

Abstract

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Father's smoking initiation in puberty as associated with accelerated ageing in offspring

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Background: Paternal smoking in puberty is associated with low lung function and asthma in offspring. We hypothesize it may also influence biological aging.

Aims: To examine associations between paternal preconception smoking and offspring's biological age and age acceleration.

Methods: We analyzed epigenetic clocks (GrimAge, PhenoAge and DunedinPACE) (Margiotti, K. et al. Cytog Gen Res 2023; 163:247-256) from blood DNA methylation in 892 RHINESSA study participants (offspring; mean age 28 years). Personal smoking history was reported by offspring, parental smoking by the parents (RHINE/ECRHS study participants). We used linear regressions adjusted for sex and grandparental education to estimate associations between paternal smoking around puberty (never=ref., ≤15 years and >15 years) and biological age acceleration.

Results: In the paternal line (n=415), offspring whose fathers started smoking ≤15 years (n=67) and >15 years (n=184) had older GrimAge and PhenoAge than those whose fathers never smoked (n=164). GrimAge (β -coeff. 0.7; 95%CI 0.03-1.3), PhenoAge (1.0; -0.4-2.3) and DunedinPACE (0.02; -

0.002-0.5) were more accelerated for offspring of fathers who started ≤ 15 than when fathers started >15 years (0.4 [-0.1-0.8]; 0.9 [-0.2-1.9]; 0.004 [-0.01-0.02], respectively). When excluding ever-smoking offspring, the differences by father's age of smoking initiation were more pronounced. The maternal line (n=477) showed no consistent effects of preconception smoking.

Conclusions: Our findings indicate paternal smoking in puberty may contribute to increased biological age and faster age acceleration in offspring. Whether this explains the link with offspring's respiratory health needs future studies.

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