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The Telomere Scam

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STORY AT-A-GLANCE

- Shorter telomeres are said to indicate increased risk of premature death; longer telomere length has therefore been assumed to represent health and longevity
- Research published in The New England Journal of Medicine (NEJM) revealed long telomeres are linked to cancer and clonal hematopoiesis of indeterminate potential (CHIP), a blood disorder
- Shorter telomere length is linked to degenerative diseases such as Alzheimer's disease and heart disease, while longer telomere length is associated with increased cancer risks
- Cells with long telomeres accumulate mutations, promoting tumors that might otherwise be prevented via normal telomere shortening processes
- Rather than relying on telomere length as a measure of longevity or disease, harness the power of lifestyle strategies to slow the aging process and improve your health span

Telomeres are repetitive nucleotide sequences at the end of each chromosome. Sometimes compared to the plastic tip on a shoelace, telomeres help protect DNA, preserving chromosome stability and preventing "molecular contact with neighboring chromosomes."[1]

Evidence suggests telomere length may predict morbidity and mortality, with shorter telomeres linked to an increased risk of premature death.[2] Longer telomere length has therefore been assumed to represent health and longevity, but the link is controversial.

New research suggests, in fact, that telomeres' link to aging may have been wrong all along.[3]

Is Telomere Attrition a Hallmark of Aging?

The idea that telomere length serves as a marker of aging has become scientific dogma. Writing in the journal Cell, researchers described telomere attrition as one of the hallmarks of aging that lead to most age-related disorders.[4] Telomeres, they noted, are especially vulnerable to age-related deterioration, and telomere shortening occurs during the normal aging process in humans and animals, such as mice.

When DNA damage occurs at telomeres, it leads to persistent harm and "deleterious cellular effects including senescence and/or apoptosis."[5]

Further, the team explained, when telomerase, a key maintenance mechanism of telomere length, is deficient in humans, it's associated with premature disease development, including those involving loss of regenerative capacity in tissues, such as pulmonary fibrosis, dyskeratosis congenita and aplastic anemia.[6] According to the Cell study:

> *"Genetically-modified animal models have established causal links between telomere loss, cellular senescence and organismal aging. Thus, mice with shortened or lengthened telomeres exhibit decreased or increased lifespan, respectively. Recent evidence also indicates that aging can be reverted by telomerase activation.*
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> *In particular, the premature aging of telomerase-deficient mice can be reverted when telomerase is genetically reactivated in these aged mice.*
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> *Moreover, normal physiological aging can be delayed without increasing the incidence of cancer in adult wild-type mice by pharmacological activation or systemic viral transduction of telomerase. In humans, recent meta-analyses have indicated a strong relation between short telomeres and mortality risk, particularly at younger ages."*

Risks Revealed for Short or Long Telomeres

Just because short telomeres have been linked to aging and disease, it doesn't mean long telomeres have the opposite effect. In fact, research published in The New England Journal of Medicine (NEJM) revealed long telomeres are linked to cancer and clonal hematopoiesis of indeterminate potential (CHIP) — a blood disorder.[7]

"Short telomeres were thought to be bad — people with premature aging syndromes had short telomeres — so, by analogy, long telomeres were thought to be good," study author Dr. Mary Armanios, professor of oncology at Johns Hopkins University School of Medicine, told The New York Times. "And the longer the better."[8]

Previous research by Armanios, who also directs the telomere center at Johns Hopkins University School of Medicine, and colleagues revealed, however, that the reality is much more complicated. While short telomere length (TL) was linked to disease, longer telomere length increased the risk of cancers, including lung, melanoma and glioma.[9]

"The upper threshold that increases the risk of these cancers is not known, but these recent findings add significant warning to the oversimplified interpretation of short TL being linked to aging and long telomeres to youth," they concluded in 2018. It seemed that having either very short or very long telomeres may be a risk factor for disease.

Researchers with UCSF School of Medicine and Stanford echoed this sentiment in 2020, revealing that shorter telomere length is linked to degenerative diseases such as Alzheimer's disease and heart disease, while longer telomere length is associated with increased cancer risks.[10]

"Genetically determined long and short telomere length are associated with disease risk and burden of approximately equal magnitude," they concluded.

Long Telomeres Linked to Cancer, Disease

The NEJM study involved 17 people with a POT1 genetic mutation, known to not only lengthen telomeres but also increase cancer risk. The study participants ranged in age

from 7 to 83 and had a variety of tumors, ranging from benign uterine fibroids to melanoma. They also had significantly longer telomeres than the average population — 90% longer in 13 participants and 99% longer in nine.[11]

While six of the participants had some signs of youth, including no gray hair even in their 70s, many had high rates of clonal hematopoiesis-related mutations. The mutations are linked to the development of blood and other cancers, and existed at much higher rates than expected in the general population.

One participant had cells with 1,000 mutations per clone, which the researchers believe began when the person was just 4 years old. "The long telomere length allowed the blood cell propagation since then," Armanios said.

The research suggests long telomeres are leading to CHIP and giving more time for cancer-causing mutations to develop. According to Armanios:[12]

> *"Our findings challenge the idea that long telomeres protect against aging. Rather than long telomeres protecting against aging, long telomeres allowed cells with mutations that arise with aging to be more durable … Cells with very long telomeres accumulate mutations and appear to promote tumors and other types of growths that would otherwise be put in check by normal telomere shortening processes."*

Telomere shortening, for instance, is said to represent a "major measurable molecular characteristic of aging of cells in vitro and in vivo," which may have developed as a mechanism to protect against tumors in long-lived species.[13]

Is DNA Methylation a Better Measure of Your Biological Age?

It's possible to determine your biological age, as opposed to your chronological age, by measuring your DNA methylation, and in head-to-head comparisons, DNA methylation is significantly more correlated to the aging process than telomeres.

DNA methylation is the silencing of gene transcription. Your genes have promoter sites at the beginning of the DNA strand, and methylation is measured at those sites. The level of methylation at the promoter site correlates to the degree of expression of that particular gene.

Ryan Smith is the founder of TruDiagnostic, a commercial testing system that **measures DNA methylation**. What's being measured is not your ability to methylate or not methylate. Rather, it measures the actual expression of your DNA. And, contrary to conventional genetic testing like 23andMe, which is done once, DNA methylation can be measured multiple times as the actual expression of your DNA is alterable and changes over time.

DNA methylation is a better marker of disease risk and health span than telomere length, Smith said during our 2022 interview.

> *"If you were to make sure that the telomere length never decreased in a cell, you'd still see methylation-related biological aging. If you made sure that the methylation age was reset, you would still see telomere length aging. So, there's two separate processes.*
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> *In a recent review, they actually looked at twins and tried to ascribe how much of the difference in their aging process was affected by these different markers. They said right around 2% of the variance in phenotypic aging was due to telomere length, whereas right around 35% of that was based on these epigenetic methylation clocks.*
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> *So, while they both might be important, we definitely would think that the DNA methylation clocks are significantly better."*

Antiaging Strategies That Work

While measuring your rate of aging, or biological age, is intriguing, harnessing the power of lifestyle strategies to slow the aging process and ward off disease can improve your

health span and quality of life. Simple antiaging strategies you can implement today include:

- **Vitamin D optimization —** Ideally, you want to maintain a blood level of 60 ng/mL to 80 ng/mL. Smith cited an interventional trial in which overweight participants reduced their biological age by 1.8 years on average, taking just 4,000 IUs of oral vitamin D a day for 16 weeks.

- **Optimize your metabolic flexibility —** Core strategies include time-restricted eating or intermittent fasting, and eating a diet high in healthy fats and low in refined carbs to optimize your insulin sensitivity. Also, eat your last meal each day at least three hours before bed.

- **Get regular exercise and daily movement —** In one study, among 1,481 older women included in the study, those who sat the longest were, on average, eight years older, biologically speaking, than women who moved around more often.[14]

  Another study touted exercise as a "possible cure" for the declines in mitochondrial biogenesis and mitochondrial protein quality commonly seen with aging. Not only did exercise training reverse or lessen age-associated declines in mitochondrial mass, but it also "decreased the gap between young and old animals in other measured parameters."[15]

- **Stress management —** According to Smith, people who meditate or engage in other stress-reduction strategies on a regular basis tend to age at a slower rate than those who don't.

- **Limit consumption of unsaturated fats —** Omega-6 linoleic acid (LA) is particularly harmful. It's highly susceptible to oxidation, causing oxidative stress, and can remain in your cells for up to a decade. So, you want to eliminate vegetable/seed oils, which are high in LA.

In terms of supplement options, glycine is a powerful longevity enhancer that's inexpensive and has a pleasant, slightly sweet taste. Research shows glycine extends

lifespan in worms, mice and rats while improving health in models of age-related disease.[16]

If there were any doubt about its importance, consider that collagen — the most abundant protein in your body[17] — is made mostly of glycine. It's also a precursor to glutathione, a powerful antioxidant that declines with age. To gain all of glycine's healing potential, doses of 10, 15, or 20 grams a day may be necessary. I personally take three teaspoons of it a day.

Collagen is also an outstanding source of glycine. You can boost your collagen intake by making homemade bone broth using bones and connective tissue from grass fed, organically raised animals. But remember, there's no magic potion to stop the aging process.

Methods that claim to stop aging by lengthening your telomeres appear to be misguided. And if your telomeres are too long — or too short — it could be indicative of disease. The fact remains that the best longevity benefits come from leading a comprehensively healthy lifestyle.

Sources and References

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