Special Lecture

Innate lymphoid cells in allergic diseases

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There have been extensive developments on cellular and molecular mechanisms of immune regulation in allergy, asthma, autoimmune diseases, tumor development, organ transplantation, and chronic infections during the last few years. Better understanding the functions, reciprocal regulation and counter balance of subsets of immune and inflammatory cells that interact via interleukins (ILs), interferons (IFNs), tumor necrosis factor (TNF)- and transforming growth factor (TGF)- offer opportunities for immune interventions and novel treatment modalities in the era of development of biological immune response modifiers particularly targeting these molecules or their receptors. Populations of lymphoid cells that lack rearranged antigen receptors and markers for myeloid and lymphoid lineages, such as T, B, NK cells show similarities to Th1, Th2 and Th17/22 type of immune responses have been identified. These cells are defined as innate lymphoid cells (ILC) type 1, 2 and 3 cells. ILC1 mainly produce IFN-gamma, ILC2 produce IL-5 and IL-13, [36] and ILC3 produce IL-17 and IL-22. ILCs control the mucosal environment through close interaction with epithelial cells and other tissue cells, production of cytokines and induction of chemokines that recruit suitable cell populations to initiate and promote distinct types of immune response development and tissue inflammation. These cells can be detected in several body fluids and tissues such as, sputum, peripheral blood, nasal polyps, oesophagus for their characterization in allergy and asthma patients. It has been recently shown that these cells particularly the Type 2 innate lymphoid cells play a major role in the development of asthma dn allergic rhinitis. They receive signals from the epithelium such as IL-33 and produce huge amounts of IL-5 and IL-13 and contribute to eosinophilia, Th2 cell accumulation, IgE regulation as well as cause barrier leakiness.