A General Theory of Aging: Part I

Orientation

In Addendum to Aging: Cause and Cure, three long-lived mice were of special interest. The oldest of these three mice was still living when the addendum was published. She died about two months later. I am eager to report on the implications of this mouse's unusually long life span, but I need to lay some groundwork before doing so. To begin with, I need to introduce a general theory of aging to provide a scientific framework within which to understand and discuss the phenomenon of aging in a generalized way. I expect to follow this with a quantitative analysis of how the anti-aging vitamins are likely to alter human life expectancies. Once this groundwork has been laid, we should be ready to have a look at the significance of the final long-lived mouse's unusual longevity.

Setting

Growing up, we learn about aging, early on, by our observations of people we encounter. We observe that Grandmother differs from Mother, for example. Grandmother is more frail than Mother, her skin is more wrinkled, and her hair is more gray. Grandfather and Father show a similar set of differences.

We visit Great-Grandmother at the nursing home. We observe that she is yet more frail—she uses a walker to get around. Her skin is more wrinkled, and her hair is more gray and thin. We see many other people at the nursing home, male and female, who are in a physical state similar to Great-Grandmother.

We observe the family dog becoming less active with advancing age. He spends more time sleeping. His muzzle goes gray. He walks more stiffly. His appetite declines.

We deduce from our observations that deterioration of the physical body is intrinsic to living things—that time automatically and inevitably turns healthy young adult organisms into physically decrepit senior organisms. We conclude that a fixed life span has been assigned to each species. We accept that this is just the way life naturally is. We learn to call the advancing decrepitude of physical bodies, which we observe slowly happening all around us, “aging.” We think of aging as a phenomenon unique unto itself.

Our observations of aging are sound enough. Our deductions and conclusions about aging appear to be much mistaken.

Two Questions

There are two questions, foundational to the field of aging biology, which have not previously, in my opinion, found satisfactory answers:

1. What is the essence of aging—what is this ubiquitous phenomenon of aging we observe within the biological realm?

2. What is the etiology of aging—what causes biological aging?

I first asked myself these two questions roughly forty years ago. I have only recently been able to work out answers to them which seem to me to be satisfactory. The present article is a first presentation of my newfound answers.

My quest to find answers to these questions took me fairly rapidly to Alex Comfort’s book, “The
Biology of Senescence.”2 I read it in 1980, as I recall. I was a student at the University of Toronto at the time, working on a PhD in nuclear physics. Biology courses were not part of my physics curriculum at that level. My interest in aging was a private one, which I pursued on my own as time allowed. I did, however, manage to work this interest into a course on nuclear medicine I took as part of my Ph.D. program. The course involved a student seminar component. I was interested, at the time, in a theory introduced to me by Comfort’s book. The theory was that cascading cellular damage due to ionizing radiation may be the root cause of biological aging. I was especially interested in radiation damage to the DNA. I presented a seminar incorporating that interest.

From there were numerous twists and turns, most too distant now even to recount reliably from memory. But the ending is quite clear. Ultimately, the answers to my two questions were won through a struggle to fashion what I had discovered about the root cause of human aging3 into a more generalized theory of biological aging, applicable to all biological organisms.

**Aging in Present-day Humans**

I have previously argued that, in the special case of humans, aging, as we know it at present, is nothing more than a disease.4 Human aging, according to my research findings, is a vitamin deficiency disease, curable by supplementation of our diets with two closely related, previously unknown vitamins, methylphosphonic acid (MePA) and methylphosphinic acid (MePiA).

This is why the early chapters of the biblical book of Genesis record, without apology, human life spans of nearly a thousand years. According to my theory, MePA and MePiA were naturally present in rainwater back at that remote time. Our ancestors of long ago were naturally supplied with these anti-aging vitamins every time they took a drink of water.

Today, rainwater contains no appreciable MePA and no appreciable MePiA. There is no other natural source of these vitamins. As a result, the natural human diet is currently lacking both MePA and MePiA from birth on, and the vast majority of humans die before they have lived even a hundred years.

According to my research, we are not dying at relatively young ages today because this is just the way life naturally is. Rather, we are dying at relatively young ages today because we are being killed by vitamins MePA and MePiA deficiency disease (aka aging). If we were to take away vitamin C from everyone’s diets, then everybody would be dying at even younger ages, because of vitamin C deficiency disease (aka scurvy).

**Aging in General**

The foregoing explanation of the cause of human aging posits a theory of aging for the special case of contemporary humans. This theory may be succinctly summarized as follows:

***Special Theory of Aging:*** Contemporary human aging is simply progression of a congenital deficiency disease of the anti-aging vitamins MePA and MePiA.

My purpose, in the present series of articles, is to generalize this theory, first to all biological organisms over all time, and then, by treating biological organisms as complex biological machines, to the class of all machines over all time. My goal is to unmask aging—to expose to clear view its fundamental essence and etiology.

**A General Theory of Aging for Biological Organisms**

The general theory of aging for biological organisms set forth and elaborated here is this:

***General Theory of Aging for Biological Organisms:*** Aging in all organisms of all species is always simply progression of congenital disease.

This general theory of aging emerges from and is inspired by the special theory of aging summarized in the previous chapter. The special theory

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of aging equates the observed phenomenon of aging in present-day humans with a congenital nutritional deficiency disease. The general theory of aging takes this equality to be non-accidental. It does away entirely with the commonly held idea that aging is some sort of special biological phenomenon, in a category of its own. It asserts that aging is always simply the observable result of one or more disease states present within the organism from birth.

**Time’s Role**

This general theory of aging denies the idea that aging imposes an immutable time limit on life for each type of animal. It asserts that the essence of aging is *disease*. Because disease is always potentially subject to healing and cure, aging is, in principle, stoppable and reversible in all species.

This general theory of aging contradicts the idea that time is somehow responsible for aging. It holds time to be benign. It says that death due to “old age” does not exist. It denies that chronological age kills anyone. It asserts that “Aging... is always simply... disease”—nothing more. It finds no biological clock metering out a pre-programmed number of seconds to each individual. It finds only progressive congenital disease. Within its framework, the expression, “growing old,” in its current geriatric sense, is seen as entailing a deeply rooted semantic error which should be corrected by use of the more accurate expression “growing sick.”

The intimate association of chronological age with aging arises only because the diseases responsible for aging are congenital. Chronological age and congenital disease start out together near zero at birth and progress together throughout life. This makes aging appear to be an age-specific phenomenon, but this appearance is coincidental and circumstantial only. Age does not cause aging, and age does not schedule aging. Age merely furnishes a convenient time parameter for charting the progression of the specific congenital disease(s) causing aging in each instance.

This emphasizes that the congenital diseases which are responsible for aging are ordinary diseases, each conforming fully to the normal definition of disease. For diseases in general, time is the parameterizing variable—the progression of the disease is charted versus time. The progression of the congenital diseases responsible for aging is also charted versus time, but age serves conveniently as the time variable because age begins at the same time the congenital disease begins.

**The Congenital Diseases of Aging**

This general theory of aging denies the idea that there is any one congenital disease responsible for aging in all species. Specifically, this general theory of aging is not that aging in all species is always due to deficiency of MePA and MePiA. When the general theory of aging uses the term “congenital disease,” it means to encompass a vast, unspecified constellation of potential congenital diseases.

Today, the great majority of the congenital diseases of aging are little understood, unnamed, and without cures—vitamins MePA and MePiA deficiency disease being a rare example of an exception. To the present time all of the congenital diseases of aging have, unwittingly, tended to be lumped together as if they were all one and the same thing. This general theory of aging says that they should not be lumped this way. The congenital disease (or superposition of multiple individual congenital diseases) responsible for a life span of just one month in fruit flies is not likely to be the same congenital disease (or superposition of multiple individual congenital diseases) responsible for a life span of two years in mice.

This distinction has important implications for aging research. For example, it clarifies that it is a mistake to suppose that an intervention which significantly lengthens the life spans of mice must not have anything to do with aging because it fails to lengthen the life spans of fruit flies. A hypothetical example in this category might be the discovery of a congenital virus in a strain of mice. Imagine that elimination of the virus from that strain is found to lengthen significantly the average life span of the mice, but the same virus is found to be non-pathogenic to fruit flies. According to this general theory of aging, the congenital viral infection of the mice would be a congenital disease of aging for the mice, and its cure would represent real progress against aging in that particular strain of mice.

Though little is known about the congenital diseases responsible for aging across nearly all species
at present, these diseases should not be regarded as intractable. Many of the diseases familiar to medicine today were little understood, unnamed, and without a cure not all that long ago. Parkinson’s was characterized only 200 years ago. Pasteur’s cure for rabies was advanced just over 130 years ago. The cure for pellagra was discovered just over 80 years ago. The polio vaccine was developed only 65 years ago. Due to the accelerating pace of medical research, additional examples abound in recent decades. It seems probable that cures of numerous congenital diseases responsible for aging in varied species will be found in the near future.

**The Take-Home Lesson from Part I**

The essence of what we call “aging” in biology is simply congenital disease. ♦