



Note on Cellular Level Aging of Human Telomere

Park Geon Hyung*

Department of Geriatrics, Seoul University, Seoul, South Korea

ABOUT THE STUDY

Aging increases the risk of disease and death at the cellular level, aging is characterized by an increase of senescent cells in the organism, caused by several factors, including oxidative stress, systemic inflammation, mitochondrial dysfunction, deregulated nutrient sensitivity, autophagy dysfunction, and telomere shortening. However, in the discussion of telomere shortening here, telomere shortening is a well-known hallmark of both cellular sensitivity and organic aging. An accelerated rate of telomere attrition is also a common feature of age-related diseases. Therefore, Telomere Length (TL) has long been recognized as one of the best biomarkers of aging.

Telomeres play a major role in cell fate and aging by adjusting the cellular response to stress and growth stimulation based on previous cell division and DNA damage. Telomere repeats of at least a few hundred nucleotides must "cap" the end of each chromosome to prevent activation of DNA repair pathways. Telomeric DNA typically ends in a single-strand G-rich overhang of between 50 and 300 nucleotides at the 3' end, which has been proposed to fold back onto duplex telomeric DNA forming a "T-loop" structure.

Progressive shortening of telomeres can lead to aging, apoptosis or oncogenic transformation of somatic cells, affecting a person's health and longevity. Short telomeres are associated with an increase in disease and poor survival. Telomeres are DNA bits at the ends of chromosomes that protect chromosomes from sticking to or entangling each other, causing DNA to malfunction. When cells replicate, the telomeres shorten at the end of the chromosomes and this process is associated with aging or cellular aging. One theory is that progressive damage of noncoding DNA caps telomeres that protect the ends of

chromosomes promotes aging and that genetic instability caused by such telomere dysfunction leads to malignancy.

Currently, peripheral blood Leukocyte Telomere Length (LTL) is frequently detected in cross-sectional and longitudinal analyzes when comparing some patient peers with age and gender-matched individuals. LTL is commonly used as a traditional biomarker of aging. LTL is a very dynamic parameter that often reflects transient changes (e.g., after triggering immune responses) and has nothing to do with the aging process per se. Whether the change in TL is the cause or effect of aging is still unclear. Furthermore, LTL is largely dependent on blood-sample leukocyte composition. In addition, it is still unclear whether LTL is a reliable surrogate marker for TL changes in other body tissues, especially in those with low proliferative activity (e.g., central nervous system), which are currently identified as major drivers of the aging process. An important point in the context under discussion is that aging is a highly complex phenomenon involving multiple pathways and cannot be measured accurately with a single biomarker, as it operates at different levels of the biological organization of the living system. Different measurements of biological age clearly measure different aspects of the aging process.

Therefore, the biological age estimates obtained with different measurement methods may not be identical to each other. Given this, it is reasonable to assume that TL (if included) enhances the hypothetical potential of mixed measurements of biological age, but its use as a single biomarker of aging may in many cases be questionable. In fact, each person included in the mixed score may indicate different aspects of the aging process, such as measurement development program (DNA methylation), replication history of the cellular lineage (TL), and environmental stress (mitochondria) etc.

Correspondence to: Dr. Park Geon Hyung, Department of Geriatrics, Seoul University, Seoul, South Korea, E-mail: jiminiaseok@gmail.com

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