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Acute air pollution exposure alters neutrophils in never-smokers and at-risk humans

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ABSTRACT Outdoor air pollution exposure increases chronic obstructive pulmonary disease (COPD) hospitalisations, and may contribute to COPD development. The mechanisms of harm, and the extent to which at-risk populations are more susceptible are not fully understood. Neutrophils are recruited to the lung following diesel exhaust exposure, a model of traffic-related air pollution (TRAP), but their functional role in this response is unknown. The purpose of this controlled human-exposure crossover study was to assess the effects of acute diesel exhaust exposure on neutrophil function in never-smokers and at-risk populations, with support from additional *in vitro* studies.

18 participants, including never-smokers (n=7), ex-smokers (n=4) and mild–moderate COPD patients (n=7), were exposed to diesel exhaust and filtered air for 2 h on separate occasions, and neutrophil function in blood (0 h and 24 h post-exposure) and bronchoalveolar lavage (24 h post-exposure) was assessed.

Compared to filtered air, diesel exhaust exposure reduced the proportion of circulating band cells at 0 h, which was exaggerated in COPD patients. Diesel exhaust exposure increased the amount of neutrophil extracellular traps (NETs) in the lung across participants. COPD patients had increased peripheral neutrophil activation following diesel exhaust exposure. *In vitro*, suspended diesel exhaust particles increased the amount of NETs measured in isolated neutrophils. We propose NET formation as a possible mechanism through which TRAP exposure affects airway pathophysiology. In addition, COPD patients may be more prone to an activated inflammatory state following exposure.

This is the first controlled human TRAP exposure study directly comparing at-risk phenotypes (COPD and ex-smokers) with lower-risk (never-smokers) participants, elucidating the human susceptibility spectrum.