

EDITORIAL

ASTHMA AND MAST CELL BIOLOGY

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Asthma is a chronic inflammatory disease of the lung and its pathophysiology is initiated by mast cell activation in response to the antigen binding to IgE receptor as well as by TH2 cell activation. Mast cells are well established effector cells in asthma where they exacerbate the inflammatory response, playing a key role in early phase, degranulating and increasing histamine. Human mast cells possess high affinity IgE receptors and are ubiquitous but predominantly localized in mucosal and connective tissue and are distributed along blood vessels. There are two types of mast cells: connective tissue mast cells (TC) and mucosal mast cells (T mast cells). TC mast cells contain more heparin, whereas T mast cells contain more chondroitin sulfate. In asthma, mast cell activation can trigger degranulation, releasing secretory granule complex and preformed mediators, such as histamine and proteases, along with the synthesis of leukotrienes and prostaglandins, and induction of cytokines and chemokines. Leukotriene inhibitors and omalizumab, which inhibits IgE, both relieve the asthma exacerbation when administered to humans and permit to reduce the use of other drugs. The release of cytokines by mast cells, such as TNF-alpha, IL-1, IL-6 and IL-33, participate in the pathogenesis of asthma. Stress worsens asthma, and this effect is also mediated by mast cell activation through the release of cytokines. Administration of IL-33 in experimental animals provokes pathological effects in the mucosal tissues and augments antibody IgE and IgA in blood vessels. Here, we report the impact of mast cell biology in asthma pathogenesis.