Running Against Time: The Effects of Endurance Exercise on Telomere Dynamics and Longevity

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Abstract

Telomere attrition, recognized as the hallmark of aging, is linked to an increased risk of certain negative health outcomes such as cardiovascular disease, metabolic dysfunction, and neurodegenerative disease. Regular endurance exercise is one potential solution to lengthening telomeres; however, since high-intensity endurance exercise causes oxidative stress, the direct impact of running on telomere dynamics remains unclear. This review synthesizes current literature on leukocyte telomere length (LTL) in endurance runners and evaluates emerging biotechnological interventions targeting telomere attrition. In addition, it examines cross-sectional and longitudinal studies comparing LTLs in trained endurance athletes, sedentary individuals, and age-matched controls. From analyzing different studies, evidence indicates that habitual aerobic exercise, particularly running, is associated with longer LTL and delayed biological aging, though excessive high-intensity training may accelerate telomere shortening. Other solutions to telomere shortening can be mitigated through various biotechnological approaches, including telomerase activators (e.g., TA-65), telomerase gene therapy, and senolytics. Although these approaches have been recognized for reducing senescent cells and extending telomere lengths, concerns regarding cancer risk and long-term safety persist. Overall, the findings suggest that endurance running slows biological aging by attenuating age-related telomere attrition. Further integrating exercise with emerging telomere-targeted therapies offers complementary strategies for promoting healthspan.

Keywords

Telomere Attrition, Cellular Aging, Endurance Running, Exercise, Longevity, Telomere Biotechnology

Introduction

Longevity and healthy aging are of profound importance to both society and the scientific community. As the global population ages, interventions aimed at delaying age-related decline and promoting healthspan could reduce the healthcare burden for many. One major hallmark of aging is telomere attrition, or shortening (Figure 1) (López-Otín et al., 2023). Telomeres are repetitive DNA sequences (TTAGGG in all humans) located at the ends of chromosomes, protecting genomic integrity and stability (Figure 1).

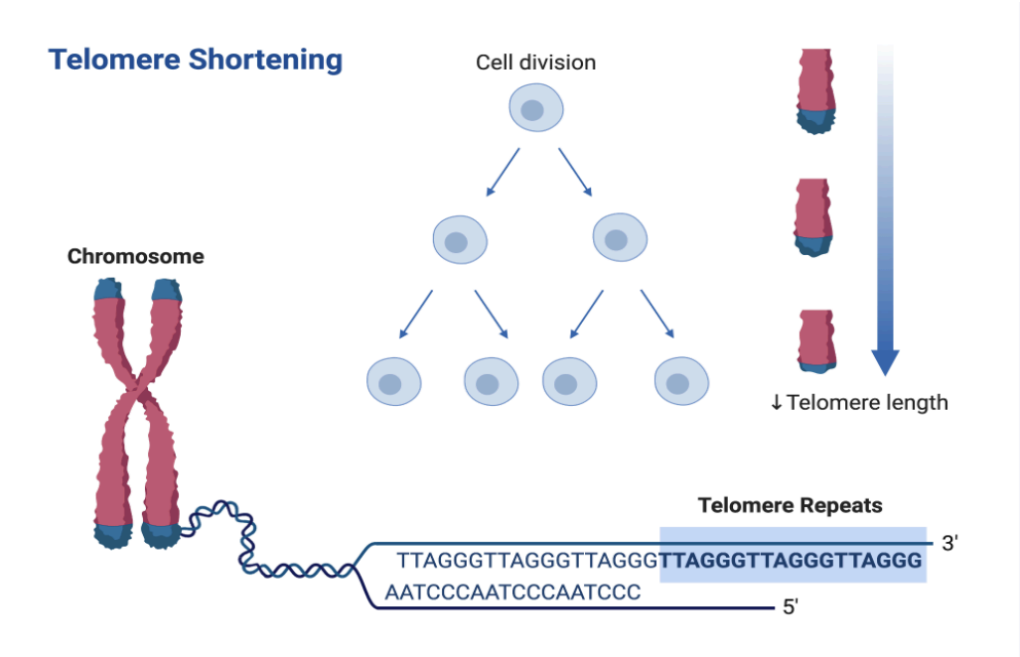

Figure 1. Telomere shortening during successive cell divisions. As the cells replicate, telomeres at the ends of chromosomes shorten, eventually leading to cellular senescence. The schematic illustrates how telomere repeats (TTAGGG) become progressively reduced with each division. Figure generated from BioRender (2025).

In healthy somatic cells, telomeres shorten with each cell division. This process eventually triggers cellular senescence, where cells have become aged and have stopped dividing, or cell death through apoptosis (Zhang et al., 2022). Other factors, like oxidative stress and cellular inflammation, can also negatively impact telomere length. Thus, telomere length serves as a key indicator of a cell's "biological age" (Vaiserman & Krasnienkov, 2021). Furthermore, shorter telomere length in white blood cells, or leukocytes, has been associated with an increased risk of cardiovascular disease, metabolic syndrome, dementia, and even death (Denham & Sellami, 2021). Conversely, lifestyle factors such as regular physical activity, a healthy diet rich in antioxidants, and not smoking correlate with delayed telomere shortening and better health outcomes (Cherkas et al., 2008; Chen et al., 2024; Paul, 2011) .

Previous reports suggest that higher cardiorespiratory fitness and regular activity levels may reduce mortality and risk of chronic disease. Specifically, moderate exercise (30 minutes of physical activity) has been linked to longer telomere lengths in humans (Denham et al., 2013). Endurance running and other high-volume aerobic sports provide a unique context for studying telomere dynamics. Chronic endurance training induces profound cardiovascular and metabolic adaptations, and competitive runners often exhibit "healthy aging" phenotypes, as characterized by lower levels of inflammation and low-density lipoprotein (LDL) (Denham et al., 2013). LDL can be associated with healthy aging as it has been shown to reduce the risk of cardiovascular death, heart attacks, strokes, etc (MD, 2021). Conversely, extreme endurance exercise, such as ultra-marathons (in which athletes run in excess of 42 kilometers), acutely generates high oxidative stress and inflammation, potentially leading to accelerated telomere attrition. Previous studies of endurance athletes have yielded conflicting results regarding telomere length, with

some finding no significant difference between trained athletes and sedentary individuals, while others report significantly longer telomeres in athletes (Denham et al., 2016).

Given these mixed findings, the significance of telomere dynamics in runners for aging is unclear. This review aims to critically examine evidence on telomere length in long-distance runners and its implications for human aging and healthspan interventions. First, we will present foundational telomere biology and background, then review studies in telomere length (TL) in runners, and explore how running may influence disease risk via telomere maintenance. Finally, we conclude with strategies to leverage exercise and related therapies to modulate telomeres and promote longevity in humans.

Telomeres: Guardians of Chromosomal Stability

Often described as a hallmark of aging or even the biological clocks of our cells, telomeres play a crucial role in determining how and when our cells age and die. Telomeres are protective nucleotide “caps” at the end of chromosomes. They comprise linear TTAGGG nucleotide repeats bound by a six-protein shelterin complex (TRF1, TRF2, POT1, TIN2, TPP1, RAP1), forming a protective, T-loop structure at each chromosome end (Figure 2) (Balan et al., 2018). This protective cap hides the DNA end from damage sensors, preventing unintended DNA damage responses (Balan et al., 2018). Furthermore, the protective cap function prevents chromosomes from being recognized as DNA damage since chromosome ends resemble DNA double-strand breaks, which would otherwise trigger the cell's DNA damage response (de Lange, 2002). In most somatic cells, telomerase (an enzyme that adds nucleotides to telomeres) is inactive, so as cell divisions continue in the body, telomeres progressively shorten with age. More specifically, during each cell division, telomeres lose a small segment (~50–150 base pairs per division) due to incomplete replication. (Denham et al., 2013). When telomeres shorten below a critical length, dysfunctional telomeres elicit a DNA damage response and thus induce replicative senescence (Balan et al., 2018).

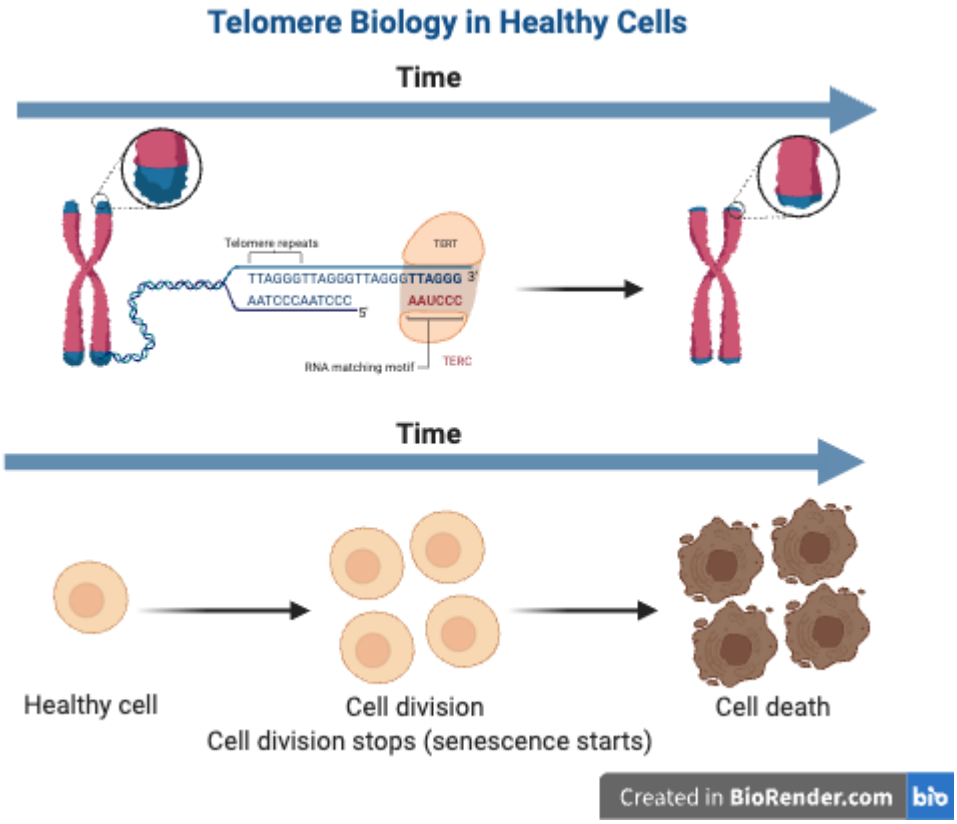

Figure 2. Telomere biology in healthy cells as time progresses, telomere shortening during cell division leads to cell senescence and ultimately cell death. Figure made using BioRender (2025)

Telomere length varies among cell types and individuals. Apart from age as a main factor to shortened telomeres, lifestyle factors such as smoking, obesity, and chronic inflammation also accelerate telomere loss (Arsenis et al., 2017). One of the most commonly studied telomere lengths in humans is leukocyte telomere length (LTL), as these can be readily measured from blood samples. Mean LTL typically declines 20-40 base pairs per year in midlife adults due to the natural aging process (Denham et al., 2013).

Since telomeres are linked to cellular aging, shortened or damaged telomeres can cause many chronic diseases. Shortened LTLs in the blood predict a higher risk of cardiovascular (CVD) and metabolic disease, cognitive decline, and neurodegeneration. Similarly, in a genetic analysis conducted by Deng et al. (2022), longer telomeres were associated with lower risk of atherosclerosis, myocardial infarction, heart disease, and stroke (though intriguingly, a slight increase in risk of high blood pressure was noted). Another result of telomere attrition is metabolic syndrome (MetS), a group of conditions that together can raise your risk of severe health problems, including diabetes, stroke, heart disease. In a meta-analysis study by Ibraheem Shelash Al-Hawary et al. (2024), it was concluded that patients with MetS may have shorter LTL. Also, in the brain, shorter LTLs have been reported in Alzheimer's disease (AD). One case-control study found AD patients had ~41–48% shorter LTL than controls (significant even after adjusting for age, sex, ApoE4) (Crocco et al., 2023). Overall, the anti-senescence effect of exercise contributes greatly to its disease protection. Together, these data suggest that

the longer telomeres seen in active individuals may underlie their lower incidence of age-related diseases (e.g., CVD, metabolic disorders, cognitive decline). Shortened telomeres also trigger cellular senescence, which contributes to tissue dysfunction and inflammation (Armanios, 2013), making this process a significant underlying factor in the pathology of many age-related diseases.

The Link Between Endurance Exercise and Telomere Maintenance

Numerous studies have compared telomere length in endurance athletes versus less-active individuals with mixed but intriguing findings. In general, endurance athletes, specifically long-distance runners, exhibit longer leukocyte telomere lengths (LTL), which is interpreted as a biomarker of delayed biological aging. Several studies support this notion. For example, in a cross sectional study evaluating training durations, researchers found that individuals engaging in regular physical activity over the course of six months had significantly longer telomeres than the sedentary controls. Their findings demonstrated a lower rate of telomere shortening by approximately 0.02 kilobases (Song et al.,2022). This suggests that physical activity enhances cellular resilience with potential longevity benefits.

Similarly, Sánchez-González et al. (2024) found that older adults who consistently engaged in moderate-to-vigorous physical activity, like high-intensity interval training (HIIT), had lower biological age markers and longer LTL compared to less active peers. Notably, HIIT training specifically stood out compared to resistance training or moderate aerobic activity, showing a LTL increase of about 0.15 kb (95% CI [0.03, 0.26]). Moderate endurance exercise thus may positively impact telomere length through mechanisms such as reduced oxidative stress and decreased systemic inflammation, as well as by increasing telomerase activity, which slows the rate of telomere shortening (Baliou et al., 2025).

Furthermore, large-scale cross-sectional data from over 4,400 U.S. adults revealed that individuals who ran or jogged at least 75 minutes per week had significantly longer telomeres—averaging around 200 base pairs longer— than those with minimal (>10 minutes) to no activity (Blackmon et al., 2023). In another study of runners aged 50-60 years old (average age 51.6 years (±5.2)), athletes' telomeres were as long as those of young adults (average age 21.8 years (±4.0)) and far longer than age-matched non-athletes (Sousa et al., 2019).

At the molecular level, exercise has been found to influence telomere maintenance pathways. Regular aerobic training has been shown to upregulate telomerase and certain shelterin proteins, preserving telomere integrity. For example, long-term endurance athletes exhibit higher expression of TERT (telomerase reverse transcriptase) and TPP1 (a shelterin component) than sedentary controls (Denham et al., 2016). This occurs because exercise transiently increases TERT expression and telomerase activity, which protects chromosome ends, leading to delayed cellular senescence (Kim et al., 2023). Taken together, these findings suggest that endurance exercise can act as a stimulus that helps boost cellular repair pathways by enhancing telomerase and DNA repair. This in turn may attenuate age-related telomere shortening.

However, it has been noted that these benefits are not universally consistent. Arsenis et al. (2017) observed that while moderate endurance training can improve telomerase activity– the enzyme responsible for maintaining telomere length– excessive training might increase oxidative stress and inflammation, thus leading to accelerated telomere attrition. This conclusion was echoed by Baliou et al. (2025), who report that prolonged high-intensity exercise without adequate muscle recovery (48-72 hours) can lead to mitochondrial dysfunction and telomere

shortening (Pocari et al., 2015). This suggests a plateau effect, that beyond a certain point, extra mileage and/or intensity does not extend/lengthen telomeres further.

Biotechnological Interventions for Telomere Maintenance

Beyond exercise, there are many emerging biotechnological approaches aiming to maintain and/or elongate telomeres. These strategies target telomerase activation or the cellular consequences of telomere attrition, often involving telomerase-activating compounds, gene therapy, and senolytic drugs. All these approaches can complement exercise strategies by directly addressing telomere biology.

Telomerase Activators

Currently, small molecules that upregulate telomerase are under active investigation, one of them being Cycloastragenol. Cycloastragenol (marketed as TA-65) is a plant-derived telomerase activator. Various studies across humans and animals show that TA-65 can elongate short telomeres without apparent pro-cancer effects. For instance, in older adults (45-75 year olds), a placebo-controlled trial of TA-65 reported a net increase of ~530 base pairs in average lymphocyte telomere length in one year compared to placebo control (Singaravelu et al., 2021). In that study, TA-65 also significantly reduced senescent (CD8+CD28–) T cell counts, suggesting improved immune aging (Singaravelu et al., 2021). While this study was effective, clinical data are still limited. A recent review notes TA-65 is the only compound so far shown to lengthen telomeres in humans. However, safety remains a concern because telomerase is reactivated in ~80% of cancers (Yu et al., 2018). In the study by Yu et al. (2018), the rate of adverse events was essentially 0, as there were no adverse effects observed for the human data. However, since telomerase can promote tumor growth, the potential for serious adverse effects still exists.

Gene Therapy

Genetic upregulation of telomerase is another powerful experimental strategy in the world of longevity. In one report, Bernardes de Jesus et al. (2012) tested the effect of telomerase gene therapy in aged mice (1-year-old and 2-year-old) using an AAV9 vector to express the telomerase reverse transcriptase (TERT) in adult mice, which delayed aging phenotypes (i.e., bone loss, glucose intolerance, neuromuscular decline). They found that gene therapy delivering the TERT gene markedly improved health and lifespan. More specifically, median life span was extended in both mice by 24% and 13%, respectively, without increasing risk of cancer (Bernardes de Jesus et al., 2012). In another study, Jaijyan et al. (2022) used a cytomegalovirus (CMV) vector to deliver telomerase reverse transcriptase (TERT) and follistatin (FST) (a protein that plays a crucial role in regulating muscle growth) to see whether it can be an effective gene delivery method. Their results showed that mouse cytomegalovirus (MCMV) carrying exogenous TERT/FST gene therapy extended median lifespan by 41.4% and 32.5% and prevented age-related telomere shortening. This was accomplished by improving glucose metabolism and physical performance, even preventing the risk of alopecia and reducing body mass (Jaijyan et al., 2022).

It is important to highlight that neither study saw a rise in tumors, indicating that gene therapy is a potential way of boosting the telomerase systemically without causing destruction to the body and instead reversing mammalian aging. Currently, human applications of telomerase gene therapy remain theoretical; however, continued experimentation translating this research

to humans could have many benefits for the future and aging. Although the trials done on mice had no detection of tumors, it is crucial to still consider the potential cancer risk. Overall, this approach could offer a genetic complement to exercise/running-induced telomere maintenance.

Senolytic Drugs

Senolytics are a type of drug that selectively clears senescent cells (Kirkland & Tchkonia, 2020). By eliminating senescent cells, senolytics can drastically reduce aging and improve telomere maintenance. For example, studies show that senolytic treatment lowers telomere-associated DNA damage foci (TAF) in multiple tissues, including the aorta, brain, bone, and more, indicating reduced telomere dysfunction (Eppard et al., 2023). Furthermore, early human trials using senolytic drugs provide support for telomere maintenance; in a pilot trial of dasatinib + quercetin (D+Q) in patients with diabetic kidney disease, a significant decrease in senescent cell markers after treatment and improved physical function were reported (Hickson et al., 2019). In a study with aged mice, chronic D+Q improves vascular functions and even extends healthspan (Roos et al., 2016). In all, senolytic drugs don't directly "lengthen" telomeres, but rather aid in removing cells burdened by shortened telomeres. Combined with exercise, these drugs may potentially help maximize telomere-based rejuvenation.

Discussion and Conclusion

Endurance running has many benefits when it comes to cellular aging, in part by attenuating telomere attrition. From the range of observational studies and meta-analyses, it is evident that habitual aerobic exercise, specifically running, slows the telomere “clock”. This is accomplished by reducing oxidative and inflammatory stress and upregulating telomerase. However, the relationship is not entirely linear–excessive or high-intensity training without proper recovery can significantly affect telomere regulation. As described recently, high-intensity training/exercise can lead to mitochondrial dysfunction, oxidative stress, and greater telomere attrition (Sánchez-González et al. 2024). This indicates a potential “plateau effect” in which the benefits of endurance exercise on telomere lengths might level off, and instead reverse. While moderate to vigorous exercise is beneficial, there is a tipping point where the physiological stress of overtraining may outweigh its protective effects on cellular aging. Thus, it is crucial to balance training and incorporate active recovery to ensure this is an effective strategy for promoting longevity and long-term telomere health. It is also important to note that when analyzing these various studies, these benefits are not universally consistent due to differences in study design, participant demographics, training protocols, measurement techniques, and more.

Emerging biotechnologies are beginning to directly target aging mechanisms, including telomere maintenance. For example, experimental telomerase gene therapy has been tested in mice and showed a significantly extended lifespan (~24% increase in adult mice, 13% in older mice), without any cancer incidence (Harrison, 2012). Additionally, biotech companies like Telomir Pharmaceuticals are developing novel telomere-lengthening drugs called Telonir-1 that reportedly “reverse” cellular aging in preclinical models (Telomir Pharmaceuticals, Inc., 2024). While many upcoming and new interventions remain largely experimental, any telomere-extension therapy must carefully balance potential longevity benefits against the risk of enabling unchecked cell growth (Haseltine, 2024). While endurance training remains a promising and accessible intervention for promoting longevity, future research should focus on

optimal training thresholds and how they can be synergized with emerging biotechnological strategies to preserve overall healthspan and mitigate age-related decline.

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