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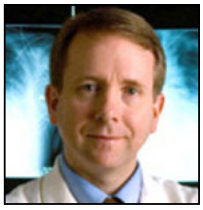
Airway mast cells mediate ozone-induced bronchial hyperresponsiveness



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Abstract

Ozone is a highly reactive environmental pollutant with well-recognized adverse effects on lung health. Bronchial hyperactivity is one consequence of ozone exposure, particularly problematic for individuals with underlying lung disease. Here we show that ozone induces substantial ATP release from human airway epithelia in vitro and into the airways of mice in vivo, and that ATP is a potent inducer of BHR. Both mast cell deficient and P2X7 deficient mice have markedly attenuated BHR to ozone and to ATP, suggesting that ATP induces BHR indirectly through activation of P2X7 purinergic receptors on mast cells. ATP-induced mast cell activation is eliminated in mast cells from P2X7 deficient mice and in human mast cells treated with a selective P2X7 receptor antagonist. Collectively, these data suggest that mast cell activation by ATP is central to the pathogenesis of ozone induced BHR. Strategies targeting mast cells, mast cell products, or P2X7 receptors on high ozone days may be therapeutic for vulnerable patients with underlying lung disease.

Biography

Stephen Tilley completed his MD from the University of North Carolina (UNC) in 1988, and post-doctoral training at Vanderbilt University and UNC. He has been a faculty member at UNC for the past 21 years. Presently he is Associate Professor in the Department of Medicine and Director of the UNC Lung Disease Models center. He is a reviewer for multiple pulmonary, allergy, and immunology journals and serves on grant study sections for the American Lung Association and NIH.

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