Teleomere shortening & metabolic/vascular diseases.

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BACKGROUND: Telomeres are specialized DNA-protein structures located at the ends of eukaryotic chromosomes whose length is progressively reduced in most somatic cells during ageing. Over the past decade, emerging evidence has shown that the telomeres are essential regulators of cellular life span and chromosome integrity in a dynamic fashion. By inducing genomic instability, replicative senescence and apoptosis, shortening of telomeres is thought to contribute to organismal ageing.

DISCUSSION: While the aetiology of cardiovascular diseases and diabetes represent a complex interaction between various risk factors overlaid on different genetic backgrounds, the conventional risk factors often did not explain the inter-individual variability related to predisposition of disease states. This underscores the need for biological indicators of ageing in evaluating the aetiology of several age-related disorders, and recent studies indicate that telomere length could qualify as an ideal marker of biological ageing. Short telomeres have been detected in senescent endothelial cells and vascular smooth muscle cells from human atherosclerotic plaque as well as in myocardial tissue from patients with end-stage heart failure and cardiac hypertrophy. In addition, telomere shortening has been demonstrated in WBCs from patients with coronary heart disease, premature myocardial infarction, hypertension and diabetes mellitus.

SUMMARY: In this review, we discuss the telomere hypothesis of ageing as well as human studies that address the role of telomeres in cardiovascular, diabetes and other cardio-metabolic pathologies.

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