



Nutrition Influences on Telomere Length and Longevity

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Abstract

Telomeres are the dynamic chromosome-end structures that protect the genome. They are one of the most essential factors that influence aging and longevity. Telomere length (TL) has been linked to stress, DNA damage, and the onset of aging-related illnesses in research. Nutritional food intake, physical activity, obesity, and stress, among other genetic and environmental factors, have been shown to influence telomere dynamics and health. The goal of this review was to summarize and discuss the most important nutrition components and nutritional food intake that have been linked to TL and longevity. In addition, the review serves as a foundation for future study.

Keywords: Telomeres, Telomere length (TL), Nutrition, Longevity

Introduction

Telomeres, which are nucleoprotein structures at the ends of eukaryotic chromosomes, protect the chromosome's end against degradation and end-to-end fusion (1). The telomeres are gradually attrited with each somatic cell division, resulting in telomere length shortening with increasing age (1). Telomere length (TL) has been postulated as a potential biomarker of aging (1), with greater TL being associated with healthy aging. The preservation of TL in healthy people compared to people with multiple morbidities is regarded to be one of the mechanisms that can explain the development of chronic illnesses and mortality (1).

Although aging is the most important factor in TL shortening, nutrition can also play a significant role. Healthy eating habits may help to slow TL shortening as people get older (2). Single dietary components and food groupings associated with good eating patterns like a Mediterranean style diet and the Dietary Guidelines for Americans (DGAs) have been linked to

longer TL in various cross-sectional studies (2). Individuals who consumed more dietary fiber, nuts, seeds, fruits, and vegetables while consuming less saturated fat, sugar sweetened beverages, and processed meats had longer TL (2).

The purpose of this review was to synthesize and explain the most essential nutritional components and food intake that have been connected to TL and longevity. Furthermore, the review gives inspiration for further research.

What are telomeres?

Telomeres are nucleoprotein structures that function to maintain genome stability. They are found at the end of each chromosome arm. Telomeres are made up of repeat DNA sequences which are connected by several proteins known as telomeric interacting proteins. In mammalian cells, telomere DNA has double-stranded tandem repeats of TTAGGG followed by single-stranded hanging at the terminal 3' G (3,4). The T-loop

structure of telomere DNA is formed when the telomere end folds back on itself and the 3' G strand overhang occupies the D-loop structure of double-stranded DNA (3).

Telomere attrition is a natural process that is widely acknowledged as one of the characteristics of aging (5). A wide number of population-based studies have found that as people get older, their leukocyte telomere length (LTL) decreases (6). Researchers discovered a link between telomere shortening and a variety of clinical symptoms. Telomere shortening is influenced by lifestyle factors including oxidative stress and inflammation, as well as metabolic abnormalities such as abdominal obesity, hyperglycemia, and hypertension (7, 8). As a result, whatever molecular pathway produces increased oxidative stress, such as during cancer treatment, could be linked to telomere maintenance dysregulation (9).

TL can be used as a biomarker for a cell's biological (rather than chronological) age, as well as the possibility of additional cell division (9). Telomerase is a catalytic enzyme that promotes telomere lengthening, which helps to solve the end replication problem (9). Telomere shortening has also been associated with prenatal conditions and early difficulties, including potentially adjustable lifestyle factors like bad nutrition and physical inactivity (10, 11). In fact, evidence from several researches reveal that a decent sleep pattern, a balanced diet, and low stress were all linked to longer telomeres (9).

TL and Aging process

The aging mechanisms of mitochondria and telomeres have been linked, and a direct molecular link between telomere attrition and mitochondrial dysfunction has been discovered (9). P53 activation driven by telomere shortening has been demonstrated to downregulate the expression of Peroxisome proliferator-activated receptor-gamma coactivator (PGC1) and so reduce mitochondrial biosynthesis and function, in addition to terminating the cell cycle. Dysfunctional mitochondria show metabolic abnormalities such as reduced ATP production, higher reactive oxygen species (ROS) production, and lower levels of ROS-detoxifying enzymes, all of which cause further damage to mitochondrial and genomic DNA, including telomeres (12).

Aging is influenced by both endogenous and exogenous factors as summarised in Figure 1. Aging is a malleable process that may be impacted by a variety of endogenous and external variables in both positive and negative ways. Telomeres and mitochondria are crucial components in cellular aging control, and thus the aging process at the organismal level, according to expanding research. According to a recent study, critically short telomeres activate the DNA damage response, which results in an increase in the p53 protein (13).

Increased levels of p53 inhibit PGC1 and the main promoters of mitochondrial biogenesis, resulting in mitochondrial malfunction, in addition to activating cell cycle arrest mechanisms through p21 activation (13). Dysfunctional mitochondria emit more ROS into the cytoplasm, producing more cell damage and hastening senescence (13). External causes such as irradiation, pollution, and environmental stress, as well as bad habits such as smoking, alcohol consumption, unhealthy eating, lack of exercise, obesity, and other variables, can all lead to increased ROS generation and other aging-related deleterious effects (13). Cellular defense systems such as ROS scavenging enzymes and antioxidants such as vitamins, flavonoids, and carotenoids, on the other hand, can limit ROS production, protect telomeres, prevent mitochondrial damage, and extend our lives. Positive lifestyle behaviors such as regular moderate exercise, relaxation, and a nutritious diet can also help us age better (13).

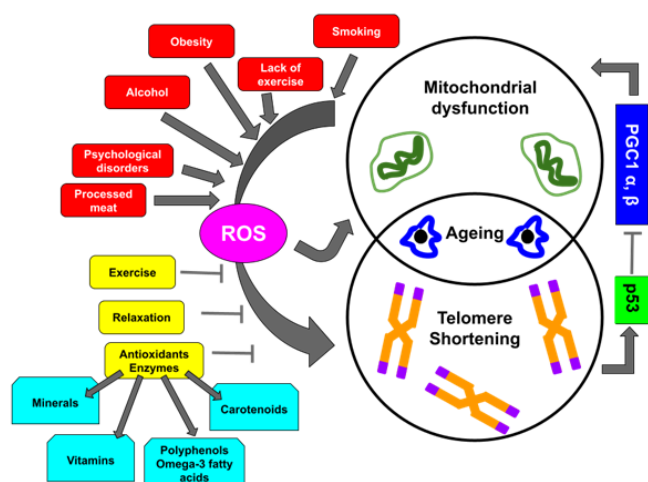


Figure 1 Both endogenous and external variables influence aging. Modified from Vidacek NŠ, et al. Can We Really Navigate Our Aging? *J Gerontol A Biol Sci Med Sci.* 2017 Dec 12;73(1):39-47 (13).

Influences of Macronutrients on TL

The two cross-sectional studies found a positive relationship between dietary fiber consumption and TL (14,15), implying that carbohydrate quality, particularly dietary fiber, may have a potential beneficial effect on telomere health while also lowering the risk of chronic diseases (16, 17). When it comes to telomeres, however, dietary fats have received more attention than the other macronutrients. Fat is a vital dietary component that has been shown to contribute to inflammation. To better study this, longer RCTs with large samples of people will be required in the future.

Omega-3 fatty acids have been identified as critical chemicals for well-being and, in particular, for the cardiovascular system, over the previous decade. There is a negative correlation between the blood level of marine omega-3 fatty acids (docosahexaenoic and eicosapentaenoic acids) and the rate of telomere attrition in individuals with coronary heart disease during a 5-year period (18). In a recent randomized controlled four-month research, it was discovered that the ratio between omega-3 (n-3) and omega-6 (n-6) fatty acids is more essential than the omega-3 itself, since telomere length increases with decreasing n-6:n-3 plasma ratios when compared to baseline telomere length (13).

These individuals' telomerase activity remained unaltered throughout the trial, but there was a substantial negative correlation between telomere length and biomarkers of oxidative stress and inflammation, two factors that influence telomere shortening (19). As a result, people with higher endogenous n-6:n-3 polyunsaturated fatty acid ratios would benefit more from a simple nutritional intervention like omega-3 supplementation (13).

Influences of Micronutrients on TL

Micronutrient consumption has also been connected to TL. In a number of ways, micronutrients such as vitamins, minerals, and other bioactive compounds have been shown to protect against oxidative stress

and DNA damage (20-22). Because of their relevance in purine and pyrimidine synthesis, folate and vitamin B-12 have prompted a lot of research (9). Vitamin D, a fat-soluble vitamin, has been linked to lower all-cause mortality, type 2 diabetes, and inflammation in multiple studies including hemodialysis patients. (23, 24).

Vitamins C and E, in particular, have been shown to play important biological roles in the context of TLs (14, 25, 26). Finally, because of the wide range of study, establishing beneficial effects of minerals and vitamins on telomere health is extremely difficult (in terms of design, type of participants investigated, and length) (13). As a result, in order to completely appreciate the influence of these nutrients and their beneficial dosages on telomere biology, future research will need to be larger and longer.

TL and food groups

Several epidemiological research and clinical trials have looked into the link between telomere health and dietary types, based on the concept that antioxidative and anti-inflammatory activities may influence the aging process. Polyphenols, unsaturated fatty acids (in the case of nuts), and fiber are abundant in vegetables, fruits, legumes, and nuts. Consuming them has been linked to improved inflammation and oxidative stress markers, as well as insulin resistance and other cardiovascular risk factors, in addition to longer telomeres (27, 28). Unhealthy foods include processed meats, alcoholic beverages, sweetened carbonated beverages, and other foods high in saturated fatty acids, alcohol, and sucralose.

Conclusion

It has been shown that eating a diet rich in specific micronutrients can help to prevent telomere shortening. The mechanisms of cell senescence and aging have been largely described by researchers. One of the key drivers of cellular and organismal aging in higher mammals is telomere shortening. Aging is a disease that affects everyone, until we find a cure, we can slow down the aging process by making TL significantly longer. Importantly, nutritional food intake was found on rigorous scientific information regarding elements that are both good and harmful to our health and aging.

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