

Abstract

Arterioscler Thromb Vasc Biol. 2003 May 1;23(5):842-6.

White cell telomere length and risk of premature myocardial infarction.

Brouillette S, Singh RK, Thompson JR, Goodall AH, Samani NJ.

Division of Cardiology, Department of Medicine, University of Leicester, United Kingdom.

OBJECTIVE: Biological age may be distinct from chronological age and contribute to the pathogenesis of age-related diseases. Mean telomeres lengths provide an assessment of biological age with shorter telomeres, indicating increased biological age. We investigated whether subjects with premature myocardial infarction (MI) had shorter leukocyte telomeres.

METHODS AND RESULTS: Mean terminal restriction fragment (TRF) length, a measure of average telomere size, was compared in leukocyte DNA of 203 cases with a premature MI (<50 years) and 180 controls. Age- and sex-adjusted mean TRF length of cases was significantly shorter than that of controls (difference 299.7±69.3 base pairs, $P<0.0001$) and on average equivalent to controls 11.3 years older. The difference in mean TRF length between cases and controls was not accounted for by other coronary risk factors. Compared with subjects in the highest quartile for telomere length, the risk of myocardial infarction was increased between 2.8- and 3.2-fold ($P<0.0001$) in subjects with shorter than average telomeres.

CONCLUSIONS: The findings support the concept that biological age may play a role in the etiology of coronary heart disease and have potentially important implications for our understanding of its genetic etiology, pathogenesis, and variable age of onset.

PMID: 12649083

FREE FULL TEXT

