Review Subcell Biochem. 2018;90:221-308. doi: 10.1007/978-981-13-2835-0_9.

Telomeres, Telomerase and Ageing

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PMID: 30779012 DOI: 10.1007/978-981-13-2835-0_9

Abstract

Telomeres are specialised structures at the end of linear chromosomes. They consist of tandem repeats of the hexanucleotide sequence TTAGGG, as well as a protein complex called shelterin. Together, they form a protective loop structure against chromosome fusion and degradation. Shortening or damage to telomeres and opening of the loop induce an uncapped state that triggers a DNA damage response resulting in senescence or apoptosis.Average telomere length, usually measured in human blood lymphocytes, was thought to be a biomarker for ageing, survival and mortality. However, it becomes obvious that regulation of telomere length is very complex and involves multiple processes. For example, the "end replication problem" during DNA replication as well as oxidative stress are responsible for the shortening of telomeres. In contrast, telomerase activity can potentially counteract telomere shortening when it is able to access and interact with telomeres. However, while highly active during development and in cancer cells, the enzyme is down-regulated in most human somatic cells with a few exceptions such as human lymphocytes. In addition, telomeres can be transcribed, and the transcription products called TERRA are involved in telomere length regulation.Thus, telomere length and their integrity are regulated at many different levels, and we only start to understand this process under conditions of increased oxidative stress, inflammation and during diseases as well as the ageing process.This chapter aims to describe our current state of knowledge on telomeres and telomerase and their regulation in order to better understand their role for the ageing process.

Keywords: Ageing; DNA damage; Senescence; Telomerase; Telomere.

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