Telomere length in circulating leukocytes is associated with lung function and disease.

Several clinical studies suggest the involvement of premature ageing processes in chronic obstructive pulmonary disease (COPD). Using an epidemiological approach, we studied whether accelerated ageing indicated by telomere length, a marker of biological age, is associated with COPD and asthma, and whether intrinsic age-related processes contribute to the interindividual variability of lung function. Our meta-analysis of 14 studies included 934 COPD cases with 15 846 controls defined according to the Global Lungs Initiative (GLI) criteria (or 1189 COPD...
cases according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria, 2834 asthma cases with 28 195 controls, and spirometric parameters (forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC) and FEV1/FVC) of 12 595 individuals. Associations with telomere length were tested by linear regression, adjusting for age, sex and smoking status. We observed negative associations between telomere length and asthma ($\beta = -0.0452$, $p=0.024$) as well as COPD ($\beta = -0.0982$, $p=0.001$), with associations being stronger and more significant when using GLI criteria than those of GOLD. In both diseases, effects were stronger in females than males. The investigation of spirometric indices showed positive associations between telomere length and FEV1 ($p=1.07 \times 10^{-7}$), FVC ($p=2.07 \times 10^{-5}$), and FEV1/FVC ($p=5.27 \times 10^{-3}$). The effect was somewhat weaker in apparently healthy subjects than in COPD or asthma patients. Our results provide indirect evidence for the hypothesis that cellular senescence may contribute to the pathogenesis of COPD and asthma, and that lung function may reflect biological ageing primarily due to intrinsic processes, which are likely to be aggravated in lung diseases.

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