Occasional Paper

Has Anyone Ever Died of Old Age?

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The International Longevity Center–USA (ILC–USA) is a not-for-profit, nonpartisan research, education, and policy organization whose mission is to help individuals and societies address longevity and population aging in positive and productive ways and to highlight older people's productivity and contributions to their families and society as a whole.

The organization is part of a multinational research and education consortium, which includes centers in the United States, Japan, Great Britain, France, and the Dominican Republic. These centers work both autonomously and collaboratively to study how greater life expectancy and increased proportions of older people impact nations around the world.

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Preface

At the 2002 annual meeting of the Gerontological Society of America, a symposium was held with the provocative title "Has Anyone Ever Died of Old Age?" Sponsored by the International Longevity Center-USA, it drew participants from a variety of disciplines into a lively discussion relating to our need to understand the underlying biology of aging that predisposes us to death.

We are pleased to publish two essays presented at that symposium: the pungent and remarkable essay by Leonard Hayflick, pioneering gerontologist who has made many scientific contributions but whose name is forever associated with the limits of cell replication—the Hayflick Limit; and the sophisticated and shrewd observations by philosopher and writer, Harry Moody, Jr.

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Has Anyone Ever Died of Old Age?

Leonard Hayflick, Ph.D.

IT'S ILLEGAL TO DIE OF OLD AGE

The answer to the question posed by this symposium title is easy to give. Since 1951, no one in the United States has died of "old age" because "old age" was cured in that year.

The cure resulted from a Public Health Conference on Records and Statistics in which all state and federal agencies were ordered to adopt a standard list of 130 contributing and underlying causes of death.[1] In 1951, the list deleted a cause of death attributed to "old age."

Thus, with a single stroke of a typewriter key, old age was cured as a cause of death in this country. Because death certificates are legal documents, you should be advised that should you wish to die of old age it is illegal in this country. It is also not legally possible for you to die of a broken heart nor are you allowed to die laughing. However, if you insist on dying of old age, there are a few foreign countries in which you can legally do so.

The standard list of causes of death has been revised several times since 1952, and, effective with deaths in 1999, the United States began using the tenth revision of the International Statistical Classification of Diseases and Related Health Problems, called the ICD-10.[2] So, for the last 50 years no one in the United States has died legally of old age.

However, if you would like to be executed in the United States, you are within the law because it is legal to die in the electric chair or by lethal injection, the euphemism for which appears in the ICD-10 as cause 109—"legal intervention." So by committing an act that is illegal you can achieve a death that is legal. Is this a great country or what?

A MODEST PROPOSAL FOR ELIMINATING THE LEADING CAUSES OF DEATH

I propose that this unique method of curing causes of death be implemented for resolving the leading causes of death in this country. For example, if we would agree to delete cardiovascular disease from the standard list of causes of death we would immediately gain seven years in life expectancy, save billions of dollars in medical costs, eliminate much misery, force many pharmaceutical companies into bankruptcy and drive thousands of cardiologists and cardiovascular surgeons into unemployment lines.

Now, if you think that you can dodge the prohibition on dying from old age by choosing to die from "natural causes"—forget it. You can't do that either. Just as it's illegal to die of "old age," it's also illegal to die from "natural causes."

About 15 years ago I speculated in print why natural causes disappeared as a cause of death in the United States. I remembered that as a boy I had heard of people dying from natural causes; yet as an adult I had never heard of anyone dying from natural causes. What mystified me was how a major cause of death could have been resolved without my knowing the biological basis for its resolution. I speculated that the discoverers of the cure for natural causes were very modest

scientists. No one could recall their names, they didn't publish their work, the Nobel Prize committee ignored them, and they achieved their monumental success with no grant support. Now, I have found a simpler answer to my question. All you have to do to eliminate a cause of death in this country is simply to have it deleted from the ICD.

WHAT CAUSED THE 1998 ALZHEIMER'S DISEASE EPIDEMIC?

It is also possible to do the reverse of eliminating a cause of death. That is, it is legal to make deaths from a disease increase. This magic was performed four years ago. In the year 1998, there occurred a 55 percent increase in deaths from Alzheimer's disease in the United States. I will quote from the U.S. National Vital Statistics Report of May 2001 in which you will find a description of exactly how to increase deaths from a disease without requiring an epidemic.[3]

"In absolute terms, over 10,000 more deaths were classified to Alzheimer's disease in ICD-10 than in (the earlier) ICD-9. Nearly all of this increase (about 95 percent) comes from deaths classified in ICD-9 as Presenile dementia (290.1). In ICD-9 a definitive diagnosis was required for classification as Alzheimer's disease. Terms such as 'Alzheimer's-type dementia' or 'Alzheimer's dementia' were classified as Presenile dementia rather than Alzheimer's disease. In addition, in ICD-9 if an unspecified organic psychotic condition (294.9) is mentioned with Alzheimer's disease, the two conditions form a linkage and are coded to 290.1 (Presenile dementia). Under ICD-10 this linkage does not exist, strictures regarding definitive diagnosis are relaxed, and thus any mention of Alzheimer's disease is classified as Alzheimer's disease (G30). This involves the reclassification of nearly all cases of Presenile dementia to Alzheimer's disease."

So, this is how by the stroke of a few keyboard keys 10,000 new cases of Alzheimer's disease deaths suddenly appeared in 1998 and have continued annually since that date.

NO ONE DIES FROM ANY OF THE LEADING CAUSES OF DEATH

The statisticians would have you believe that older people must die from a particular pathology, but I am one of a very few biologists who believe the opposite. That is, I believe that older people do not die from any of the 130 legal causes specified by the statisticians.

Here's why: Most of the biomedical community believes, to the point of uttering it as a mantra, that the major risk factor that contributes to the leading causes of death—cardiovascular disease, stroke, and cancer—is the aging process. In fact, I have argued that no one arbitrarily over, say, the age of 75 has ever died from any of the 130 causes on the ICD. What people over the age of 75 do die from is the continuous loss of physiological capacity that is the hallmark of the aging process and that increases their vulnerability to the leading causes of death. Two-thirds of people over the age of 75 will die from one of the three leading causes of death. By this reasoning the ultimate cause of death for everybody over age 75 is, in fact, "old age" or "natural causes." What is legally written on the death certificates of old people is simply irrelevant detail.

The biological evidence that the direct cause of all deaths in old age is the aging process is incontrovertible, but the statisticians seem to be unaware of this. The statisticians are not alone in their misunderstanding. They have been joined by most of the biomedical community, who are dedicated to the proposition that the study of the leading risk factor for death—the aging process—is unimportant. The irony is that

despite the belief of most biomedical professionals that the greatest risk factor for the leading causes of death is the aging process, the resources that they have decided to devote to understanding the aging process are microscopic compared to the resources made available for the study of age-associated diseases.

This discrepancy in funding is indefensible because the unifying concept that underlies the etiology of all of the leading causes of death is the fact that old cells are more vulnerable to pathology than young cells. Thus, the fundamental question that is rarely addressed, and for which few resources are made available, is this: Why are old cells more vulnerable to pathology than young cells?

UNDERSTANDING AGING IS NOT DEPENDENT ON RESOLVING AGE-ASSOCIATED DISEASES

What are the consequences of our present policy of devoting most of our resources to an understanding of age-associated diseases? Of the several major consequences, one is that we might resolve all of the leading causes of death in old age and thereby force most physicians, biomedical researchers and all geriatricians into an unemployment line. With respect to the increase in life expectancy that this monumental achievement would produce, an increase of about 15 years would occur.

What then would be the cause of deaths? The ICD would now have to be modified to reveal only one cause of death for those over age 75—namely, old age. Thus, having resolved the leading causes of death we will then have revealed the underlying cause of all deaths in old age—the aging process. For the first time in human history, most people will be found to die of old age.

What the biomedical community does not realize is that the resolution of age-associated diseases will advance our knowledge of aging processes to the same extent that the resolution of pediatric-associated diseases, such as poliomyelitis, acute lymphocytic leukemia, Wilms' tumors, and iron deficiency anemia, increased our knowledge of childhood development; that is, no increase in knowledge occurred at all.

The aging process is not a disease so the probability of resolving it as a cause of death is, in my view, close to zero. It is close to zero because even with the most advanced technology known today, we cannot control the rate of aging in something as infinitely less complicated as our own automobiles. Even the desirability of having the power to interfere with the aging process is filled with unintended consequences, a subject that, although peripheral to the subject of this discourse, has been discussed elsewhere.[4,5]

The distinction between aging and disease is central to understanding why the resolution of the leading causes of death in old age will tell us little about the fundamental biology of age changes.[4,5] Aging is not a disease because, unlike any disease, age changes[1] occur in every animal that reaches a fixed size in adulthood;[2] unlike any disease, age changes cross virtually every species barrier;[3] unlike any disease, age changes occur in all animals that reach a fixed size in adulthood and only after sexual maturation;[4] unlike any disease, age changes occur in animals removed from the wild and protected by humans, even when that species has not been known to experience aging for thousands or millions of years;[5] unlike any disease, age changes increase vulnerability to death in 100 percent of the animals in which it occurs;[6] and unlike any disease, age changes occur in both animate and inanimate objects.

THE UNRELIABILITY OF DEATH-CERTIFICATE DATA

In the minds of most of the public, policymakers and many biomedical scientists, no one suffers or dies from aging. We suffer and die from the diseases associated with the aging process. Yet, age changes are the cause of the increased vulnerability to everything that is written on the death certificates of the elderly.

How reliable are the legal causes of death currently written on death certificates? The fact is that there are multiple pathologies in older people so the true cause of death in old people is rarely known. This fact is substantially ignored by those who depend slavishly on the statistics that rely on what is written on death certificates. Because there are few autopsies and little research on the etiology of death in older people, the cause of most deaths in old age is still hidden in the proverbial black box. The numbers of autopsies that have been done on the elderly have continued to decrease in the last few decades. And the small number of autopsies that have been done is inversely proportional to age. In those rare instances where autopsies have been performed on a large number of old people, the findings have shown that from 40 percent to 50 percent of the causes of death appearing on the death certificates have been inaccurate.[6-8] In the most recent study of 93 postmortem examinations done in an Israeli hospital over a 20-year period, 42 percent of the causes of death written on the death certificates were incorrect.[9] Over this 20-year period the rate of autopsies dropped from 2.8 percent to 0.25 percent. These findings should cause considerable concern for the many political, economic, actuarial, and scientific decisions that, without benefit of autopsy, may have a 40 percent error rate.

Both lay and science policymakers, properly impressed with the future demographics of the graying of all economically developed countries, are basing important policies and decisions on a flawed understanding of what constitutes aging research and what the results of that research might accomplish.

Understanding how the molecules of an old cell differ from those in a young cell and how those differences lead to pathology has the potential to increase our understanding of the etiology of cancer, cardiovascular disease, stroke, and even age-related accidents because the etiology of all of these conditions is rooted in those differences.

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Dying From Old Age: Two Horns of a Dilemma

Harry R. Moody, Ph.D.

"DID ANYBODY EVER DIE OF OLD AGE?"

Logically, there are two answers to this question: Yes and no. Both could imply the "end of aging as we know it," and the question itself has assumed controversial status.[1,2] Thus, if we answer the question with:

(1) Yes, people do die of old age, it means aging is a disease, so let's find a cure for it. (And people will live indefinitely.)

(2) No, it means people die of some other disease, so let's find a cure for those diseases. (And people will live indefinitely.)

Hmmm. Peculiar answers to what may be a peculiar question. Let's examine these answers—yes and no—in terms of the logic of their presuppositions and their implications for research about aging. We will find that approaching the question, "Did anybody ever die of old age?" raises, in turn, profound questions about gerontological research, about fate and accident, and even about the human condition itself, questions with a long and venerable history.[3]

AGING IS A DISEASE

First let's examine "Yes, … aging is a disease." Admittedly, it is peculiar to speak of aging itself as a "disease." Mainstream gerontology has spent a generation or two drumming into everyone's head the dogma that "aging isn't a disease, but it increases our susceptibility to disease," etc.[4,5] But refusing to call aging a "disease" is a presupposition we could change.[6,7] The fact that aging is familiar and "normal" (as in "normal aging") doesn't mean we can't declare it to be a disease and change our linguistic habits. We could begin to change our ordinary language this way, just as we do when we speak of insomnia as a disease or alcoholism as a disease.

To think of aging as a disease becomes the strategy for "anti-aging medicine," which may not now be a legitimate clinical field but could become an agenda for legitimate research in biogerontology.[8] Indeed, some advocates of "transhumanism" in ethics argue strongly that life extension and even immortality should become our explicit research agenda. To call aging a disease isn't a description of current language so much as it is a decision about how we're going to use language. Ordinary language, like scientific language, can change over time. But the decision here has a big consequence, and the consequence is that we begin to think of aging as a condition that can be changed, even altered or abolished, just as one might imagine a cure for progeria (a disease that causes accelerated aging in childhood).

CURE DISEASES ONE BY ONE

This first conclusion—aging is a disease—will be unpalatable to many, especially those who accept

mainstream gerontology's opinion on the matter. So let's take the second alternative—that nobody ever died of old age. Ironically, by following this answer we might end up with a life-extension strategy not very different from that urged by radical advocates of life extension. We might decide that because aging isn't a disease, nothing prevents us, in theory, from eliminating all causes of death, permitting people to live indefinitely. Thus, we also reach the radical life-extension point of view by the opposite logical route. But note in this view we would get there only incrementally, as ethicist John Harris has suggested. That is, we would, step by step, eliminate distinctive diseases of old age and, step by step, push back life expectancy at every age, without limit.

Now what happens if we follow this second strategy? Here we must have recourse to rudimentary epidemiology. In the short run, the results are disappointing. Even if we cured the big killers (heart disease, cancer, and stroke) average life expectancy in the population would only rise 12 years or so (from 77 to 89 in the United States). The reason is that once we save people from dying of one disease, they die of another disease. So we try the following thought experiment: What happens if we could cure (or prevent) all diseases? Answer: People would die from accidents. Again and again in theoretical gerontology we come back to the comparative role of fate and accident in shaping the limits of life. It turns out, as the authors of Greek tragedy understood, that fate and accident are intertwined in mysterious ways.

To understand why, consider a prosaic example. Note that the sigmoid (S-shaped) curve for the "life expectancy" of drinking glasses in a restaurant actually resembles the same formal curve as we observe for death rates in populations: the familiar life tables of demography. Of course, we might succeed in shifting the curve or modifying its shape by various preventive medical interventions. We could push the curve to the right (compression of morbidity and mortality); we can even displace it substantially to the right by big medical breakthroughs. Still, the glasses would continue to break by chance (accident) at various times, some early, some late, and before long we're back to the familiar sigmoid curve again.

Here we reach the heart of the matter. These incremental moves—a disease-by-disease strategy toward indefinite life—don't get us very far because human bodies, like drinking glasses, are still vulnerable and subject to accidents. To change the role of accident, we will have to dig deeper, to the level of "fate": that is, what makes drinking glasses (or human bodies) subject to statistically predictable accident curves? Along these lines, Jay Olshansky[9] and others have argued that any strategy for health promotion will have only minimal effects on average life expectancy and maximum lifespan unless we have a deeper understanding of the biology of aging itself.

THE BASIC QUESTION: WHAT IS AGING?

So we come to the inescapable question: What is aging? Evidently, the answer is not simply chronological passage of time but rather what we sometimes call "biological aging"—some underlying process, perhaps the same process that is modified (but not eliminated) by caloric restriction, which is the sole proven intervention for lifespan prolongation.

But to refer to a technology or mode of intervention (like caloric reduction) explains nothing. We might therefore be tempted to think of aging in more analytic or mathematical terms. Thus, one definition of aging might be "increasing probability of death [the Gompertz curve] along with impairment of function" with advancing time. The trouble with this approach is that it

doesn't explain anything either. "Increasing probability" after all doesn't actually cause anything. We need to find some specific reason for this "impairment of function": for example, a link to immune function or some other physiological failure that could be the basis of increasing probability of death.

Think of the aging body in comparison to an aging car or aging aircraft and consider the engineering concept of "mean time to failure." Just as aging aircraft are more vulnerable, so are aging bodies. But, again, mean time to failure doesn't cause anything; it's just another statistical statement about probable longevity, in aircraft or organic systems.

The difference is that biological systems, unlike other natural objects such as a car or an aircraft, have within them a capacity for repair and regeneration. Along these lines, Plato gave the example of an old coat, constantly repaired, its material components always changing but its identity of form remaining the same. So, too, repair mechanisms at the cellular and molecular level give formal continuity, "sameness," to the human body.

If we define aging as "impaired function with increased probability of death," then we need to look at the case of animals that do not age, such as the hydra or sea anemone, which regenerates itself (through budding) and shows a constant (not increasing) probability of death from predators, accidents, and the like. The hydra, then, is not immortal—that is, it is not invulnerable. But it lives indefinitely, which is the desired outcome for the life-extension strategy.

So we have come some distance in understanding the logic of the question "Did anybody ever die of old age?," and we understand something of the conceptual alternatives entailed by the question. It is not formal or statistical description or redescription that we need but a better causal understanding of the process of aging itself. To push matters further, let us distinguish here between proximate and underlying causes. Here I want to argue that the question "Did anybody ever die of old age?" is logically ambiguous because it entails different kinds of causality.

To understand the causal logic involved, compare our question to another question: "Did anybody ever die of AIDS?" Now, we could reasonably answer, "No, nobody ever died of AIDS because AIDS is simply a progressive collapse of immune function." Thus, people don't die of "impaired immune function," they die from an opportunistic infection or from a cancer or other organ failure, but not from AIDS itself.

This would be a logical answer, but it's an implausible one: a bit like saying, "Guns don't kill people, people kill people." The statement may be literally true, but it's not very helpful because the proximate cause (a firearm) only becomes effective when someone actually shoots the gun. In the same way, rare forms of pneumonia kill patients who have been compromised by AIDS. We wouldn't want to say, "Nobody ever died of AIDS" and therefore devote all our research efforts to finding cures for specific opportunistic infections that end up on the death certificates of some AIDS patients.

Thus, as with AIDS, the answer to the question "Did anybody ever die of old age?" is both yes and no, because old age only "kills" when conjoined with some specific (proximate) cause.

Conceived in this way, the strategy for curing AIDS becomes comparable to the strategy for "curing" aging: namely, eliminate those factors that permit proximate causes to be fatal.

Here lies the strategy for regenerative medicine that could permit organisms to regenerate

themselves like sea anemones do. It may be that in coming generations stem-cell research will provide clues that permit regenerative medicine to become the reality for which so-called anti-aging medicine is only a simulacrum.

THE ILLUSION OF IMMORTALITY

Now we come to the big question, the one that animates this entire agenda. Suppose our strategy for regenerative medicine succeeds. Suppose we "cured" aging itself? Would we live forever—or at least indefinitely? What would happen if we could maintain the human body at its maximum or optimal state of regeneration? Here John Harris,[10] citing the work of Tom Kirkwood, argues that we would not live forever but rather for a very long time. Kirkwood suggests we could say just how long by looking at the body of an 11-year-old, the time in life when regenerative (healing) capacity as at its natural maximum. Using this example—imagining we are all 11-year-olds forever—actuarial calculations suggest that we could conceivably live for 1,200 years.

What would kill us? Alas, accidents. Whether human or sea anemone, disease is not the only thing that causes death. But, as I've suggested, the very term "accident" is just another way of describing an inherent vulnerability of the organic system. Consider again the drinking glasses in the restaurant. The "aging" of those drinking glasses is another way to speak about mean time to failure. But no specific glass ever breaks from mean time to failure, the glass always breaks for some specific cause that shatters it.

Following the sigmoid curve, eventually all the glasses do break. But they wouldn't break at all if they were made of, say, stainless steel. Made differently, they could escape vulnerability. So too, if the human body were made of a biological equivalent of stainless steel, we too could escape vulnerability. True, drinking glasses made of stainless steel wouldn't be "glasses" in the ordinary sense of the word. But they might function perfectly well as drinking vessels.

So too if human bodies were reengineered not simply by regenerative medicine (to be like 11-year-olds, very long-lived) but for far, far greater regenerative capacities, then we too might live much, much longer. Here lies another strategy for the hypothetical science of life extension: artificial organs and systems stronger than our own.

We might begin by perfecting the artificial heart. Nobody ever died of old age in the same way that nobody's heart ever stopped because of impaired cardiac function. There will always be some specific cause—some coronary obstruction, arrhythmia, whatever. A generic term like "heart failure" simply describes the weakening of a capacity to resist or recover from a specific cause that stops the heart from beating. But is heart failure ever a cause of death? According to the line I have argued here, perhaps not. In the entire discussion so far, we've offered different definitions of aging but we haven't defined death, so I will now offer one. In *How We Die,*[11] Sherwin Nuland argues that all deaths are caused by loss of blood supply to the brain.

DISPOSABLE SOMA

No matter what scenario of life extension we adopt, it's hard to imagine circumstances in which the brain is absolutely invulnerable to loss of blood supply by some biological or accidental catastrophe. Brain vulnerability stands as an ultimate barrier to life-extension technology. Maybe the conclusion is that we are all simply "disposable soma," to use the phrase from Kirkwood's memorable theory about the genetics of aging.[12] In that case we could achieve a kind of immortality through the germ line (living on through our successors), which is the kind of immortality many believe in today. But if we want

to preserve our individuality, we have to preserve our cognitive uniqueness and history, that is, preserve the information properties of the human brain. Do we then "download" onto a computer, as some transhumanists and science-fiction writers have suggested?[13] The only scenario where the brain would be invulnerable would be one in which we have downloaded its contents onto some backup system, perhaps along the lines anticipated by Kurzweil.[14]

We now pass beyond the bounds of biogerontology and enter into more turbulent waters of mind-body questions and murky matters of individual identity, questions perhaps best left to fiction writers and theologians. If we eliminate aging, would we eliminate altogether what we understand, however dimly, as the human condition?[15] What is clear enough is that pushed to the extreme, a question like "Did anybody ever die of old age?" raises profound questions about our human condition and about steps toward "enhancement" of what we accept as our human condition, including the life cycle itself.[16,17]

As I write this manuscript on my computer I am filled with a sudden panic because I realize that I've forgotten to back it up. Suppose my computer were to fail, and everything I've written were lost? Perhaps that panic is a little like sudden death: the recognition of vulnerability and realization that everything can disappear in a flash. Until we are able to download ourselves (and would it really be ourselves?), we will probably have to live with that vulnerability, which so far at least seems to be part of our human condition.

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About the Authors

Leonard Hayflick is an internationally renowned scientist, who, in 1962, overturned scientific dogma with his discovery that, contrary to what had been believed since 1900, cultured normal human cells have a limited capacity for replication. This phenomenon is known as "The Hayflick Limit." His discovery that mortal and immortal mammalian cells exist became the basis for much of modern cancer research. He suggested that the finite replicative capacity of cultured normal human and animal cells is the in vitro expression of aging and longevity determination.

Dr. Hayflick also developed the first normal human diploid cell strain (WI-38) that is used worldwide wherever cultured normal human cells are required. He proved the safety and efficacy of the first poliomyelitis vaccine produced in WI-38. Today most of the world's human virus vaccines are produced in WI-38 or similar cell strains. Hayflick also discovered the cause of primary atypical pneumonia ("Walking Pneumonia") in humans to be a mycoplasma, the smallest free-living micro-organism.

Dr. Hayflick is the author of over 250 scientific papers, book chapters and edited books, of which four papers are among the 100 most cited scientific papers of the two million papers published in the basic biomedical sciences from 1961 to 1978. He is the author of the popular book *How and Why We Age,* published in 1994 by Ballantine Books and translated into nine languages.

Dr. Hayflick is currently professor of anatomy at the University of California, San Francisco. He received his Ph.D. from the University of Pennsylvania in 1956.

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Dr. Moody, a philosopher by background, is the author of many articles and books in gerontology on applied ethics, the humanities and arts, and the search for meaning in later life. Dr. Moody was national program director of the Robert Wood Johnson Foundation's Faith in Action Programs and executive director of the Brookdale Center on Aging.

Dr. Moody received his Ph.D. from Columbia University.

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