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| **A novel COPD mice model investigating the progressive effect of low-dose PM2.5 exposure on the lung health** |
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| **Introduction/Aim:**  Air pollution is a global issue not limited to areas with severe air pollution. PM (particulate matter) 2.5 lower than the current annual limit potentially remains harmful.  **Methods:**  Five-week-old male Balb/c mice (n=60) were exposed once daily to either low-level PM2.5 (10ug) or saline as the control for 4, 8 and 12 weeks.  **Results:**  Interestingly, low-dose PM2.5 exposure significantly induced airway hyperresponsiveness (AHR) regarding increased Rn (%) both in the short and long term (P<0.001 and P<0.05). As expected, short or moderate-term exposure to low-dose PM2.5 had no significant impact on ventilation function based on indexes (P>0.05 for IC, FEV0.2, FVC, PEF, Rrs, Crs, Ers, Rn, tissue damping or elastance). However, long-term exposure to low-dose PM2.5 increased Rn (P<0.05), indicating it induced airway resistance, mainly conducting airways. Differential cell counts from BALF showed low-dose PM2.5 significantly increased neutrophils in absolute and relative numbers after moderate and long-term exposure (P<0.001). Moreover, macrophages and eosinophils were only elevated by long-term PM2.5 exposure (P<0.05). Histological staining revealed inflammatory cell infiltration, increased collagen deposition around the large airway and thickness of smooth muscle layer rather than epithelium after long-term low PM2.5 exposure.  **Conclusion:**  Low-dose PM2.5 exposure induced airway remodelling, impaired lung function and compliance in the long term. AHR and increased neutrophil-dominated inflammation occurred in the early stage of progression that could be therapeutically targeted.      **Grant Support:**  N/A |