Telomeres and Longevity

By: Clark M. Thomas

Abstract

Telomeres in humans are caps at the two ends of chromosomes that largely determine our normal life expectancy. Chromosomal senescence typically approaches before our death, and no amount of botox can stop it. Interestingly, some people live longer than their seventies, with delayed senescence. Even though quality of life is more important than quantity, most of us want both quality and quantity, with minimal time spent in senescence. To what degree could this optimum be under our control? If we all could approach seeming immortality, what could that life extension mean for our individual and social lives?

Most people drift through life inside a consciousness fog that minimizes personal mortality, even while we reluctantly realize how "gravity seems to accelerate" as we age. We also assume that elaborate ideas of impending death belong to our chosen species alone. However, multiple advanced species, such as elephants and orcas, have individual death as a core part of their psychosocial consciousness.

Humans alone have, with the help of various religions, elevated self-serving consciousness into formal ideas of deity-enhanced afterlives. This global eternity fixation is a fundamental reason why we have become this planet's only *global hyperkeystone species*, for better and worse.**[1]**

Billions of us welcome physical death, believing we go onward to eternal Heaven, thanks to a generous Jesus who is ready to reward us all with forgiveness for our confessed sins. The extreme version of this Easter fantasy is called *universalism,* where every conscious creature enjoys a post-mortem Heaven, passing through nothing worse than Purgatory.

Mythical, pre-scientific religious traditions of virtually infinite reincarnation life cycles, through *transmigration of souls,* help to keep billions of otherwise conscious people away from fully valuing the few years they do have.

When I was a child I considered the value of one life wherein we choose much of our personal future. I concluded it is better to die at age 10 trying to save the life of a family member in mortal danger, than to die at age 95 after a smooth life of selfish greed. I learned *it is quality, not just quantity, that is the true measure of a worthy and full human life*.

That pure insight doesn't go well in many parts of the world: Most greedy people, if given the chance, would value quantity over quality, with Jesus or some similar rescue raft standing by to redeem repentants. So far, artificial intelligence (AI) is not part of this conversation; but that may change sooner rather than later.

Enter the modern discovery of telomeres: For the hopeful, telomeres are a channel for eventual immortality, with some life extension. Spiritual myopia and fear are common companions. That is why we have robust objective telomere science. The quest for long quantity/quality lives is becoming a trillion-dollar trend.

The idea of living well and long is an ideal. Every life has bumps on the road. Most of us myopically navigate those bumps until "gravity gets us" in our seventies. Compare how one full human life measures up against the seventy thousand plus species years of genetically modern humans. Why can't we at least live just twice as long as ordinary people do? That is a fair question, but the best that science offers is a modest boost in statistical longevity, perhaps a decade or two, built mostly around enhanced *chromosomal telomeres*, and good individual fortune.

In that light, let us first look at the classical physics dimension of chromosomal telomeres, focusing on what we maybe could do about their progressive shortening. Here is some honest science, not fanciful delusion. Realistic extension of our "statistical fate" is not boring. It is a path toward optimizing quantity and quality.

The biological science of diet and aging is within the classical physics dimensions that we can experimentally verify. There is now emerging a lot of data that gives us hope for some quality longevity. However, a rope can only be stretched so far before it breaks.

Telomeres are DNA caps at the ends of our chromosomes. These caps wear down somewhat during each of the average 52 divisions. This number is known as the *Hayflick limit*, after which a chromosome is senescent. As death approaches, the final stroke could come from something like cancer that was allowed by incorrect replication from telomeres grown too short to

prevent mutations. *Murphy's law* applies the longer we live: "If something bad can happen, it eventually will." Many television ads for burial insurance like to insert the goofy Murphy phrase, "If something happens…"

The emerging literature of telomeres, is impressive. A collection of 150 science references from 2023 is available from the federal government.**[2]** There are additionally more recent quality science references, a few of which will be described in context below. The *summary abstract* from that 2023 trove is reprinted below:

> "The ends of human chromosomes are defended by DNA–protein complexes named telomeres, which inhibit the chromosomes from fusing with each other and from being known as a double-strand break by DNA reparation proteins. Telomere length is a marker of biological aging, and disfunction of telomeres is related to age-related syndromes. Telomere attrition has been shown to be accelerated by oxidative stress and inflammation. Telomere length has been proven to be positively linked with nutritional status in human and animal scientific research, as several nutrients influence it through mechanisms that imitate their function in cellular roles including oxidative stress and inflammation. Data reported in this article support the idea that following a low-in-fat, and rich-plant polyphenols food diet seems to be able to slow down the shortening of telomeres."

The key general takeaway from all these studies is how we can indeed have some influence over how long, and how well, we can live. Still, the total potential life span among Earth's over eight billion people is hardly more than 115 years. Opportunities may be better in the realm of quality over quantity. (Nothing in this essay of mine should be interpreted as diagnostic for clinical prevention and treatment.)

When I was a pre-teen I tried to help other pre-teens in my neighborhood quit tobacco smoking. Almost all of these wannabe adults refused to envision themselves beyond their smoking peers. My "best results" were achieved after fellow pre-teens would say they didn't care if they lived eight years less than non-smokers. I then reminded them that the real loss starts decades sooner, with reduced beauty and vitality before a hastened death. Sadly, smoking is just one way to stress and thus shorten our telomeres.

Today I have a friend who has worked as a respiratory therapist in hospitals for more than twenty years. I recently asked him how many of his

gasping hospital patients have been smokers. He said virtually all of his seriously ill patients have been tobacco smokers. (Thank you, Big Tobacco.)

Toward Optimizing Ourselves

Very few of us have ideal health upbringing. Nevertheless, young bodies have more ability than old bodies to self-repair. Ironically, when we are in youth best able to optimize our longer telomeres, we are least likely to do so. When we are least able as seniors to restore our shortened telomeres, we are most likely to try something. Life itself offers us many opportunities to be the best we can be at each age. The trick is to know and act on what is best for us at each age. Here below are some ideas of general value that may interest you on your personal journey from alpha to omega:

There is no single magic bullet for optimizing telomeres. We could, for example, do a lot of ***aerobic exercise*** for decades in our middle years. That alone may be very good for the efficiency of our cardiovascular systems and emotional feelings. Full-body lymphatic drainage can be enhanced by aerobic activities. I spent almost twenty years from my mid-twenties until my mid-forties vigorously running, hiking, and riding mountain bikes several times each week. I even ran the Pikes Peak Marathon at age 35. Now in my seventies I still have optimal systolic and diastolic numbers, along with optimal beat rates. Also, I have never smoked, and thereby never filled my lungs with garbage. During my earlier decades I didn't even know what a telomere was, nor know the key role of efficient brain waste drainage.

I will soon enter the statistically uncommon cohort of **SuperAgers**.**[3]** That's where and when people who are eighty-plus still have nimble minds working as well as in their middle decades. Importantly, nimble senior brains typically have as much a*myloid plaques* as do cohorts entering dementia. Have we misidentified the real source of senior dementia, and are we clinically treating the wrong thing?

A key difference between alert SuperAgers, and those developing degeneration, is the much less presence of **tau tangles: [4]**

> **"**The tangles are made of the tau protein which [normally] forms structures that transport nutrients within the nerve cell. These tangles disrupt the cell's transport system, hampering communication within the neuron and preventing nutrients from performing their particular job within the cell. The end result of tangle formation is cell death."

Recently, a study of the effects of daily *2000 units of common Vitamin D3* demonstrated the power of this one vitamin in amounts only twice the daily recommendation to modestly protect telomere lengths.**[5]** Beyond vitamin D3, an even better strategy may be the daily food consumption of quality *phytochemicals,* preferably organic from multiple sources.**[6]** Even *statins* have demonstrated possible bonus benefits, due to their anti-inflammatory properties.

The Inflammation Hypothesis

If you have carefully followed me through this scientific weed thicket, you may have noticed an *important clue* for people of all ages. Even this model is not a panacea that supersedes the normal protective functions of telomeres. This elegant hypothesis is that *inflammation between and among brain cells is a major risk factor for early aging*, and an accelerant for shortening of telomeres.

We like to think that daily fecal and urinary action is how we get rid of metabolic waste. Surprisingly, our skin is our largest excretory organ, in surface area at least. This is all well and good, but it is most important to ask how the rest of our body collects metabolic waste, and how it all is delivered to our skin and alimentary canal. Our brain's initial delivery channeling is critical. It has also been hypothesized that the *glymphatic drainage* occurs most efficiently *during long sleeping*, which could be critical for keeping daytime conscious minds sharp.

Brain cells are very active, creating a lot of *soluble waste products* that must be properly eliminated within glymphatic channels, leading to our liver, kidneys, lungs, and skin. When the brain's "plumbing" is even partially blocked, the entire body suffers. Telomeres have been shown to be shorter among cohorts with chronic stress, than among those leading a naturally hygienic life. Genetics plays a role, but that role can be less than life choices, which is why people who choose to smoke for decades generally age more quickly. Their brains have been floating in a pool of dirty brain fluids.

In 2021 I wrote a thesis on an extremely rare disease called *Alternating Hemiplegia of Childhood.***[7]** This genetic syndrome appears not long after birth, and haunts the sufferer until an earlier death. Because there is a genetic component that can interfere with the brain's drainage system, the innocent child has periodic episodes of near paralysis until his or her brain drains and temporarily resets.

I hypothesized that to develop full AHC symptoms another common risk often is present. That is an allergy to wheat, barley, and rye products, all containing gluten, which is a mixture of two proteins.**[8]**

Celiac disease affects the small intestine's lining, but actually it works on multiple areas of the body as both acute and chronic inflammation, among other symptoms. So, in AHC we have perpetual brain dysfunction caused by that rare rogue gene, and episodes of celiac inflammation energizing the body's severe episodes. The best way to minimize that evil synergy may be to not eat inflammatory gluten products. In the best cases some children with rare AHC can thereby live beyond their childhood, with gluten-free food.

In common adult senility *drainage issues are mostly linked to tau protein tangles, not to amyloid plaques*, and are a key for understanding the much larger population of disabled people without the AHC genetic mutation.

This year, in 2026, the normal brain cerebrospinal glymphatic drainage system was clearly mapped, flowing ultimately to our detoxifying liver.**[9]** Real science is always searching for real solutions. What great new things will we know by 2036?

Enhanced attention to inflammation, especially inside the brain, has the potential to also help revolutionize the telomere story, along with the story of avoidable misery for many thousands of American seniors. It would be ideal for most adults to welcome the wisdom of age, not to fear a frailty fog.

References

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