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Telomere length and cardiovascular aging.

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Abstract

Telomeres are located at the end of chromosomes. They are composed of repetitive TTAGGG tandem repeats and associated proteins of crucial importance for telomere function. Telomeric DNA is shortened by each cell division until a critical length is achieved and the cell enters senescence and eventually apoptosis. Telomeres are therefore considered a 'biological clock' of the cell. Telomerase adds nucleotides to telomeric DNA thereby contributing to telomere maintenance, genomic stability, functions, and proliferative capacity of the cell. In certain rare forms of progeria, point mutations within the telomere lead to accelerated telomere attrition and premature aging. Endogenous factors causing telomere shortening are aging, inflammation, and oxidative stress. Leukocyte telomere length (LTL) shortening is inhibited by estrogen and endogenous antioxidants. Accelerated telomere attrition is associated with cardiovascular risk factors such as age, gender, obesity, smoking, sedentary life-style, excess alcohol intake, and even mental stress. Cardiovascular (CV) diseases and CV aging are usually but not invariably associated with shorter telomeres than in healthy subjects. LTL appears to be a biomarker of CV aging, reflecting the cumulative burden of endogenous and exogenous factors negatively affecting LTL. Whether accelerated telomere shortening is cause or consequence of CV aging and disease is not clear.