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Editorial

When the Calendar Fails: What Telomere Length Reveals About Biological Age

Chronological age has long served as the primary framework through which medicine and society understand ageing. The number of years lived determines retirement, disease screening protocols, insurance structures, and assumptions regarding physiological decline. Yet the human body frequently resists such chronological simplicity. Individuals born in the same year often display remarkably different levels of physical resilience, immune competence, metabolic health, and disease susceptibility. Some remain physiologically robust into later life, while others experience premature frailty and chronic illness decades earlier. Such differences reveal a crucial distinction between chronological age and biological age, with the latter reflecting the actual functional state of the body rather than the simple passage of time.

In recent years, telomere biology has emerged as one of the most prominent molecular indicators proposed to capture this distinction. Telomeres provide insight into how environmental conditions, lifestyle patterns, psychosocial stress, and cumulative life experiences influence cellular ageing over time. Thus, it challenges the assumption that ageing progresses uniformly across all individuals and populations (Vaiserman and Krasnienkov, 2021).

Telomeres as a Biological Clock

Telomeres are repetitive nucleotide sequences (TTAGGG repeats in humans) located at the ends of chromosomes that preserve genomic stability during cellular replication (Blackburn, 2001). The landmark discoveries on telomeres and telomerase were recognized with the 2009 Nobel Prize in Physiology or Medicine, awarded jointly to Elizabeth H. Blackburn, Carol W. Greider, and Jack W. Szostak "for the discovery of how chromosomes are protected by telomeres and the enzyme telomerase." Their pioneering work established telomeres as protective chromosomal end structures and identified telomerase as the enzyme responsible for maintaining telomere length, thereby providing crucial insights into cellular ageing, genomic stability, cancer biology, and

hereditary disorders. These discoveries laid the scientific foundation for contemporary studies exploring Relative Telomere Length (RTL) as a biomarker of biological ageing and disease susceptibility. The primary role is to prevent chromosomal degradation, end-to-end fusion, and activation of DNA damage responses. Because conventional DNA replication mechanisms cannot fully replicate chromosomal ends, telomeres progressively shorten with each round of cell division. Over time, this gradual attrition reduces the ability of cells to maintain genomic integrity and continue normal replication. The concept of finite cellular replication was first demonstrated through the Hayflick limit, which established that normal somatic cells possess restricted replicative capacity (Hayflick and Moorhead, 1961).

When telomeres erode to a critical threshold, they either enter a state of permanent growth arrest, known as senescence, or are eliminated through apoptosis. The accumulation of senescent cells contributes to impaired tissue regeneration, chronic low-grade inflammation, immune dysfunction, and physiological decline, all of which are hallmarks of ageing (Cristofalo et al., 2004; López-Otín et al., 2013). In this regard, telomere length functions as a biological clock, reflecting cumulative cellular wear rather than chronological time alone. The telomere length is now understood to be a driver, not merely a marker of the physiological decline that characterises growing old.

Importantly, telomere shortening does not occur uniformly across all tissues or individuals. Even within a single individual, telomere attrition rates vary considerably depending on cell type, replication history, oxidative stress burden, inflammatory status, and environmental exposure. Telomere length has been widely studied in relation to life expectancy, biological ageing, and ethnic variation. Telomeres shorten progressively with cellular division and are therefore considered important biomarkers of ageing. Shorter telomeres have been associated with age-related diseases, reduced longevity, and increased physiological stress.

Studies have reported significant variation in RTL among different ethnic groups, influenced by genetic background, environmental exposure, lifestyle, nutrition, and psychosocial stress. However, the relationship between telomere length and life expectancy is complex and not deterministic, as longevity is shaped by multiple biological and social factors. Nevertheless, RTL provides a useful molecular indicator for understanding population differences in ageing, health disparities, and disease susceptibility. In anthropological and population health research, telomere studies across ethnic groups are increasingly valuable for understanding health disparities, demographic transition, reproductive ageing, and evolutionary adaptation. Such investigations may also contribute to precision medicine and population-specific approaches to preventive healthcare.

Consequently, contemporary telomere research increasingly focuses on RTL rather than absolute telomere measurement. RTL estimates telomeric DNA content relative to a single-copy reference gene, allowing for a comparative assessment across populations and physiological conditions. Because of its sensitivity and applicability in large epidemiological studies, quantitative polymerase chain reaction (qPCR)-based estimation of RTL has become one of the most widely adopted methodological approaches (Joglekar et al., 2020).

Beyond serving as markers of cellular ageing, telomeres actively influence genomic function and gene regulation. Critically shortened telomeres activate DNA damage signalling pathways associated with senescence, apoptosis, inflammation, and altered cellular metabolism. Additionally, telomere length may regulate nearby sub-telomeric gene expression through chromatin reorganization, a phenomenon referred to as the telomere position effect (TPE). Telomere shortening influences cellular transcriptional activity, while genetic expression and epigenetic regulation simultaneously shape telomere maintenance. Lifestyle factors, psychosocial stress, nutrition, and environmental exposures further modulate these pathways through inflammatory and oxidative mechanisms (Andreu-Sánchez et al., 2022; Lee et al., 2021). Importantly, telomere shortening does not occur at identical rates in all individuals. Two people of the same chronological age may possess profoundly different telomere lengths depending on their cumulative exposures and physiological stress burden across the life course. Biological ageing, therefore, reflects not only time lived but also the conditions under which that life has unfolded (Vaiserman and Krasnienkov, 2021).

The Exposome Meets the Genome

Ageing has fundamental connotations of biology and social aspects. For example, chromosomal aberrations such as Down's syndrome are known to be associated with increased maternal chronological age. However, an important question arises: do all women of advanced maternal age give birth to children with Down syndrome? In this context, the assessment of RTL, a biomarker of biological ageing and cellular senescence, may provide valuable insights into individual reproductive ageing beyond chronological age alone. RTL assessment could therefore substantially enhance the process of genetic counseling by helping to identify women who may be at greater biological hazard despite similar chronological ages. The broader demographic trend of delayed motherhood is perhaps associated with higher educational attainment, increased age at marriage, and consequently, a later age at first conception. Human bodies do not age independently of their surroundings. Instead, socioeconomic conditions, environmental quality, cultural practices, and social inequalities influence physiological

resilience and disease vulnerability across populations. Chronic psychosocial stress associated with poverty, labor precarity, discrimination, social isolation, or environmental insecurity contributes to sustained inflammatory burden and accelerated telomere attrition. Studies have repeatedly demonstrated associations between chronic stress and shorter telomere length (Epel et al., 2004).

Exposome, referring to the cumulative environmental and lifestyle exposures experienced throughout life, play a crucial role in influencing biological ageing through their impact on telomere dynamics. Factors such as poor nutrition, smoking, physical inactivity, psychological stress, and environmental pollutants increase oxidative stress and chronic inflammation in the body. These processes accelerate the shortening of telomeres, which are particularly vulnerable to oxidative damage because of their guanine-rich nucleotide sequences (TTAGGG repeats in humans). Progressive telomere attrition reduces the capacity of cells to divide and maintain genomic stability. When telomeres become critically short, cells enter senescence or undergo apoptosis, leading to cellular ageing and reduced tissue regeneration. The accumulation of senescent cells contributes to physiological decline and impaired immune function. Over time, these cellular changes increase susceptibility to non-communicable diseases such as cardiovascular disorders, type 2 diabetes, and neurodegenerative conditions. Therefore, telomere shortening provides a biological influence between lifestyle factors and cellular ageing, ultimately shaping morbidity patterns, population health outcomes, and longevity (Liu et al., 2019).

Importantly, these influences are not merely behavioural but structurally patterned. Lifestyle factors themselves are shaped by broader social realities. Dietary quality depends upon economic access and food environments. Opportunities for exercise are influenced by occupation, infrastructure, and leisure availability. Psychological stress frequently emerges from financial instability, unsafe environments, or chronic social adversity. Thus, telomere dynamics reflect not only personal choices but also unequal exposure to environmental and social stressors across the life course (Blackburn and Epel, 2012).

The Population Dimension

The distinction between chronological and biological age becomes especially important at the population level. For example, chromosomal aberrations (Down's syndrome) are associated with the mother's chronological age, but across the world, non-communicable diseases are increasing in parallel with urbanisation, environmental degradation,

sedentary lifestyles, processed dietary patterns, and rising psychosocial stress. These changes are reshaping human biology long before overt disease manifests clinically.

In rapidly urbanizing societies such as India, populations frequently experience intersecting burdens of nutritional transition, occupational stress, environmental pollution, metabolic disease, and social inequality (Joshi et al., 2025). Such cumulative exposures may accelerate biological ageing even among relatively young populations. Consequently, chronological age alone increasingly fails to capture actual physiological health and vulnerability.

Biological anthropology has long demonstrated that health and disease are inseparable from ecological and social context. Telomere dynamics now provide molecular evidence supporting this perspective. Unequal access to healthcare, nutrition, clean environments, education, and stable living conditions contributes to unequal patterns of biological ageing across populations. In this sense, biological age becomes not only a physiological measure but also a reflection of broader social conditions.

At the same time, shielding cultural and social factors may buffer physiological stress. Strong kinship networks, community cohesion, traditional dietary practices, and stable social relationships can promote resilience and reduce inflammatory burden (Chakraborty et al., 2019). Human ageing, therefore, is neither exclusively genetic nor entirely behavioral. It emerges through continuous interaction between biology, environment, culture, and society.

The Clinical Provocation

The implications of biological ageing for medicine are substantial. Contemporary healthcare systems remain heavily dependent upon chronological age thresholds for disease screening, treatment decisions, and preventive interventions. Yet individuals of identical chronological age often possess markedly different biological risk profiles.

Telomere biology challenges the assumption that chronological age adequately represents physiological health. A chronologically younger individual experiencing accelerated biological ageing may carry substantially greater risk for cardiovascular disease, metabolic dysfunction, immune decline, or neurodegenerative disorders than an older individual with preserved cellular integrity. This raises important questions regarding how ageing-related disease risk should be assessed in clinical practice.

At present, telomere measurement itself is not yet sufficiently standardized for routine clinical implementation. Variability in assay techniques, tissue-specific heterogeneity, population differences, and the limitations of single time-point

measurements continue to restrict its widespread diagnostic use. Nonetheless, the conceptual importance of telomere biology remains significant. It reinforces the understanding that ageing is dynamic rather than fixed, and that biological decline may be modifiable through changes in lifestyle, environment, and psychosocial conditions.

Equally important, telomere research strengthens the case for preventive medicine. Improving nutrition, reducing smoking, increasing physical activity, addressing chronic psychological stress, and improving environmental quality may influence not only disease incidence but also the underlying pace of biological ageing itself. Meta-analytic evidence increasingly supports associations between perceived stress and telomere shortening across populations (Mathur et al., 2016).

Looking Forward

The growing distinction between chronological and biological age represents one of the most important conceptual shifts in contemporary ageing research. Telomere biology demonstrates that ageing is not simply the inevitable consequence of time, but a dynamic process shaped by lifestyle, environment, and cumulative lived experience.

It is still an early stage of integrating molecular biomarkers of ageing into mainstream clinical and public health practice. Future approaches to healthy ageing will likely integrate multiple biomarkers, including telomere length, epigenetic clocks, inflammatory markers, and metabolic indicators, to provide more comprehensive assessments of biological age. However, the broader lesson emerging from telomere research extends beyond molecular diagnostics alone.

The body does not age according to the calendar in isolation. It ages according to the environments it inhabits, the stresses it endures, the nutrition it receives, the pollutants it encounters, and the social conditions under which life is lived. Chronological age measures time. Biological age measures the physiological consequences of that time.

In this regard, telomeres represent more than molecular structures at the ends of chromosomes. They reveal how deeply human biology is shaped by lived reality, and why ageing remains one of the clearest reflections of the unequal conditions under which human lives unfold.

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