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**From Cell to Cognition: Can Changes in Telomere Length Indicate  
Patterns of Cognitive Aging?**

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## From Cell to Cognition: Can Changes in Telomere Length Indicate Patterns of Cognitive Aging?

Review by Zhu et al. [1] is one of several excellent recent review papers on telomeres and aging that, taken together, point to the importance of bridging the gap between research on cellular and cognitive aging. Researchers who study trends in aging across societies have shown that some factors can affect longevity. For instance, Fraser & Shavlik [2] have shown that optimal choices regarding diet, physical exercise, cigarette smoking, body weight, and hormone replacement therapy in women, had significant influence on longevity in Californian Adventists. They followed up 34,192 California Adventists from 1976 to 1988 and found higher life expectancy in this group compared to other white Californians by 7.28 years in men and 4.42 years in women. Other large cohort studies conducted in different countries have also reported that life expectancy can be extended even up to 18 years when associated, for instance, with physical exercise and refraining from smoking [3]. These clearly substantial gains in life expectancy are probably worthwhile only if the extended life span also matches the brain span. This is becoming a prominent issue as the population worldwide rapidly ages.

The issue of how to study the impact of biological age on nonpathological cognitive aging is tightly related to the question of when cognitive decline begins. Theories on cognitive decline typically assume that deterioration occurs at older age. However, not all researchers agree with this assumption, presenting evidence that indicates that declines in speed processing, reasoning, memory, executive function, language and so on actually begin in our 20s and 30s (e.g., [4]). While cognitive decline is part of normal aging, individual differences in how we age may reveal how genetics, general health, diet, lifestyle, and other factors affect cognitive aging.

When it comes to the role of biological age in cognitive aging, one particularly interesting question is whether telomere shortening is a marker for cognitive aging. Telomeres and their associated proteins have in particular been linked with cancer, cardiovascular disease, and neurodegenerative diseases, but apart from a broad conclusion that telomere shortening is a marker of cellular aging and its association with the risk of Alzheimer's disease (AD), chronic stress exposure, and depression, little is known about how exactly changes in telomere length affect cognition and emotion in the course of aging. Studies on this topic that have been conducted so far typically report that telomere shortening is a distal correlate of cognitive decline [5]. However, some researchers have reported different findings, indicating that telomere length is not a reliable predictor of decline or mortality (e.g., [6]). These controversial findings on the impact of telomere length on cognitive aging clearly indicate a need for studying this issue in more detail.

For instance, one could investigate whether different patterns of cognitive aging are associated with different patterns of telomere shortening. Ideally, examining the relationship between telomere length and cognition should be based on measuring telomeres in neural tissue. Although telomere length in leukocytes may act as a surrogate marker for telomere length in other tissues, it may not be applicable to all cell types. However, Takubo et al. [6] measured telomere lengths of 5 organs (cerebral cortex, myocardium, liver, renal cortex and spleen) in a large number of autopsy cases and found "a robust correlation of telomere length among these organs in any given individual". Their data also suggest a specific telomere length in each person. Given these findings, it seems appropriate to investigate the relationship between the peripheral blood leukocyte telomere length and cognitive functions.

If the hypothesis on the impact of telomere shortening on cognitive performance holds, we can expect to find different patterns of nonpathological cognitive decline associated with differences in telomere shortening. Finding out exactly what types of telomere length-cognitive decline patterns exist across different age groups would represent an important step in bridging the gap between the research on cellular and cognitive aging.

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