Title of Dataset

Inconsequential Role for Chemerin-Like Receptor 1 in the Manifestation of Ozone-Induced Lung Pathophysiology in Mice_Dataset

Introduction

Chemerin, a non-chemokine chemoattractant, and resolvin E1 (RvE1), a specialized pro-resolving lipid mediator, are endogenous ligands for chemerin-like receptor 1 (CMKLR1), a $G_{i/o}$ protein-coupled receptor expressed by leukocytes and non-leukocytes. In mice, exogenous administration of chemerin or RvE1 diminishes the severity of lung inflammation and airway hyperresponsiveness (AHR) induced by antigen sensitization and challenge, which mimics phenotypic features of atopic asthma in humans. However, the contribution of chemerin or RvE1 to features of non-atopic asthma is unknown. Therefore, to indirectly assess if chemerin or RvE1 facilitates development of a non-atopic asthma phenotype, which includes AHR to acetyl-β-methylcholine chloride, lung hyperpermeability, airway epithelial cell desquamation, and lung inflammation, we quantified features of these sequelae in wild-type mice and mice failing to express CMKLR1 (CMKLR1-deficient mice) following cessation of acute inhalation exposure to either filtered room air or ozone, a criteria pollutant and non-atopic asthma stimulus.

Methods Collection

- Through this laboratory-based study, we investigated whether lung injury, lung inflammation, and AHR, which are three phenotypic features of asthma, differed between wild-type mice (C57BL/6NCrl) and CMKLR1-deficient mice following acute inhalation exposure to ozone.
- Wild-type and CMKLR1-deficient mice, which were at least twelve weeks of age, were exposed for three hours to either filtered room air or ozone (2 parts/million). Either four- or twenty-four-hours following cessation of exposure, mice were euthanized so that blood could be collected, a bronchoalveolar lavage (BAL) performed, and lungs harvested. A second cohort of mice was anesthetized twenty-four-hours following cessation of exposure. In these mice, pressure-volume curves were generated and airway responsiveness to acetyl-β-methylcholine chloride was assessed. A third cohort of mice was euthanized twenty-four hours following cessation of exposure to filtered room air to measure the lung wet weight, dry weight, and wet-to-dry weight ratio.
- Endpoint measurements include:
    - Body mass
    - Total blood leukocytes
    - Expression of lung tissue chemerin chemokine-like receptor 1 (*Cmklr1*) mRNA
    - BAL adiponectin, albumin, chemerin, chitinase-3-like protein 1, ciliated epithelial cells, hyaluronan, interleukin (IL)-6, IL-11, keratinocyte chemoattractant (KC), macrophage inflammatory protein (MIP)-2, MIP-3α, macrophages, neutrophils, osteopontin, protein, and receptor for advanced glycation end product (RAGE)
    - Serum chemerin
    - Airway responsiveness to methacholine indices: R_{aw}, airway resistance; G, coefficient of lung tissue damping; H, coefficient of lung tissue elastance
    - E_{stat}, quasi-static respiratory system elastance
    - Lung wet weight, dry weight, and wet-to-dry weight ratio

Citations – Publication Based on the Dataset

Johnston RA, Pilkington IV AW, Atkins CL, Boots TE, Brown PL, Jackson WT, Spencer CY, Siddiqui SR, and Haque IU. Inconsequential role for chemerin-like receptor 1 in the manifestation of ozone-induced lung pathophysiology in mice. *J Appl Physiol*. [In clearance].

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