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Editorial

Chemo-cytokines network is main target for control of Allergic asthma

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Asthma is a chronic respiratory disease which characterized by recurrent airflow obstruction, wheezing, chest tightness and coughing. Management of allergic asthma especially in children, is main problem for industrial world. Immunological factors have critical role in pathogenesis of allergic asthma. Cytokines as major controller of immune system, are important in this reaction. Allergic asthma is a disease with symptoms: eosinophilic inflammation, mucus hyper secretion, airway obstruction, airways hyperresponsivness, IgE high level production, smooth muscle spasm. Cytokines have main and complicated role in pathophysiology of allergic asthma.

Also T helper (Th) 2 cytokines is associated with asthmatic attack, but Th1 cytokines have power connections with Th2 cytokines in a big network to modulate immune responses and inflammatory process. These connections are necessary to create balance between pro-inflammatory and immunomodulatory bioactivities in the allergic reaction process. Many outcomes of allergic asthma are under control of cytokines balance (in all over of body and target tissue) [1-3]. IL-5 as Th2 cytokine of immune system, promotes maturation, activation and survival of eosinophils and develops pulmonary eosinophilia and airway obstruction. Chemokines such as eotaxin can attract eosinophils in to respiratory system and leads to eosinophils trafficking. IL-13 Overexpression induces mucus overproduction, and airway hyperresponsiveness. IL-13 can induces goblet cells hyperplasia. Using antagonist of IL-5 and eotaxin can reduce eosinophils activation and eosinophilia in airway and using antagonist of IL-13 can reduce mucus hyper secretion and airway obstruction. IL-4 induces Immunoglobulin E (IgE) production from activated plasma cells. Produces IgE can bind mast cells receptors (sensitization) and secondary arrived allergen binds to IgE on the mast cells receptors that leads to allergic mediator release from mast cells. IL-9, IL-17, IL-25, IL-33 have strong role in phatogenesis of allergic asthma [2,3].

Chemokines as chemotactic cytokines, have the distinctive duty to elicit the leukocytes to sites of inflammation [4]. Eotaxins as chemokine (CC family) are potent attractants of eosinophils and contribute to allergic asthma. Three type eotaxins have been identified: CCL11 (eotaxin-1), CCL24 (eotaxin-2) and CCL26 (eotaxin-3). They bind to CC chemokine receptor 3 (CCR3), which is expressed by eosinophils. Eotaxin-1 has chemotaxis effect on eosinophils and mobilizes of eosinophils progenitors from the bone marrow and causes cells degranulation. Biologic roles of eotaxin-2 and eotaxin-3 in the allergic asthmatic reaction is known. Therefore, eotaxin-1 is important in allergic asthma but eotaxin-2 and 3 may have role in eosinophils attractant [4,5].

The identification and characterization of cytokine's network connections and interaction of chemokines with this network are an important therapeutic approaches for allergic asthma. Manipulation of one of each part of this network may be potential key to control of asthma, but pleiotropism and redundancy in this network is very

complicated. The individual contribution of each cytokines and chemokine can have main regulator effect in destiny of disease. Therefore, all approaches should be defined carefully, checked connections in network with together and then manipulated.

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