|  |
| --- |
| **Paternal pre-pubertal passive smoke exposure is associated with impaired lifetime lung function in offspring: a cohort study across two generations** |
| Jiacheng Liu1, Jennifer L. Perret1,2, Caroline J. Lodge1, Don Vicendese1,3, N. Sabrina Idrose1, Bircan Erbas4, Peter Frith5, Michael J. Abramson6, E. Haydn Walters1,7, Shyamali C. Dharmage1,8, Dinh S. Bui1 |
| *1Allergy and Lung Health Unit, Melbourne School of Population and Global Health, The University of Melbourne, VIC, Australia.**2Institute for Breathing and Sleep, VIC, Australia.**3School of Engineering and Mathematical Sciences, La Trobe University, VIC, Australia.**4School of Psychology & Public Health, La Trobe University, VIC, Australia**5College of Medicine and Public Health, Flinders University, SA, Australia.**6School of Public Health & Preventive Medicine, Monash University, VIC, Australia.**7School of Medicine, University of Tasmania, TAS, Australia.**8Murdoch Children’s Research Institute, VIC, Australia.* |
| **Introduction/Aim:** Paternal passive smoke exposure before completing puberty may increase the risk of childhood asthma in future offspring, possibly due to epigenetic changes in the germ line. However, this potential impact of parents on offspring’s lung function is unknown. We investigated associations between pre-pubertal passive smoke exposure in parents and lifetime lung function trajectories in their offspring.**Methods:** Data were analysed from 816 father-offspring and 1,149 mother-offspring pairs from the Tasmanian Longitudinal Health Study (TAHS). The offspring in the analysis were probands who underwent spirometry at six-time points from ages 7 to 53 years, with lung function (FEV1, FVC and FEV1/FVC) life-course trajectories derived using group-based trajectory modelling. Parents of these offspring reported their own passive smoke exposure before age 15 years. Multinomial logistic regression models were used to investigate associations between paternal and maternal pre-pubertal passive smoke exposure with offspring’s lifetime lung function trajectories, separately. Potential mediation and interactions were assessed by parental active smoking, offspring sex, their childhood respiratory conditions and active smoking history over the life-course to middle age.**Results:** Paternal pre-pubertal passive smoke exposure was associated with offspring lifetime Below Average FEV1 trajectory (adjusted multinomial odds ratio [aMOR]=1.62 [95%CI: 1.09-2.41]) and Early Low-Rapid Decline FEV1/FVC trajectory (aMOR=2.30 [95%CI: 1.07-4.93]). The association with the Below Average FEV1 trajectory was stronger for those with paternal exposure and offspring’s own childhood passive smoke exposure (i.e., parental active smoking during offspring childhood) (aMOR=2.70 [95%CI: 1.52-4.80]; p-interaction=0.038). Of the observed associations, the indirect effect related to familial smoking history and offspring’s childhood respiratory conditions was limited (<15%). No adverse associations were observed for maternal pre-pubertal passive exposure.**Conclusion:**Pre-pubertal passive smoke exposure in fathers may critically impair their future offspring’s lifetime lung function trajectories via epigenetic changes to sperm precursor cells. This underscores the urgency to restrict pre-pubertal smoke exposure to safeguard possible intergenerational impacts.**Grant Support:** National Health and Medical Research Council; JL is supported by the China Scholarship Council - University of Melbourne PhD Scholarship and “Population Health Investing in Research Students’ Training”. |