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Title: Bronchiolar wall structure is altered in adult mice following neonatal hyperoxia

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Body: Background: Very preterm infants often require supplemental oxygen, and can have an increased risk of poor lung function and asthma in later life. Follow-up studies suggest that factors associated with very preterm birth can cause long-term changes in the the small conducting airways. Aim: To determine if bronchiolar wall structure is persistently affected by neonatal exposure to hyperoxic gas. Methods: Neonatal mice (C57BL/6J) breathed 65% O_2 from birth until postnatal day 7 (P7), after which they lived in room air until P56 (n=26). Controls breathed room air from birth (n=27). Bronchiolar walls, lung parenchyma and bronchoalveolar fluid (BALF) were analysed at P56. In bronchioles, we measured epithelial thickness, proportions of proliferating epithelial cells, ciliated and Clara cells, the amount of collagen and airway smooth muscle (ASM), and the number of alveolar-bronchiolar attachments. In lung parenchyma, we measured percent tissue space and mean linear intercept (MLI). Results: In bronchioles, adult mice exposed to neonatal hyperoxia had significantly thicker epithelium, more ASM and more collagen than controls ($p<0.05$). Compared to controls there were no significant differences in bronchiolar epithelial cell proliferation, Clara cells or ciliated cells. In lung parenchyma, MLI was increased and tissue fraction reduced (both $p<0.05$) in hyperoxia-exposed mice, but there was no effect on the number of alveolar-bronchiolar attachments. In BALF there were 60% more immune cells ($p<0.05$) after hyperoxia. Conclusions: Exposing the developing lung to hyperoxic gas results in persistent airway remodelling and increased numbers of pulmonary immune cells suggesting on-going inflammation in adulthood.