

Abstract

Lancet. 2007 Jan 13;369(9556):107-14.

Telomere length, risk of coronary heart disease, and statin treatment in the West of Scotland Primary Prevention Study: a nested case-control study.

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BACKGROUND: Inter-individual differences in biological ageing could affect susceptibility to coronary heart disease. Our aim was to determine whether mean leucocyte telomere length is a predictor of the development of coronary heart disease.

METHODS: We compared telomere lengths at recruitment in 484 individuals in the West of Scotland Primary Prevention Study (WOSCOPS) who went on to develop coronary heart disease events with those from 1058 matched controls who remained event free. We also investigated whether there was any association between telomere length and observed clinical benefit of statin treatment in WOSCOPS.

FINDINGS: Mean telomere length decreased with age by 9% per decade (95% CI 3.6-14.1; $p=0.001$) in controls; much the same trend was seen in cases (-5.9% per decade, -3.1 to 14.1; $p=0.1902$). Individuals in the middle and the lowest tertiles of telomere length were more at risk of developing a coronary heart disease event than were individuals in the highest tertile (odds ratio [OR] for coronary heart disease: 1.51, 95% CI 1.15-1.98; $p=0.0029$ in the middle tertile; 1.44, 1.10-1.90, $p=0.0090$ in the lowest). In placebo-treated patients, the risk of coronary heart disease was almost double in those in the lower two tertiles of telomere length compared with those in the highest tertile (1.93, 1.33-2.80, $p=0.0005$ in the middle tertile; 1.94, 1.33-2.84, $p=0.0006$ in the lowest). By contrast, in patients treated with pravastatin, the increased risk with shorter telomeres was substantially attenuated (1.12, 0.75-1.69, $p=0.5755$ in the middle tertile; 1.02, 0.68-1.52, $p=0.9380$ in the lowest).

INTERPRETATION: Mean leucocyte telomere length is a predictor of future coronary heart disease events in middle-aged, high-risk men and could identify individuals who would benefit most from statin treatment. Our findings lend support to the hypothesis that differences in biological ageing might contribute to the risk--and variability in age of onset--of coronary heart disease.

PMID: 17223473