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Editorial

Telomeres Role in Locking the Aging Process

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EDITORIAL NOTE

DNA is present in the nuclei of our cells, where it is made up of structures called chromosomes. Each chromosome contains specific genetic information in the form of genes. When the cells in our body divide, chromosomes need to repeat so that each cell has a complete set of chromosomes in its nucleus. At the ends of each of our chromosomes is a stretch of DNA called telomeres. Telomeres help protect the ends of chromosomes from being damaged or merging with nearby chromosomes.

Some have suggested that telomere shortness may be a major contributor to the aging process and disease development. But no one can fully understand the impact of telomere shortening on our overall health. The lymphocytes exposed to polycyclic aromatic hydrocarbons have significantly fewer telomeres, and evidence of increased DNA damage and genetic instability compared with control factors shows signs of age-related macular degeneration DNA damage and decreased telomere function with age [1]. It can be noted that the relationship between the increased mortality rate from diseases [2]. But this study was about 20 years old and had only 143 participants. Recent Meta-analyzes also suggests a link between short telomeres and coronary heart disease or certain types of cancer. Research is ongoing on the link between Telomere Shortening and Death.

Although it is known that chromosome replication reduces telomeres, some experts believe that oxidative stress may also reduce them. Oxidative stress causes damage to DNA and other molecules from reactive oxygen species. Reactive oxygen species are created by both the natural cellular processes and inflammation in body. You can also get them a reliable source from environment through things like pollution, smoking or alcohol. Over time, damage to DNA and other molecules caused by oxidative stress can contribute to health problems associated with aging. Again, this is a new area of research, so there is not much conclusive evidence. This study was conducted on stress associated with the release of glucocorticoid hormones by the adrenal gland. These hormones reduce the levels of antioxidant proteins and can therefore cause oxidative damage to DNA and rapid telomere shortening [3-5].

Short telomeres increase the risk of cancer, although no one knows for sure. Specific cancers associated with small telomeres: bladder, lungs, kidneys, gastrointestinal tract, neck and head. In addition, the hallmark of cancer cells compared to other cells is that they grow and divide rapidly. Cancer is usually an age-related genetic disorder that occurs only when normal cells accumulate genetic instability over a period of time and acquire the ability to replicate immortality. Telomere attrition triggers chromosome instability during successive cell divisions and contributes significantly to genetic reorganization leading to tumorigenesis. Telomeres at the ends of chromosomes, repetitive (TTAGGG) DNA-protein complexes, are crucial for the survival of cancer cells. In most tumors they are maintained by an enzyme called telomerase. The mechanisms underlying Telomere Length (TL) management and telomerase expression include transcriptive, post-transcriptional, and epigenetic regulation, and in-depth understanding of these mechanisms can provide novel biomarkers for disease detection, diagnosis, and development therapeutics. Telomerase, an enzyme that reduces telomere shortening in specific cells, is reactivated or increased in more than 90 percent of cancers. Remember, this enzyme is not found in most cell types. But cancer cells can use telomerase to protect their telomeres, delaying their degeneration. Based on this information, some new cancer treatments target telomerase to help destroy cancer cells faster.

Studies have shown that common hTERT promoter mutations are found in most cancers at all stages and grades, indicating that hTERT mutations are usually the starting point in the process of carcinogenesis [6]. In order to establish the role of these mutations as early events in the malignant transformation process, it would be interesting to determine whether these mutations are more likely to occur during periods of cell crisis. Cancer cells also achieve proliferative immunity by activating or regulating the silent Human TERT Gene (hTERT) that encodes telomerase, which is a reverse transcriptase activated by complexes to make ribonucleoprotein. Rarely, another DNA recombination mechanism called telomere Alternating Length (ALT) reverses telomere attrition to bypass sensibility. Although hTERT is generally silenced in almost all somatic cells, it is

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significantly expressed in 90% of human cancers. The underlying mechanisms of hTERT activation are still being explained, but they mainly include mutations in the hTERT promoter, changes in the alternative splicing of the hTERT pre-mRNA, hTERT amplification, epigenetic changes and/or Telomere Position Effect (TPE) machinery [6]. Recent reports have shown that telomerase activation in cancer cells involves two cancer-specific hTERT promoter mutations (mainly C>T mutations). These mutations in the -124 Base Pairs (BP) or -146 bp upstream from the TERT translation startup site were found to be associated with increased telomerase activity. Therefore, the molecular mechanisms that regulate hTERT expression and telomerase assembly are subject to serious research [7-11]. Our food may play a part in determining the length of our telomeres, according to a study involving over 5,000 adults, which indicated that consuming more fiber was associated with longer telomere length. Fiber's capacity to assist manages blood glucose levels could be the reason for this. Higher blood glucose levels are linked to inflammation and oxidative stress, according to the researchers. Both of these factors may contribute to telomere shortening [12].

CONCLUSION

Telomeres aid in the protection of chromosomes. Telomeres shorten as a result of this process, which is linked to ageing and disease development. However, new study suggests that a combination of diet, stress management, and exercise may be able to thwart this process. While these findings are still preliminary, we already know that an active lifestyle, combined with a healthy diet and stress management strategies, can bring a variety of additional health benefits.

REFERENCES

1. Pavanello S, Pesatori AC, Dioni L, Hoxha M, Bollati V, Siwinska E, et al. Shorter telomere length in peripheral blood lymphocytes of workers exposed to polycyclic aromatic hydrocarbons. Carcinogenesis. 2010;31(2):216-221.
2. Cawthon RM, Smith KR, O'Brien E, Sivatchenko A, Kerber RA. Association between telomere length in blood and mortality in people aged 60 years or older. Lancet. 2003;361(9355):393-395.
3. Patel R, McIntosh L, McLaughlin J, Brooke S, Nimon V, Sapolsky R. Disruptive effects of glucocorticoids on glutathione peroxidase biochemistry in hippocampal cultures. J Neurochem. 2002;82(1): 118-125.
4. Von Zglinicki T. Oxidative stress shortens telomeres. Trends Biochemical Sci. 2002;27(7):339-344.
5. Song Z, Figura G, Liu Y, Kraus JM, Torrice C, Dillon P, et al. Lifestyle impacts on the aging-associated expression of biomarkers of DNA damage and telomere dysfunction in human blood. Aging Cell. 2010;9:607-615.
6. Chiba K, Johnson JZ, Vogan JM, Wagner T, Boyle JM, Hockemeyer D. Cancer-associated TERT promoter mutations abrogate telomerase silencing. Elife. 2015;4:e07918.
7. Horn S, Figl A, Rachakonda PS, Fischer C, Sucker A, Gast A, et al. TERT promoter mutations in familial and sporadic melanoma. Science. 2013;339(6122):959-961.
8. Huang FW, Hodis E, Xu MJ, Kryukov GV, Chin L, Garraway LA. Highly recurrent TERT promoter mutations in human melanoma. Science. 2013;339(6122):957-959.
9. Vinagre J, Almeida A, Pópulo H, Batista R, Lyra J, Pinto V, et al. Frequency of TERT promoter mutations in human cancers. Nature Commun. 2013;4(1):1-6.
10. Killela PJ, Reitman ZJ, Jiao Y, Bettegowda C, Agrawal N, Diaz LA, et al. TERT promoter mutations occur frequently in gliomas and a subset of tumors derived from cells with low rates of self-renewal. Proc Natl Acad Sci U S A. 2013;110(15):6021-6026.
11. Borah S, Xi L, Zaug AJ, Powell NM, Dancik GM, Cohen SB, et al. TERT promoter mutations and telomerase reactivation in urothelial cancer. Science. 2015;347(6225):1006-1010.
12. Tucker LA. Dietary fiber and telomere length in 5674 US adults: an NHANES study of biological aging. Nutrients. 2018;10(4):400.