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Residential air pollution increases the risk for persistent pulmonary hypertension after pulmonary endarterectomy

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This retrospective cohort study showed that both persistent postoperative pulmonary hypertension and systemic inflammation were associated with previous exposure to air pollutants in CTEPH patients https://bit.ly/39tUy2l

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To the Editor:

Chronic thromboembolic pulmonary hypertension (CTEPH) is a life-threatening condition and a rare complication of pulmonary embolism [1]. It is characterised by increased pulmonary vascular resistance (PVR), due to unresolved fibro-thrombotic material obstructing large pulmonary arteries, and secondary microvasculopathy. Various factors are suspected to be involved in the pathogenesis and progression of CTEPH, including inflammatory thrombosis, dysregulated fibrinolysis and deficient angiogenesis [2]. The gold standard therapy is a surgical approach, namely pulmonary endarterectomy (PEA), consisting of removal of obstructing fibro-thrombotic material. Although long-term exposure to air pollution might influence several factors potentially contributing to the pathogenesis of CTEPH, including pulmonary and systemic inflammation, pro-thrombotic changes, oxidative stress, endothelial dysfunction and vasoconstriction [3, 4], no study so far has investigated the relationship between air pollution and CTEPH. Interestingly, epidemiological and translational data supports the plausibility of a link between air pollution exposure, right ventricle failure and pulmonary hypertension (PH) [5–7]. Moreover, exposure to traffic-related air pollution was recently demonstrated to be associated with disease severity and impaired survival in pulmonary arterial hypertension (PAH), characterised by progressive occlusion of precapillary arteries [8]. Eventually, a potential role of air pollution as a risk factor for developing venous thromboembolism has been suggested [9]. We consequently hypothesised that exposure to common air pollutants, including particles with an aerodynamic diameter less than 2.5 or 10 µm ($PM_{2.5}$, PM_{10}),

nitrogen dioxide (NO_2), black carbon (BC) and traffic-related air pollution, could influence the progression of CTEPH and interfere with disease outcomes.