence that age changes are governed by a genetic program, and 3) there is a huge body of knowledge indicating that age changes are characterized by the accumulation of dysfunctional molecules.

The common denominator that underlies all causes of aging is change in molecular structure and, hence, in function. It is caused by the intrinsic thermodynamic instability of complex biomolecules, or the manifestations of the Second Law of Thermodynamics. Entropy increase was, until recently, dismissed as a cause of biological aging because biological systems are open. The recent re-interpretation of the Second Law states that "Entropy is the tendency for concentrated energy to disperse when unhindered regardless of whether the system is open or closed. The 'hindrance' is the relative strength of chemical bonds." (Lambert 2009)

The prevention of chemical bond breakage until reproductive maturation is the *sine qua non* for the maintenance of life and species continuity. This is the role of longevity determinants or maintenance systems that ultimately also suffer the same effects of the Second Law as do their substrate molecules.

Thus, biological aging can be defined as the random, systemic accumulation of dysfunctional molecules that exceeds repair capacity. Dysfunctional molecules occur throughout life, but in youth the balance favors the body's enormous capacity for repair, turnover and synthesis; otherwise, individuals would not live long enough to reproduce and the species would vanish. After reproductive maturation, the balance shifts slowly in favor of irreparable, dysfunctional molecules, including those that compose the maintenance systems themselves. Then the myriad decrements that produce the aging phenotype are revealed. This accumulation of dysfunctional molecules increases vulnerability to age-associated diseases.

Blueprints contain no information instructing a car how to age yet, in their absence, molecules composing the car dissipate energy producing structural and functional losses that we recognize as age changes. Analogously, the genome also does not contain instructions for aging because, like the car, instructions are unnecessary to drive a spontaneous process.

Longevity Determinants

The second aspect of the finitude of life is longevity determination—a completely different process from aging.

Longevity is determined by the length of time that the synthesis, turnover and repair processes can maintain the biologically active state of their substrate molecules. This process is governed by the genome.

Unlike the stochastic process that characterizes aging, longevity determination is not a random process. It is governed by the enormous excess of physiological reserve produced until the time of reproductive maturation and evolved through natural selection to better guarantee survival to that age. Thus, the determination of longevity is incidental to the main goal of the genome, which is to reach reproductive maturity.

Longevity determination is an entirely different process from aging and is independent of it. One might think of longevity determination as the energy state of molecules before they incur age changes. This energy state addresses the question: “Why do we live as long as we do?”

One might think of aging as the state of molecules after they have incurred irreparable damage leading to the aging phenotype. This condition addresses the question: “Why do things eventually age, change or go wrong?”

Aging is a catabolic process that is chance driven. Longevity determination is an anabolic process that, indirectly, is genome driven. They are opposing forces.

The genome directs events until reproductive maturation, after which the aging process dominates. Thus, the genome only indirectly determines potential longevity by governing the levels of excess physiological capacity, repair and turnover. No specific genes determine longevity but, collectively, they all govern aspects of biological processes that increase the likelihood of survival to reproductive maturity. The variation in excess physiological capacity, repair and turnover governs the differences found in the longevity both within and between species.

The many popularized studies with invertebrates that have led to the view that genes are involved in aging have not revealed a reversal or arrest of the inexorable expression of dysfunctional molecules that is the hallmark of aging. Where these studies have revealed greater longevities, it is because the determinants of longevity have been manipulated well before the aging process begins. None of these studies using invertebrates has demonstrated that the manipulation of genes has slowed, stopped or reversed the aging process. Experiments on invertebrate “aging” usually have as their end point all causes of mortality. That end point tells us nothing directly about aging. It can tell us something about longevity determinants.

Age-Associated Diseases

The third and last of the four aspects of the finitude of life to be defined here are age-associated diseases. The distinction between the aging process and age-associated disease is rooted in several practical observations:

Unlike any disease, age changes 1) occur in every metazoan that reaches a fixed size in adulthood; 2) cross virtually all species barriers; 3) occur in all members of a species only after the age of reproductive maturation; 4) occur in all animals protected by humans even when that species probably has not experienced aging for thousands or even millions of years; 5) occur in virtually all animate and inanimate objects; and 6) have the same universal molecular etiology, that is, thermodynamic instability.

There is no disease or pathology that has all of these properties.

The key question is: “Why are old cells more vulnerable to pathology than young cells?”

Is it Beneficial to Have the Power to Slow or Stop the Aging Process?

Human life expectancy has increased by about 33 years in the last century but the maximum life span appears to have been fixed at about 125 years for the last 100,000 years. This latter belief is based on the remarkable relationship between maximum longevity in primates and their brain weight/body weight ratios (Sacher 1975).

Life expectancy at birth in developed countries has increased from about 45 years in 1900 to about 78 years in 2010. This 33-year increase in life expectation is equivalent to the increase in life expectation at birth that occurred from the time of ancient Rome until the year 1900. The astounding increase in life expectation in the last century will never occur again in developed countries because the causes of death from the offending infectious diseases can only be resolved once. It is the remaining chronic diseases—cardiovascular disease, stroke and cancer—that remain unresolved and that cause the majority of deaths that now occur in older people.

Slowing or Stopping the Process of Aging

To best explore the impact of an intervention that would slow or stop the rate of aging in humans, let us assume the simplest scenario in which a pill has been developed to do just that.

Furthermore, we will assume the pill is without unwanted side effects and it is administered daily or weekly. This thought experiment is offered in an effort to explore the consequences of having the power to intervene in our fundamental aging process.

Social Aspects

It is a certainty that the research and development costs for discovering such a pill will be enormous, putting it within reach of only the wealthiest and/or the most politically powerful citizens during the early years of its availability.

I am reluctant to believe these groups would be the most important people to be favored first with greater longevity. Within this group are certain to be many socially undesirable people including tyrants, dictators, murderers, those guilty of genocide and other assorted misanthropes. During the last century, we have ignored this moral dilemma in most developed countries where the advances in public health and medical treatments that resulted in the 33-year increase in life expectancy benefitted both the good and the evil. However, it is unlikely the rich and the poor benefitted equally even in developed counties. Examples of current inequalities, of the many that could be cited, are the difficulty of the poor in underdeveloped countries to have access to anti-HIV drugs, organ transplants and kidney dialysis, and that in the United States today, millions of poor people do not have access to the medical care available to the affluent. Good data exists showing that the economically and educationally advantaged citizens are, as a group, more long lived than the poor and uneducated (Hayflick 1995). It is likely that equivalent imbalances will exist with the availability of a pill that would slow the aging process or increase longevity.

Choices

Even when the pill became affordable for almost everyone, it is unlikely arresting aging would be an attractive option for the substantial part of the world's population who might be reluctant to use funds intended for food to lengthen their years to endure continuing poverty, oppression and/or chronic diseases.

The desire to arrest or slow the aging process is a luxury considered only in developed countries and by those who lead relatively happy lives. Those in underdeveloped or developing countries who might chose to seek greater longevity will usually be found only within the wealthy minority or politically powerful classes. Extending their lives might provide little, if any, benefit for the poor, sick or subjugated masses in those countries.

Timing

Most people would agree that the best time to have one's aging process slowed or arrested is when life satisfaction is greatest. How would one determine this time unless you have an opportunity to pass through that age in order to make an intelligent decision? Of course, once having passed through that age, how then would you reverse time to the earlier and better age? The only theoretical means for doing this is by engaging in time dilation by traveling close to the speed of light, but this does not seem to be a practical possibility. Absent time dilation, returning to a former happier time poses a predicament that is more science fiction than science fact. Without having a means of reversing time to a happier period, one must risk taking the pill at some arbitrary age in the hope the future will not be spent in a longer time enduring unhappiness or ill health.

Even if one's aging process could be arrested or slowed at what was perceived to be a happy time, it is a certainty that change will continue in the physical world, to say nothing of the human worlds of finance, local, national and international events, and all of the institutions in which one has a position or an interest. Thus, the certainty that change will continue to occur outside of a body whose aging has been slowed, or is ageless, might very well compromise what might have been chosen as a time when life satisfaction was thought to be best.

In a 2008 Gallup Poll survey of more than 340,000 people (both sexes) nationwide ages 18 to 85, the study found that "by almost any measure, people get happier as they get older." (Newport and Pelham 2009)

Furthermore, it was also found, Newport and Pelham wrote, that "people start out at age 18 feeling pretty good about themselves, and then, apparently, life begins to throw curve balls. They feel worse and worse until they hit 50. At that point, there is a sharp reversal, and people keep getting happier as they age. By the time they are 85, they are even more satisfied with themselves than they were at 18."

"Researchers also found that stress declines from age 22 onward, reaching its lowest point at 85. Worry stays fairly steady until 50, then sharply drops off. Anger decreases steadily from 18 on, and sadness rises to a peak at 50, declines to 73, then rises slightly again to 85. Enjoyment and happiness have similar curves: they both decrease gradually until we hit 50, rise steadily for the next 25 years, and then decline very slightly at the end, but they never again reach the low point of our early 50s."

An example of the increase in happiness experienced by some as they age is to consider the lifestyle of hundreds of thousands of septuagenarians, octogenarians and even older people who today will tell you that this is the happiest time of their lives. They would not have chosen to have had their aging process arrested at an earlier age, which would have resulted in more years working, child rearing or conducting other activities they found onerous. They would have been denied for a longer time the contentment of retirement, travel, freedom from child-rearing responsibilities and

unlimited time to pursue other interests. Hundreds of these folks who drive their recreational vehicles from summers spent in Canada to winters spent in Florida would not want to trade more working years for more leisure time.

On the other hand, there are just as many people who at these later ages are sick, poor, abused or neglected and who would not want their aging process slowed or stopped. Those with some forms of dementia might not even understand what the pill does.

If the determinant of when to take the pill is the time when you are free from stress and worry, then information obtained in another recent Gallup Poll will be of interest.

In a second analysis (Newport and Pelham 2009) f more than 650,000 (both sexes), a Gallup Poll conducted in 2008 and 2009 shows that "worry" is a much more common emotion among young and middle-aged Americans than among seniors. Well over a third (37 percent) of those in their 40s report having experienced worry "a lot of the day yesterday." This figure drops to about 23 percent among those in their late 60s and drops further to 15 percent among those 91 and older (Figure 1).

In the same study, it was reported that 43 percent of those age 18 to 20 experienced stress. This rises to a range of 44-48 percent at age 50 then drops to 31 percent at age 65 and 20 percent at age 71, and continues to drop to 13 percent at 91 and older (Figure 2).

This data indicates that happiness increases and stress and worry decrease with age making, the decision about when to slow or stop the aging process an enigma.

Be Careful What You Wish For

There are other dilemmas that arise in making a decision as to when to take the pill.

It is a certainty that infants, children and many young adults will not be given the pill or, if they are given the option, will not choose to take it. Arresting or stopping one's normal development in youth does not seem to be a rational choice. In fact, arrested development is viewed today as a serious pathology. Many would argue that the development of their ideas, talents and other important traits did not peak until a time well after physical development had ended, thus compounding the dilemmas even further.

For women, slowing or stopping their aging process prior to menopause presents special problems. It might be a blessing for those who want a dozen or more children but a curse for those who are poor and struggle to control their number of offspring. There is also an effect on husbands where more working years will be required to support more progeny.

Consider the probability that for adults who choose to slow their aging process, the children in their families might reasonably choose not to take the pill. These children would soon find they are approaching the biological age of their chronologically older parents. If, for several of the reasons given above, these children chose not to arrest their aging process, they might find that although their parents were chronologically older than them, their parents were biologically the same age or even younger. This example is one of many in which asynchronous age relationships might produce bizarre outcomes where one person, group or population chooses to delay or stop the aging process and others, for good reason, do not.

It is a basic aspect of human life that our relationship with others is fundamentally influenced by our perceptions of the age of our fellow human beings. Disrupting these relationships by slowing or stopping the aging process in some, but not in every human, would have unintended negative consequences limited only by one's imagination. The consequences would not only produce grotesque effects on individuals but it would also disrupt almost all human institutions. These situations would be exacerbated further if the affect of the pill was to produce different rates of aging among those taking the pill.

Health

Good health is merely one expression of the slowest possible rate at which one can age.

However, slowing the rate of aging would result in a lengthened period for increased vulnerability to pathology and a longer period to endure that pathology. It would also result in a longer period of fertility, which would impact an already-overpopulated planet and add decades of parenting. There is no data on the impact that slowing or stopping the aging process in the human brain would have on memory or sense of self. Until that critical question is answered, only speculation remains.

When the thought experiment is proposed to most people, administration of a pill to slow the aging process becomes an acceptable procedure only when it is accompanied by the imposition of specific conditions. These usually include the requirements that health and quality of life be maintained at an acceptable level. The likelihood that a pill to slow the aging process will also simultaneously guarantee good health and quality of life flies in the face of logic and all that we know about biology.

In a study in which the possibility for extending longevity was proposed to 14 international researchers on aging, 11 "expressed an interest" and all "stated that their interest was contingent on the technology allowing them to maintain their health and a certain minimum quality of life." (Underwood, Bartlett and Hall. 2009)

None of the 11 researchers explained the biological basis on which their two contingencies might be met.

A pill that slows the process of aging cannot be expected to slow the appearance of pathology or to provide some undefined minimum quality of life because the aging process simply increases vulnerability to age-associated diseases. It follows that if aging is slowed, then the period of vulnerability to pathology might well be extended. Thus, whatever advantages there might be in extending one's longevity might be compromised by the longer period of time spent accumulating more pathologies. This expectation is supported by the fact that, in usual aging, multiple pathologies are highly associated with increased longevity.

For many people, it is likely an increase in longevity at the expense of experiencing more of the vicissitudes of old age would be a price worth paying. Yet, it is certain the result would be an enormous strain on the already stressed health care system and would necessitate the need for even more massive reforms.

Living to 100 by Manipulating Longevity Determinants

Other than slowing the aging process and eliminating causes of death appearing on death certificates, the third method for increasing life expectancy would be to manipulate the systems that the forces of evolution govern to indirectly achieve an increase. Those systems are the longevity determinants described above.

These anabolic processes of repair, turnover and synthesis exceed the catabolic forces of aging until after the period of reproductive success when their functions decrease and reveal the aging phenotype. The belief that increasing the efficiency and stability of anabolic processes is how natural selection has increased species longevity is based on three observations. First, there is a remarkable relationship between the brain weight/body weight ratio and longevity in primates. The greater the weight of the brain compared to body weight, the more long lived is the species (Sacher 1975). Second, there is good evidence that for all vertebrates for which there is a long enough fossil record, the brain weight/body weight ratio increases over evolutionary time scales and provides these species with increasing longevity. The basis for this increase in longevity appears to occur as the result of more perfect repair systems, a phenomenon for which there is considerable evidence. Third, because the aging phenotype either does not exist at all, or can only exist briefly in feral animals, the likelihood is remote that natural selection could favor increased longevity because of changes in the rate of aging.

It is for these reasons that if increasing longevity is to be a goal, then research on longevity determinants would be the wiser choice. In fact, the evidence for this approach has been amply demonstrated in recent years by results with worms and flies where manipulating the genes involved in repair, turnover and synthesis (longevity determinants) has revealed large increases in longevity. This has been reported despite the fact that the investigators have interpreted their results to have effected the aging process when, in fact, they have manipulated longevity determinants or age-associated pathology. The inaccurate interpretation is because the end point used in experiments with these invertebrates is all-cause mortality.

Parts Replacement

The booming field of regenerative medicine that rocketed into prominence a few years ago has as one of its major goals the production of new specialized cell populations, tissues or even organs by programming the genes in the cultured cells of either donors or recipients. Some have seen this field as one in which young, or new, parts might be used to replace those that have aged and have lost physiological capacity. The belief that human aging might be circumvented by replacing worn tissues or organs with younger parts is analogous to the common practice of replacing old or worn parts of a complex machine to increase its longevity.

Assuming that this would be possible in humans, there is one organ that, even if it could be replaced, should not be. That organ is the brain. If it could be replaced, the recipient would not be the same person. The sense of self would be lost because a person is who they are mostly because of retained memory. In that sense, the new brain recipient would be dead. Absent science fiction scenarios where the content of an aging brain might be uploaded to a main frame, restored to its youthful state and then downloaded to a new, blank brain, the probability that brain replacement would be a desirable option is close to zero.

Replacement of other vital parts to achieve a more youthful state is fraught with both practical and philosophical problems. Organ transplantation has become an almost routine procedure and thousands have had their lives extended by this means. In almost all cases, the procedure includes the use of organs from donors younger than the recipient. Yet, there are no data to suggest that replacing damaged organs with younger parts has resulted in greater longevity for the recipients. In fact, the opposite is more likely to be true. Although it is certainly possible, there is no published data reporting that recipients of major organs (kidney, heart, liver) derived from younger donors, have lived longer than those of the same age in the general population. Also, there are no super centenarians (older than 110) who, when younger, have had organ transplants (Personal Communication, L. Stephen Coles, Director, Gerontology Research Group, Supercentenarian Research Foundation, Los Angeles, CA 90024-2767; USA, http://www.grg.org).

Even if vital organs could be replaced with younger ones and longevity extended, there is the philosophical question of how much of our body would need to be replaced before it could be argued we are a different person. This is especially important if the reprogrammed cells used to form the new parts have the genome of someone other than the part's recipient. The situation is analogous to owners of antique cars. Few, if any, of these cars have in them every part present at the time of manufacture. Most have a few, or even all of their parts, either replaced with new manufactured parts, modified parts or parts cannibalized from identical cars of various ages. The same philosophical question posed above for parts replacements in humans is relevant for antique cars. When, in the process of replacing original parts, should one consider an allegedly antique car to no longer be an antique?

Of equal importance is the fact that virtually all of our molecules are either replaced, repaired or turn over with time. The only cells thought to be present today that might be in our bodies at the start are some brain and smooth muscle cells. Nevertheless, although these cells might still exist as discrete entities, the molecules of which they are presently composed probably are not the same molecules present at the start. The starting molecules have long since been replaced in the molecular lineage leading up to those in the present cells. To be accurate when you celebrate your birthday, you should know you are celebrating the birthday of very few of the molecules, and even fewer of the cells, that were present at your birth. In short, you are never the person that you were (Hayflick 2000).

Detecting an Increase in Life Span

Because the evidence from the fossil record makes it likely that the human lifespan has been increasing, there is reason to believe it is still increasing. However, detecting this increase is formidable if the rate of increase can only be measured on an evolutionary time scale.

For example, if we assume the increase in the maximum human lifespan is occurring at the astounding rate of about one day per year, then it would take at least 365 years to detect a one-year increase. To detect this change would require keeping accurate age-at-death records for a statistically significant number of humans and for several hundred years. Even with the advances made in the last century, the availability of similar records to make this measurement cannot be found even today. And, because there are no plans to undertake this study, whether or not a naturally occurring increase in the human lifespan is occurring will remain unknown.

On Being Anti Anti-Aging

Those who have taken the position that the ability to slow the aging process in humans will result in many unintended consequences are usually labeled pessimists.

Yet, the opposite is probably true. First, the many negative consequences to individuals and to society described above are clearly undesirable. A second reason for optimism is based on extrapolating the failed predictions made periodically in the last 3,600 years, and especially those made in the last half century by biogerontologists, that we will soon be able to stop the aging process (Hayflick 2004b).

The more recent failures have occurred not from lack of trying but because of the inability to understand the distinctions between aging, longevity determinants and age-associated disease. This failure has resulted in the trivial amount of funds available to do research on the fundamental process of aging. This has occurred in spite of the fact there is a general belief that we are indeed providing enormous support for research on biological aging. We are not and the reasons for this are discussed below.

The Goal of Biogerontological Research

The research goal of many biogerontologists is analogous to that of embryologists whose goal is not to slow or stop fetal development but to understand the underlying biological processes. Similarly, the goal of most biogerontologists is not to slow or stop the aging process but to understand its cause. No one asks an embryologist if their goal is to slow or stop fetal development because it is intuitively obvious that is an unacceptable goal. The same reasoning should apply to the goal of most biogerontological research.

Much of basic scientific research is rooted in curiosity and has no practical objective.

Yet, it is true that some of the results of basic research ultimately do have practical use.

Research on aging or longevity determinants will be more productive if the goal is to discover why old cells are more vulnerable to disease or pathology than young cells. If the genesis of all age-associated diseases is in the accumulation of unrepaired dysfunctional molecules found in old but not young cells, then a common etiology of all age-associated disease might be found. Discovering how to delay or prevent the accumulation of these molecules could resolve all age-associated diseases and allow humans to live disease-free lives until the maximum life span is reached—and then drop dead. Of all of the imperfect outcomes that are likely by having the power to manipulate the finitude of life, the least imperfect would be to tamper with processes to reduce or prevent the occurrence of disease or pathology.

Overcoming the Anarchy of Words to Become a Centenarian

The major contributor to the failure to make significant progress in understanding the fundamental biology of aging and of longevity determinants is use of the term “aging research” or, more properly, “research on aging.”

“Research on aging” embraces all aspects of the finitude of life, only one small part of which is the study of the fundamental biology of aging and longevity determinants.

The common belief, held especially by policymakers and funding agencies, is that supporting “research on aging” includes research on age-associated diseases whose resolution will somehow provide insights into human longevity. It will not.

No successes in geriatric medicine will provide insight into the fundamental biology of aging. To believe that it will is the basis for the present $1 billion misunderstanding, which is what is spent in the belief that disease or pathology resolution will result in understanding the etiology of age changes (Hayflick 2003). It will not, for the same reasons the resolution of childhood diseases, such as Wilm’s tumors, iron deficiency anemia, measles and poliomyelitis, did not increase our understanding of childhood development.

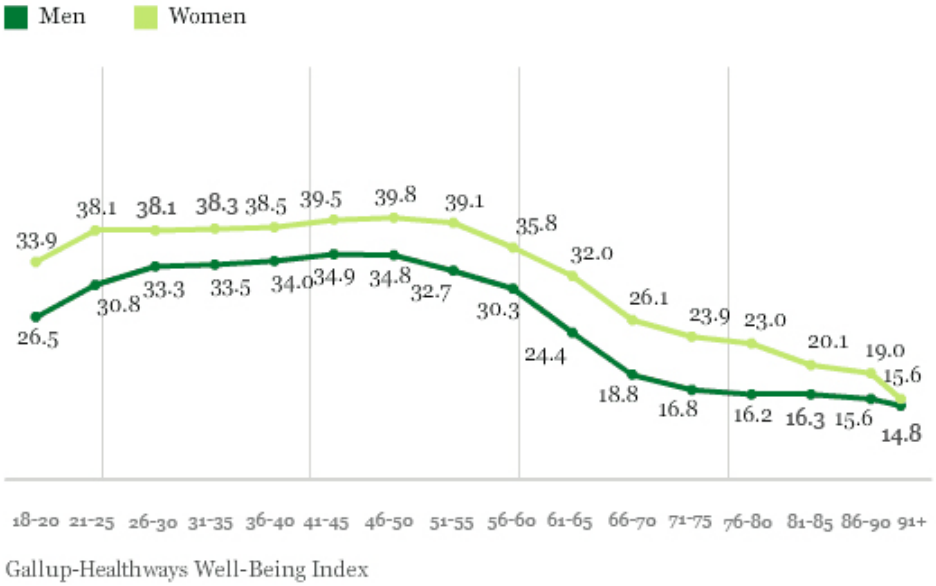
The spurious belief that controlling age-associated pathologies will provide insight into the fundamental aging processes, ironically, is contradicted by another common, but accurate, belief in medicine. It is the dogma that “The greatest risk factor for the leading causes of death is the aging process.”

The irony is that it does not require a great leap of intellect to ask: “Then, why is the funding for research on the fundamental biology of aging infinitesimal when compared to the funding for research on the leading causes of death?”

Figure 1
All Americans*

Percentage Who Say They Experienced Worry During a Lot of the Day "Yesterday," January 2008-October 2009

By age and gender

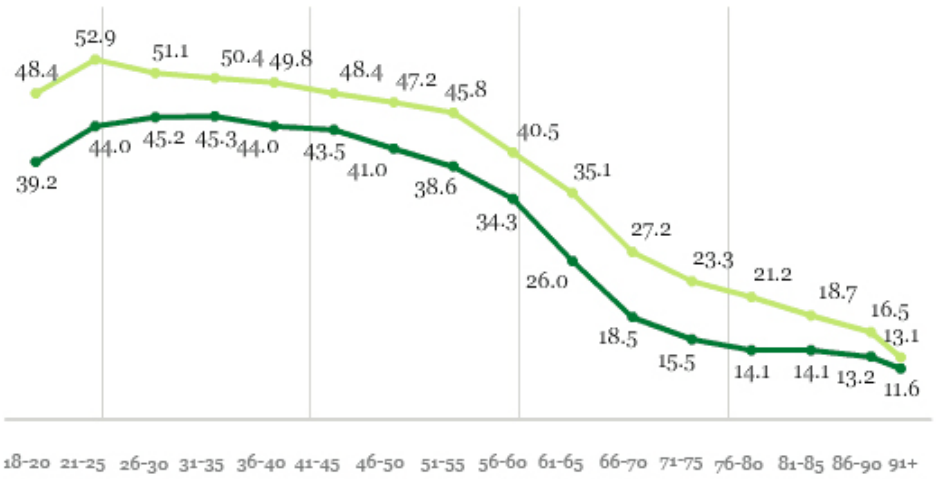


Gallup-Healthways Well-Being Index

Figure 1
All Americans*

Percentage Who Say They Experienced Stress During a Lot of the Day "Yesterday," January 2008-October 2009

By age and gender



Gallup-Healthways Well-Being Index

* All Americans, by Frank Newport and Brett Pelham, Dec. 14, 2009.

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