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Editorial

IL-25 and IL-33 as Effective Target-Therapy of Allergic Asthma

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Editorial

Allergic asthma is one of the main problem in health of worldwide which characterized by breathlessness, whizzing, coughing and dyspnea. Type 2 cytokines have important role in pathophysiology of allergic asthma and can be trigger for infiltration of inflammatory cells, mucus hyper secretion, Immunoglobulin E production and airway hyper responsiveness [1,2]. Asthma causes 250,000 deaths annually and billions of dollars in healthcare were spend for control and cure of asthma [3].

Innate immune response cells (especially eosinophils) are critical for the development of asthma. Innate immune response cells activation can be induced by two power cytokines, IL-25 and IL-33.IL-25 is structurally a member of the IL-17 cytokine family. Th2 cell differentiation is promoted by IL-25 in an IL-4 and STAT6-dependent manner. Overexpression of IL-25 induces type 2 cytokines responses, eosinophilia associated inflammation in the lung and mucus production [4-6]. IL-33 as a member of the IL-1 family of cytokines is produced by epithelial cells, which is inducible by IL-1α, IL-1β. ST2 is the receptor for IL-33. IL-33-receptor interaction recruits MyD88 (adapter molecule), leading to MAP kinase activation and NF-κB translocation and induction of Th2 cytokines responses. IL-33 also activates eosinophils as main orchestrating cells in allergic airway inflammation [7].

Using monoclonal antibody as antagonist of IL-25 and IL-33 bioactivity or using with gene silencers to regulate of IL-25and IL-33 gene expression, can be control type 2 inflammations and airway hyper responsiveness in allergic asthma. Co-blockade of IL-25 and IL-33 receptors can be effective for control of asthma and remodeling of respiratory system. A combination treatment asthmatic case with both soluble IL-33receptors and sIL-25R can reduce mediators of inflammatory cells.

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