Abstract

Asthma prevalence is increasing yearly with a paucity of new therapeutics to target airway inflammation. Corticosteroids are the current mainstay to address airway inflammation; however, a subset of asthmatics are refractory to steroids. As such, new targets to alleviate airway inflammation are critical in the management of this disease. Airway inflammation following viral infection, a main asthma trigger, is regulated by immune cells including airway macrophages. Macrophages also promote resolution of airway inflammation by producing pro-resolving molecules including eicosanoids. We hypothesize that viral infection stimulates the production of pro-inflammatory and pro-resolving eicosanoids via the purinergic, P2X7, receptor signaling pathway in alveolar macrophages and that an imbalance favoring pro-inflammatory eicosanoid production contributes to airway inflammation and remodeling in individuals with asthma. Further characterization of signaling pathways involved in airway inflammation may result in new therapeutic targets for the treatment of asthma, especially in patients who are unresponsive to corticosteroids.