The participation of heme oxygenase-1 in allergic inflammation

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The development of allergic inflammation is influenced by both genetic and environmental factors. Mast cells are effector cells in IgE-mediated allergic inflammation such as asthma and allergic rhinitis. Mast cells express FceRI on their cell surface. Aggregation of FceRI by multivalent antigen initiates a cascade of biochemical events that lead to not only the early phase response by degranulation and secretion of inflammatory mediators but also the late phase responses by the production of cytokines and chemokines, which induce the infiltration of several cells to the site of inflammation. Furthermore, it is suggested that mast cells could modulate T cells and B cells in the chronic allergic inflammation.

HO-1 is the rate-limiting enzyme in the conversion of heme to biliverdin, carbon monoxide (CO) and free iron, and induced a variety of stimuli. Recent reports using a variety of immunocytes, including mast cells, basophils, macrophages and T cells, have suggested that HO-1 and its products have an ability to regulate the cell activation and function. These findings suggest that HO-1 can modulate the allergic responses. In mast cells, although HO-1 suppresses degranulation, whether HO-1 suppresses cytokine synthesis in mast cells is unknown. In this symposium, I would like to show and discuss the role of HO-1 on production of mediators in the activated mast cells, which are effector cells in IgE-mediated allergic inflammation.