

The Biblical Chronologist

WHAT HAS BEEN IS REMOTE AND EXCEEDINGLY MYSTERIOUS. WHO CAN DISCOVER IT?

(Ecclesiastes 7:24)

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A General Theory of Aging: Part III

Parts I¹ and II² of this series have discussed the essence of aging. This final part turns to the etiology of aging, followed by a discussion of the integration of the general theory of aging with evolutionary biology.

The general theory of aging for biological organisms answers explicitly what aging in biological organisms is. It is progression of congenital disease. This clarifies the fundamental essence of aging, but it leaves the etiology—the root cause—of aging still shrouded in mystery. How do the congenital diseases of aging arise?

The degree of complexity exhibited by even the simplest biological organisms makes it difficult to elucidate the etiology of aging. To understand the cause of aging, so as to be able to answer such basic questions as why aging is such a ubiquitous phenomenon in the biological realm despite the great diversity of biological organisms, it is helpful to extend the general theory of aging into the mechanical realm.

Biological organisms may be regarded as extremely complex biochemical machines. When the theory of aging is extended to encompass all machines, biological and mechanical, it becomes:

General Theory of Aging: Aging in all machines of all types is always simply progression of one or more disorders stemming from intrinsic design flaws.

¹Gerald E. Aardsma, “A General Theory of Aging: Part I,” *The Biblical Chronologist* 10.2 (February 12, 2020): 1–4. www.BiblicalChronologist.org.

²Gerald E. Aardsma, “A General Theory of Aging: Part II,” *The Biblical Chronologist* 10.3 (March 4, 2020): 1–4. www.BiblicalChronologist.org.

The Etiology of Aging

Think about a fairly simple mechanical machine. For example, consider a small, two-stroke, internal combustion engine, such as might be used to power a weed eater. Imagine a cohort of a thousand such machines, all fresh from the factory. They are installed on identical weed eaters and put into constant use.

Two-stroke engines require that oil be mixed with the gasoline to lubricate their moving parts. Imagine that someone has forgotten this in the present instance so that all of the machines are being operated without oil. That is, imagine that a “disease state” has been induced in the machines by withholding oil. The moving parts of the engines wear with use (i.e., the engines age), and, after some hours, one after the other, the machines stop working (i.e., they die). Autopsy reveals that the piston rings have become too worn for adequate compression.

Plotting a survival curve for these engines is expected to yield a normal Gompertz function: the induced disease is present in the entire population of the machines, it is congenital, and the probability of death is expected to increase exponentially with time.

This is an example of mechanical aging. Aging in this particular instance of worn piston rings arises because of wear of moving parts. When metal surfaces rub against each other, atoms may be rubbed out of the contacting surfaces. These atoms may be lost from both surfaces, or they may move from one surface to the other, or they may relocate on one surface.

The disease which has been induced in these machines can be “cured” by addition of oil to the gasoline. Let us apply this cure to a second cohort of a thousand weed eater engines fresh from the

factory. Repeating the above experiment, we find that the second cohort has a much longer average life span—months instead of hours. We have successfully cured oil deficiency disease, but we have not eliminated aging. Now a new cause of death emerges. Autopsy reveals that the spark plug electrodes have worn away, inhibiting the spark needed to ignite the fuel mixture. We have uncovered a new congenital disease of these engines: electrode ablation disease. This is not an induced congenital disease; rather, it is an intrinsic congenital disease, inherent in the present design of the machine.

A cure for this intrinsic congenital disease might be found by choosing a different metal for the electrodes, one which is better able to withstand the electric arc plasma (i.e., the spark) each engine cycle. This would give the machine an even longer average life span, but it would still not entirely eliminate aging. The cause of death might now be found to be due to the air filter slowly clogging, reducing air supply to the cylinder. This causes the spark plug to become fouled with soot, so it shorts out and no longer sparks. This congenital “pulmonary fibrosis” disease is interesting because it shows an example of aging which is not due to wear. The air filter is not worn. It just needs to be cleaned.

Aging arises in all machines, including biological machines, because of congenital diseases intrinsic to the design of the machine. The greater the complexity of a machine, the more ways there are for things to go wrong. Because even a single-celled organism displays extreme complexity, the potential for congenital diseases with biological organisms is clearly enormous—hence the ubiquitous presence of aging in biological organisms.

The Prospect of Cures for Aging

Fortunately, the potential for cures is also much greater biologically than it is mechanically.

We have seen that adding oil to the gas of a two-stroke engine reduces wear between moving parts but does not completely eliminate it. Eventually, wear will cause the engine to die, even with adequate oil present at all times.

The ideal cure for wear would be for the machine to have a way of putting displaced atoms

back where they belong. While it is difficult to see how this might be accomplished with present mechanical machines, it is not at all difficult to see how it might be accomplished with biological machines. Biological machines operate at the molecular level. They have theoretical potential to renew parts continually, molecule by molecule. And indeed, such repair mechanisms do exist within biological machines. Cellular self-repair of DNA is one such example.

Aging and Evolutionary Biology

I need to break into the further development of this general theory of aging briefly at this point with an interpersonal note. I am concerned that the following discussion may offend readers who, for theological reasons, reject evolutionary biology. It should not do so. It is most certainly not intended to do so.

Those who have followed my research over the years know that I have consistently held to a high view of both the biblical historical narrative and modern science.³ I have previously shown that a proper synthesis of these two sources of knowledge yields the view that the universe was supernaturally brought into existence 5176 ± 26 B.C. with a virtual history in proleptic time.⁴ This is the view I hold. It is the only logically consistent view of ultimate origins that I have been able to find. It does

³See, for example, Chapter 1 of my first book: Gerald E. Aardsma, *A New Approach to the Chronology of Biblical History from Abraham to Samuel*, 2nd ed. (Loda, IL: Aardsma Research and Publishing, 1995). www.BiblicalChronologist.org.

⁴Gerald E. Aardsma, “Toward Unification of Pre-Flood Chronology,” *The Biblical Chronologist* 4.4 (July/August 1998): 1–10. www.BiblicalChronologist.org. Gerald E. Aardsma, “Toward Unification of Pre-Flood Chronology: Part II,” *The Biblical Chronologist* 4.5 (September/October 1998): 1–10. www.BiblicalChronologist.org. Gerald E. Aardsma, “Toward Unification of Pre-Flood Chronology: Part III,” *The Biblical Chronologist* 4.6 (November/December 1998): 1–16. www.BiblicalChronologist.org. Gerald E. Aardsma, “Toward Unification of Pre-Flood Chronology: Part IV,” *The Biblical Chronologist* 5.1 (January/February 1999): 1–10. www.BiblicalChronologist.org. Gerald E. Aardsma, “A Unification of Pre-Flood Chronology,” *The Biblical Chronologist* 5.2 (March/April 1999): 1–18. www.BiblicalChronologist.org.

*not encounter any theological difficulties with evolutionary biology that I have been able to find. It is from this basis that this closing section is written.*⁵

A great deal of time and energy seems to have been invested by various researchers in an effort to integrate one or another theory of aging with evolutionary biology to produce a logically harmonious whole. I am not aware of any that have succeeded.

The prevalence of aging within the biological realm seems to imply that biological evolution fosters aging. But the underlying tenets of evolutionary biology seem to require the opposite. Natural selection sees survival as a fundamental ingredient of evolutionary success (e.g., “*survival of the fittest*”). Aging accomplishes the opposite of survival. Thus, aging seems incompatible with natural selection, in the same sense that “A” is incompatible with “not A.” This gives the widespread existence of aging within the biological realm the appearance of a Kuhnian anomaly of no small proportions for evolutionary biology.

The general theory of aging presented in the present series of articles says that there is no real Kuhnian anomaly here, but rather only a Gordian knot. This closing section undertakes the task of cutting this knot.

It is important to cut this knot, of course. If one’s general theory of aging is unable to cut this knot, one does not have a tenable general theory

⁵My daughter-in-law, Esther, contributed the following suggestions in regard to this section.

It may be helpful to point out that the virtual history we are seeing is post-Fall, and we must view origins in the context of a broken, re-created, fallen world. Christians can relate to mosquitoes and thistles and carnivores being a result of the Fall, but I think rarely consider larger ramifications. Somehow, we expect to read a perfect world’s beginning into a broken world’s empirical past.

An understanding of virtual history frees Christians from needing to feel threatened or insecure when faced with evolutionary evidence. In my experience, it is primarily an emotional response that is expressed by Christians in the face of evolutionary evidence, one of fight or flight, rarely a measured logical response. This is probably why it has grown into such a touchy, incendiary topic.

of aging.

The present general theory of aging cuts this knot in a simple, natural way. Within its framework, natural selection, far from fostering aging, is seen to be always and only an implacable foe of aging—as I will now undertake to demonstrate.

Longevity and Natural Selection

We have just seen that changes to the design of a machine can significantly alter its spectrum of congenital diseases and thereby impact its longevity. Biological organisms are mutable, self-replicating machines. Mutation alters machine design, creating diversity in offspring. Thus, longevity will vary in biological offspring. Longevity is a heritable trait. As such, it is subject to natural selection.

Longevity Principle

What effect does natural selection have on the longevity trait? My exploration of this question causes me to formulate the following simple longevity principle:

Longevity Principle: Successive generations of mutable, self-replicating machines tend to increase in longevity.

This principle may be empirically justified as follows. First, observe that there are only three possibilities. Successive generations of mutable, self-replicating machines may tend to:

1. decrease in longevity,
2. conserve longevity, or
3. increase in longevity.

Next, consider the long-term outcome of each of these three possibilities:

1. Self-replicating machines tending to decrease in longevity from one generation to the next will eventually go extinct.
2. The only way to leave longevity unchanged long term is for there to be equal probability for longevity to trend either up or down on the short term. The statistics of the well-known random-walk-near-a-cliff problem assure us that this case, too, will end in extinction.

3. Only self-replicating machines tending to increase in longevity from one generation to the next are able to go on propagating indefinitely.

Finally, observe that there exists but one experiment on this at present. This is the experiment involving mutating, self-replicating organisms on earth. Science reports fossils of microorganisms in rocks with measured ages of 3.5 billion years. Though the experiment appears to have been running for 3.5 billion years, in all this vast expanse of proleptic time the predicted outcome of the first two possibilities—universal extinction—has not been realized. Rather, what is presently observed is existence of a plethora of long-lived (i.e., months, years, decades, centuries, and even millennia) organisms. Thus the Longevity Principle—that successive generations of mutable, self-replicating machines tend to increase in longevity—is seen to work in this solely available instance.

This empirical justification may be augmented by a simple theoretical justification. Mutable, self-replicating machines produce offspring varying in their ability to stave off death. Offspring best able to stave off death have more time in which to produce offspring. Hence, those mutable, self-replicating machines which are best able to stave off death are likely to leave more offspring. As a result, the machine population will shift with time toward machines which are better able to stave off death. Thus, longevity is naturally selected for, causing successive generations of mutable, self-replicating machines to tend toward increasing longevity.

Impediments to Increasing Longevity

Though the longevity principle states that the trend from generation to generation is to increase in longevity, it is clear that this trend will not be linear in the general case. It is also clear that the incremental change in longevity with time is unlikely to be monotonic. Two obvious impediments to increasing longevity are increasing complexity and changing environment.

Increasing Complexity

Remedies for congenital diseases generally entail increasing complexity, but increasing complexity increases the number of potential congenital diseases.

For a four-stroke internal combustion engine, the problem of frictional wear of parts is alleviated by pumping oil to moving metal joints. This frees the operator from having to remember to mix oil with the gasoline. Once again, the lubrication of rubbing metal surfaces greatly increases the longevity of the engine relative to an unlubricated engine. But this method of lubrication also significantly increases the complexity of the engine. Additional parts needed, to implement engine self-lubrication, include an oil reservoir, an oil pump, and an oil filter. Each of these parts has potential to fail and cause death of the engine. Thus, while adding the self-lubrication functionality increases engine longevity, it also adds more congenital diseases, making further gains in longevity comparatively more difficult to achieve.

Changing Environment

Environmental changes can lead to large losses in longevity.

In the mechanical machine realm, this is analogous to loss of lubricating oil from the four-stroke engines' environment. Since the engines do not make oil, oil must be supplied to the engines from the external environment. If the environment changes in such a way as to cut off supply of oil to the engines, then the engines will revert back to their much shorter unlubricated longevity.

This is, according to the special theory of aging, what has happened to humans. Mid-Holocene loss of the anti-aging vitamins, methylphosphinic acid (MePiA) and methylphosphonic acid (MePA), from the environment has reduced human longevity from near one thousand years back then, to under one hundred years at present.⁶ Fortunately, discovery of these vitamins allows human longevity to be restored just as surely as a resupply

⁶Gerald E. Aardsma, *Aging: Cause and Cure* (Loda, IL: Aardsma Research and Publishing, 2017). www.BiblicalChronologist.org.

of oil to four-stroke engines enables their longevity to be restored.

Conclusion

According to the general theory of aging presented here, imperfect design gives rise to congenital diseases, one or more of which dominates, progressing with age and ending ultimately in death. For mutating, self-replicating machines, mutation occasions both design improvements and concomitant novel imperfections. Natural selection is ever in the process of eliminating these imperfections, one congenital disease after another.

If natural selection were allowed to act for an infinite time in a static environment, then all congenital diseases would be eliminated and immortal machines would result. But this is the (hypothetical and unattainable) final end point. The starting point is at the other end of the scale, populated by very mortal machines.

Earth's biosphere, in its present form, exists somewhere between these two termini. We learn from the historical sciences that earth's environment has been far from static. Nonetheless, earth's biota has clearly progressed a considerable distance along the longevity scale, sporting some species, such as the bristlecone pine (Figure 1), having life spans measured in thousands of years. And, according to the longevity principle, we should not regard earth's biota, including humans, as now static in regard to longevity, but rather as progressing toward yet greater life spans. ◊



Figure 1: Bristlecone pine tree, White Mountains, Nevada. (By Dcrjsr [CC BY-SA (<https://creativecommons.org/licenses/by-sa/3.0>)], via Wikimedia Commons.)

The Biblical Chronologist is written and edited by Gerald E. Aardsma, a Ph.D. scientist (nuclear physics) with special background in radioisotopic dating methods such as radiocarbon. *The Biblical Chronologist* has a fourfold purpose:

1. to encourage, enrich, and strengthen the faith of conservative Christians through instruction in biblical chronology and its many implications,
2. to foster informed, up-to-date, scholarly research in this vital field,
3. to communicate current developments and discoveries stemming from biblical chronology in an easily understood manner, and
4. to advance the growth of knowledge via a proper integration of ancient biblical and modern scientific data and ideas.

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