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Basic Science

Abstract Title

Acute wood-smoke exposure enhances methacholine-induced bronchoconstriction in precision-cut lung slices

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Background

Wood-smoke (WS) exposure during wildfires is associated with respiratory morbidity and impaired lung function. Although the WS exposure can worsen respiratory disease, little is known regarding the impact of short-term exposure (1-hour) on healthy airway function and symptoms of breathlessness. This study investigated whether physiologically relevant acute WS exposure alters airway responsiveness to methacholine in precision-cut lung slices (PCLS).

Objective

Acute WS exposure enhances methacholine-induced airway constriction.

Methods

PCLS were prepared from 8-week-old female BALB/c mice. Lungs were inflated with 2% low-melting agarose, excised, and sectioned at 230 μm using a vibratome. After 48h in culture, PCLS were exposed to clean air or 5 μg WS/slice (estimated airborne concentration of $\sim 150 \mu\text{g}/\text{m}^3$), generated by pyrolysis of 1g standardized softwood pellets at 450°C and delivered using the expoCube system. Airway responses were assessed using the physioLens platform during sequential exposure to methacholine (10^{-8} – 10^{-4} M) by measuring airway lumen area and the percentage of bronchoconstriction. Tissue viability was evaluated using calcein and SYTOX staining.

Results

Baseline airway lumen area did not differ significantly between clean-air- and WS-exposed PCLS 1h after exposure, indicating that acute exposure to WS alone did not induce airway constriction. Representative calcein/SYTOX staining showed increased SYTOX-positive signal following WS exposure, suggesting induction of cellular injury or death. Methacholine induced concentration-dependent bronchoconstriction in both groups; however, WS-exposed airways exhibited significantly greater reductions in lumen area. At maximal stimulation, net bronchoconstriction reached approximately 50% in the WS group compared with 40% in clean-air control. Despite this enhanced contractile response, methacholine pEC₅₀ did not differ significantly between groups, indicating that WS exposure increased the magnitude of airway contraction without altering methacholine sensitivity.

Conclusion

Acute exposure to 5 µg WS enhances the magnitude of methacholine-induced bronchoconstriction without altering agonist sensitivity. This suggests short-term exposure may increase feelings of breathlessness during wildfires.