Vitamin MePiA Cures Aging 2

The theory of Aging 2, introduced last issue, refines the two-phase theory of modern human aging, replacing the generic “Aging 2: a mitochondrial genetic disease” with the more specific “Aging 2: zeroed-psephomere disease” as follows.

Two-phase Theory of Modern Human Aging: Modern human aging is a syndrome of three diseases:

1. Aging 0: congenital vitamin MePA deficiency disease,
2. Aging 1: congenital vitamin MePiA deficiency disease, and

This theory, corroborated by modern actuarial data, is explicit about the role played by vitamin MePiA deficiency disease in inducing Aging 2. The fact that a dietary deficiency of vitamin MePiA induces Aging 2 immediately implies a preventive role for MePiA with respect to Aging 2—one might say that vitamin MePiA is nature’s prophylactic for Aging 2. This has been clear since discovery of Aging 2. The urgent question has been whether MePiA might also play a curative role with Aging 2.¹


Curing Aging 2

The theory of Aging 2 says that loss of mitochondrial DNA (mtDNA) psephomeres is the fundamental cause of Aging 2. Psephomeres, somewhat analogous to telomeres, keep track of the generation number of each mtDNA copy as part of a mitochondrial copy error suppression mechanism. Loss of psephomeres breaks this mechanism, which allows copy error mutations to accumulate within the mtDNA pool. To cure Aging 2, restoration of mtDNA psephomeres is required.

The idea of reaching into the mitochondria of all 37 trillion cells of the human body to restore psephomeres to thousands of copies of mtDNA per cell does not appear to be technically possible at present, nor would one wish to employ this approach even were it to be technically feasible. Loss of psephomeres results in accumulation of mtDNA genetic mutations. Restoring psephomeres to mutated mtDNA restores ability to suppress further mtDNA copy error mutations, but it does not correct already existing copy-error mutations.
Thus, Aging 2 would be cured, but existing mitochondrial genetic diseases induced by Aging 2 would not be cured. To do the job right, not only the psephomere but also the rest of the mtDNA molecule needs to be restored to its original condition.

Fortunately, according to the theory of Aging 2, the body’s own healing mechanisms, when armed with an adequate provision of vitamin MePiA, do the job right. This is revealed by the temporary reversal of aging which Noah experienced during the Spike.

Noah’s Aging Reversal

Recall that the Spike was a brief period of time (69 years) immediately following the Flood when natural environmental levels of MePiA were temporarily very high. Also recall that pre-Flood individuals are thought not to have experienced Aging 0 disease because MePA was likely present in adequate amounts in drinking water sources back at that time. This means that Noah did not have Aging 0 disease when he entered the Spike. Noah also did not have Aging 2 disease when he entered the Spike. Aging 2 was induced in individuals living before the Flood only at age 800 years, and Noah was only 600 years old when he entered the Spike. The only aging disease which Noah had when he entered the Spike was Aging 1 disease, which he had been afflicted with since birth. Congenital Aging 1 disease afflicted all pre-Flood individuals. They were all somewhat deficient in MePiA, though not nearly as deficient in this vitamin as are modern humans.

Because of his congenital Aging 1 disease, Noah was three-quarters of the way to onset of Aging 2 disease when he entered the Spike.

Upon entering the Spike, Noah’s Aging 1 disease (i.e., his MePiA deficiency) was quickly cured because of the large environmental abundance of MePiA during the Spike. Remarkably, his physiological age then began to reduce one year for every dozen calendar years.

The theory of Aging 2 now makes it possible to put this reduction in physiological age into biogenetic terms. Noah’s physiological agedness during the Spike simply equates to his average psephomere length. Recall that shortening of psephomeres is the mechanism by which the biological mechanical timer controlling onset of Aging 2 measures time. The fact that Noah’s physiological age reduced, means that his biological mechanical timer ran backwards during the Spike. This means that Noah’s psephomeres, which had been declining in average length during the first 600 years of his life, increased in average length during the Spike.

Increasing Average Psephomere Length

How is this increase in average psephomere length to be explained? Two considerations are necessary to yield an explanation in this case.

First, during the Spike, Noah’s diet provided him with all the vitamin MePiA his body could use. Free-radical damage of mtDNA would, therefore, have dropped nearly to zero. Thus, replacement of damaged mtDNA would have dropped nearly to zero. As a result, the rate of shortening of psephomeres would similarly have plummeted.

This consideration alone results in the near-suspension of progress of Noah’s mechanical timer toward induction of Aging 2. But this is not enough, of course. The biblical life span data make it clear that progression toward Aging 2 not only suspended but indeed reversed during the Spike.

How is it possible for average psephomere length not only to stop shortening but also to begin increasing? To explain this, a second consideration

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is needed. The required increase in Noah’s average psephomere length appears to demand that old cells, having short psephomeres, were slowly being replaced during the Spike by new cells having longer psephomeres.

This is entirely believable. Cells are generally designed to be replaced. Stem cells provide new replacements for old, worn-out cells. This is a slow process. The average age of cells in the human body today is 7 to 10 years. This is an average. Some body tissues replace themselves much more rapidly while others replace themselves much more slowly. But the important point is that stem cells have potential naturally to replace old cells with new cells.

It is well known that old cells have short telomeres. Short telomeres signal that the cell is old and that it is time for it to be replaced. When stem cells produce replacements for old cells, the new cells have long telomeres. Noah’s healing during the Spike teaches us that something similar must be the case with psephomeres. Stem cells must be capable of replacing old cells containing mtDNA having short psephomeres with new cells containing mtDNA having long psephomeres.

**The Crucial Point**

This leads immediately to the crucial point. Noah’s healing during the Spike teaches us that not all cells of the body advance in psephomere shortening at the same rate. Evidently, stem cell psephomeres shorten more slowly (if at all) than differentiated cell psephomeres. This means that stem cells are either not subject to Aging 2 at all, or that the induction of Aging 2 in stem cells is significantly delayed relative to differentiated cells.

And this means that, as long as the stem cells are free of Aging 2, once the body’s internal free-radical inferno has been quenched by adequate cellular levels of vitamin MePiA, old Aging 2 diseased cells can slowly be replaced by healthy cells furnished by the stem cells, curing Aging 2.

**The Healing of Aging 2**

According to the theory of Aging 2 and the reversal of Noah’s aging during the Spike, modern humans adequately supplementing their diets with MePiA can expect their bodies to stop trending toward increasing agedness and to begin trending *slowly* toward increasing youthfulness. They can expect their Aging 2 disease to heal at a rate of about one physiological year per dozen calendar years, similar to what Noah experienced during the Spike. An individual who starts supplementing vitamin MePiA at age 40 can expect that when he reaches age 52 his body, on average, will be back to what it was when he was 39.

Notice, however, that this healing of Aging 2 is more precarious and complex than just the increase in average length of psephomeres experienced by Noah during the Spike. While Aging 2 is simply zeroed-psephomere disease, zeroed-psephomere disease induces mtDNA-copy-error disease. Copy errors yield mtDNA genetic mutations. Thus, Aging 2 induces a host of potential mtDNA genetic mutation diseases.

Thankfully, these are not congenital genetic mutation diseases having propagated from the zygote (i.e., the fertilized ovum). Aging 2 is, we believe, only induced subsequent to the first decade of life. So, genetic mutations due to copy errors induced by Aging 2 begin to exist in human cells only subsequent to the first decade of life. While congenital, hereditary mtDNA genetic mutations might be expected to be present in every cell of the body containing mitochondria, Aging 2 induced mtDNA genetic mutations will initially be local to specific (unlucky) mtDNA copies in specific (unlucky) mitochondria in specific (unlucky) cells. One may think of progression of the clinical expression of Aging 2 disease as being due to new mtDNA genetic diseases of varied sorts sprouting up in local tissues all over the body. These diseases then get propagated by replication in several steps:

1. copying of the mutated mtDNA,

2. replication of the diseased mitochondrial containing the mutated mtDNA within a given cell, and

3. replication of that diseased cell, yielding other, similarly diseased cells.

The progression of all of this varied, local, mtDNA genetic disease clearly has potential to be quite a complex mess. Its main outcome is expected to be loss of proper energy supply to the cells, this being the mitochondrias’ primary role. But this is
not the only potential outcome, by far. Whether this mess can be cleaned up in its entirety by adequate cellular levels of vitamin MePiA is, so far, unknown. Clearly, the younger one is when beginning to supplement with MePiA, the better the chances of a complete cure of Aging 2 plus its induced mtDNA genetic mutation diseases.

Nevertheless, two important things are known and should not be lost sight of:

1. Adequate cellular levels of MePiA will slow progression of this mess. This is known because reduction of free-radical damage is sure to reduce or eliminate the copying of mtDNA needed to replace damaged mtDNA, and copying of mtDNA is how mtDNA-copy-error disease intensifies and spreads.

2. If the progression of this mess doesn’t kill one first, adequate cellular levels of MePiA will ultimately eliminate Aging 2 (i.e., zeroed psephomeres) from one’s body, curing both Aging 2 and its associated mtDNA mutations. This will not happen overnight. Depending on one’s age when one begins to supplement with vitamin MePiA, it may take decades or even centuries. But it will happen.

But now this doubt appears unfounded. The theory of Aging 2, identifying Aging 2 with zeroed psephomeres, combined with the reversal of Noah’s aging during the Spike, now restore the original expectation—MePA plus MePiA constitute the total cure of modern human aging.

We have come full circle, but we are not back exactly to the same place we started. I am reminded of T.S. Eliot’s musing:

We shall not cease from exploration
And the end of all our exploring
Will be to arrive where we started
And know the place for the first time.

Our knowledge of modern human aging has now deepened considerably, driving out whatever residual sanguine naïveté there may have been regarding the long and hazard-filled path back to aging-free health for those of us afflicted with Aging 2 disease.

Nevertheless, the discovery that vitamin MePiA is curative of Aging 2 is a cause for great thanksgiving.

Conclusion

It appears that we have now come full circle.

The thinking, once it was understood that there were two anti-aging vitamins, was that these two vitamins would constitute the total cure for modern human aging, just as vitamin C constitutes the total cure for scurvy. The expectation was that vitamin MePiA together with vitamin MePA would slowly—at a rate of one physiological year per dozen calendar years—heal (i.e., cure) human aging when taken in adequate daily amounts.

Then came the discovery that, unlike scurvy, human aging is a two-phase disease with an induced mitochondrial DNA disease component. Modern medicine struggles to deal with mtDNA genetic diseases at present. Cures are, so far, unknown. Thus, the discovery of this mtDNA disease component in human aging cast doubt on the idea that the two anti-aging vitamins constituted a total cure of modern human aging.