

The Biblical Chronologist

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

(Ecclesiastes 7:24)

Volume 15, Number 5

November 11, 2025

The End of Modern Human Aging Appears Imminent

An important development in the theory of modern human aging appears to bring this theory to completion. Practical implementation of this new development is underway. A complete cure of modern human aging, restoring youthful health, appears imminent.

Modern human aging contrasts with more ancient human aging. Before Noah's Flood, 3520 B.C., humans lived an average of 925 years. Today humans live an average of just 73 years. The theory of modern human aging explains this difference.

The most recent edition of *Aging: Cause and Cure* codified the theory of modern human aging as follows:¹

Theory of Modern Human Aging:

Modern human aging is a congenital nutritional deficiency disease syndrome (called MHA) of two newly-discovered, closely-related vitamins: methylphosphonic acid (MePiA) and methylphosphonic acid (MePA).

Vitamin MePiA functions as an antioxidant within the mitochondria, protecting them from free radical damage due to reactive oxygen species (ROS). The fundamental cause of death resulting from vitamin MePiA deficiency is cellular energy starvation due to decreased energy output from ROS-damaged mitochondria.

Vitamin MePA functions as its own unique vitamin, separate from vitamin MePiA. It is involved in multiple biochemical pathways, similar to members of the traditional vitamins, and like them its dietary deficiency gives rise to its own unique, potentially fatal disease.

This theory is now in need of a slight amendment. Specifically, the phrase "ROS-damaged mitochondria," at the end of the second paragraph, needs to be replaced with "ROS-sidelined mitochondria." This change is highlighted using italics below:

Theory of Modern Human Aging:

Modern human aging is a congenital nutritional deficiency disease syndrome (called MHA) of two newly-discovered, closely-related vitamins: methylphosphonic acid (MePiA) and methylphosphonic acid (MePA).

Vitamin MePiA functions as an antioxidant within the mitochondria, protecting them from free radical damage due to reactive oxygen species (ROS). The fundamental cause of death resulting from vitamin MePiA deficiency is cellular energy starvation due to decreased energy output from *ROS-sidelined* mitochondria.

Vitamin MePA functions as its own unique vitamin, separate from vitamin MePiA. It is involved in multiple biochemical pathways, similar to members of the traditional vitamins, and like them its dietary deficiency gives rise to its own unique, potentially fatal disease.

¹Gerald E. Aardsma, *Aging: Cause and Cure*, 3rd ed. (Loda, IL: Aardsma Research and Publishing, 2023). www.BiblicalChronologist.org.

Though this change is slight, its impact is profound, and most welcome, as will become apparent

below. The need for this change has come about as follows.

When it finally had clarified, back in 2019, that what had been called “vitamin X” was not the lone, newly-discovered vitamin, MePA, but rather a new vitamin duo, MePA together with MePiA,² the consequent need was to explain what physiological role MePiA played. The most obvious hypothesis, at the time, was that lack of MePiA renders mitochondria dysfunctional due to wildfire ROS damage of mtDNA.

The resulting disease state of thus-damaged mitochondria is called microheteroplasmy, and microheteroplasmy has been envisioned by me since that time as the residual, aging-induced disease needing to be cured once aging itself has been cured via daily dietary supplementation with Dr. Aardsma’s Anti-Aging Vitamins, a formulation of vitamins MePA and MePiA.

It now appears that microheteroplasmy, though a real consequence of dietary deficiency of MePiA, will be minor. The major residual, aging-induced disease now appears to be CD38 overexpression disease. As previously explained, this disease is also a consequence of wildfire ROS within the mitochondria.³ CD38 is an enzyme which destroys NAD and its precursors. Its overexpression results in NAD deficiency, which has many potential physiological consequences including, most importantly, energy starvation of cells.

The replacement of microheteroplasmy disease with CD38 overexpression disease as the major residual aging-induced disease has important consequences. While microheteroplasmy results in dysfunctional mitochondria, CD38 overexpression results only in sidelined mitochondria. The end result, energy starvation of cells, is the same in both theoretical scenarios, but the outcome for the mitochondria is quite different. Microheteroplasmy breaks mitochondria. CD38 overexpression merely sidelines them.

Using an automobile as an analogy for mitochondria, microheteroplasmy causes a break-

down of the automobile while CD38 overexpression causes no damage to the automobile. CD38 overexpression results in NAD deficiency. This may be thought of as a rationing of NAD. The effect on mitochondria of rationing NAD is comparable to the effect on an automobile of rationing gasoline. The automobile is not damaged by gasoline rationing. It is simply not able to be used as much.

This is very important. Just as ending gasoline rationing restores a sidelined automobile to full service, so ending NAD rationing—by curing CD38 overexpression disease—restores sidelined mitochondria to full service. This has potential to restore the aged human body to full health—to restore youthful health. This is not aging put on hold. This is youthfulness restored.

Clearly, the change from “ROS-damaged” to “ROS-sidelined” in the theory of modern human aging is an important one.

Experimental Evidence

The experimental evidence prompting this change is shown in my previously-published plasma NAD+ concentration graph, reproduced here again this issue (Figure 1).⁴ This shows a brief return to youthful levels, due to dietary supplementation with NR, followed by a rapid decline toward zero due to continued supplementation with excessive NR.

The key observation is that my mitochondria did not trigger more CD38 expression (due to excessive NR producing excessive NAD+, overdriving oxidative phosphorylation with consequent excessive ROS production), pushing plasma NAD+ levels lower than ever before, until *after* youthful levels of plasma NAD+ had been restored. This says that my mitochondria were able to handle youthful levels of NAD+ just fine. This would not have been true of dysfunctional mitochondria.

Implication

This change implies that a complete practical cure for modern human aging is now imminent.

²Gerald E. Aardsma, *Addendum to Aging: Cause and Cure* (Loda, IL: Aardsma Research and Publishing, July 26, 2019). www.BiblicalChronologist.org.

³Gerald E. Aardsma, “Dietary Supplementation with NR for the Post-aging Diet: Part 5,” *The Biblical Chronologist* 15.4 (September 18, 2025): 1–4. www.BiblicalChronologist.org.

⁴Gerald E. Aardsma, “Dietary Supplementation with NR for the Post-aging Diet: Part 5,” *The Biblical Chronologist* 15.4 (September 18, 2025): 3. www.BiblicalChronologist.org.

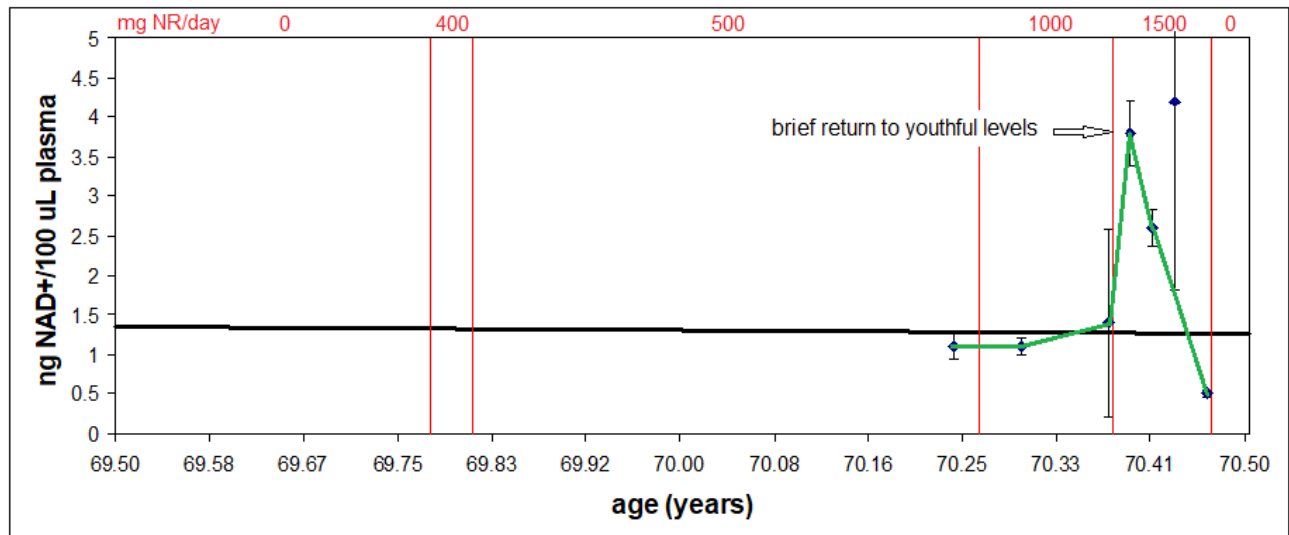


Figure 1: In-house measurements of NAD⁺ concentration in my blood plasma (blue data points). The green curve connects data points to show the overall behavior of the data. The heavy, near-horizontal black line represents normal NAD⁺ levels versus age for individuals supplementing their diets with neither NR nor Dr. Aardsma's Anti-Aging Vitamins. The red vertical lines delimit time intervals having the different daily NR intake amounts shown in red above the graph. The vertical bars shown with the data points are not error bars. They are intended only to give a visual impression of the quality of the measurement. The green curve excludes the poorest-quality data point because it is far from the overall behavior, likely due entirely to the poor quality of the measurement.

The United States Social Security Administration (SSA) actuarial data appear to guarantee this imminency. I have previously modeled and discussed these remarkable data in depth.⁵ The current change substitutes CD38 overexpression disease for microheteroplasmy disease, but this changes neither the model nor its implications. The SSA data show us definitively that there are two, and only two, components to modern human aging disease. Both components now appear to be understood.

One component behaves similarly to a traditional vitamin deficiency disease. This is the MePA deficiency disease contribution to modern aging deaths.

The MePA component has been fairly easy to understand from the very beginning because of its similarity to traditional vitamin deficiency diseases. A complete cure of this component is afforded merely by the inclusion of an appropriate amount of MePA in one's diet. This cure has been effected since 2017 when vitamin MePA was first made available to the public through the BiblicalChronologist.org web site.

The other component exhibits a novel saturat-

ing behavior. This is the MePiA contribution to modern aging deaths.

In contrast to MePA, the MePiA component has been difficult to understand, having no familiar analog.

In contrast to the ubiquitous physiological utility of each individual traditional vitamin, physiological utility of vitamin MePiA appears to be restricted to the sole function of deactivating mitochondrial ROS. What had been the physiological consequences of failure to deactivate mitochondrial ROS? This was a difficult question.

It was clear that, whatever they might turn out to have been, they were not all set right merely by inclusion of an appropriate amount of MePiA in one's diet. Though MePiA deficiency had been corrected by such dietary supplementation, aging-induced disease persisted—the individual appeared yet to be aging.

The answer to this difficult question now seems clear. Failure to deactivate mitochondrial ROS breaks the body's natural NAD regulation system, causing a progressive chronic overexpression of CD38 which yields a pernicious NAD deficiency.

This new clarity implies that the quest to understand the physiology of modern human aging is now complete. The SSA data reveal that there are but two component diseases to the modern human

⁵Gerald E. Aardsma, *Aging: Cause and Cure*, 3rd ed. (Loda, IL: Aardsma Research and Publishing, 2023), 181–201. www.BiblicalChronologist.org.

aging syndrome, and both components seem now to be understood.

Conclusion

The fashioning of a remedy for CD38 overexpression disease, now in progress in ARP Laboratory, is expected to complete the cure of the modern human aging syndrome. This is not a certainty, of course. There may yet be unforeseen twists and turns in store. But, based on my labors in this field for over forty years, it seems to me that we have very likely now come to the end of this quest to cure modern human aging. If so, human life spans—our life spans—are expected to return to the pre-Flood average of 925 years.

Curing CD38 overexpression disease will not happen overnight. Based on the rate of progress to the present time, it is expected to take some months or maybe even more than a year. But, given that this aging syndrome has been with the human race since Noah's Flood, i.e., for over 5,500 years, it seems appropriate to say that its complete cure now appears imminent. ◊

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 principles.

The Biblical Chronologist (ISSN 1081-762X) is published by:

Aardsma Research & Publishing
301 E. Jefferson St.
Loda, IL 60948

Web address: www.biblicalchronologist.org.

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