# Invariant Natural Killer T (iNKT) Cells in Asthma: A Novel Insight into the Pathogenesis of Asthma and the Therapeutic Implication of Glycolipid Ligands for Allergic Diseases

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Allergic bronchial asthma is a complex inflammatory diseases originated from dysregulated immune responses in the respiratory mucosa. The inflammatory state in asthmatic lung is characterized by massive infiltration with eosinophils, lymphocytes, and mast cells in the airway mucosa leading to airway hyperseisitivity, goblet cell hyperplasia and mucus overproduction. The inflammatory process is thought to be the result of intensive T helper (Th) 2-biased immune response. Over the past several years, there has been enormous progress in understanding the mechanisms for development of Th2-biased responses after inhaled exposure to allergens and the characteristics of CD4+ T cells prominently involved in this process. Recently, a new population of T cells, invariant natural killer T (iNKT) cells has been shown to play an important role in the pathogenesis of mouse model of allergic airway inflammation. iNKT cells are one of the most potent immune modulators through a massive production of a various cytokines including IL-4 and IFN-γ upon activation, and are involved in a variety of immunoregulations including infection, autoimmunity, and tumor surveillance. The potent pathogenic role of iNKT cells in the development of bronchial asthma is due to their ability to produce predominant Th2 cytokines in a given condition. The involvement of iNKT cells in the pathogenesis of asthma might have been underestimated in the past studies demonstrating the involvement of CD4+ T cells in asthma because of the difficulty in the detection of iNKT cells. Meanwhile, growing evidences have demonstrated that iNKT cells could be a promising target for immune-based therapies for autoimmune diseases, tumor, and infection due to the invariance of their TCR usage, the restriction to the evolutionally-conserved non-polymorphic antigen-presenting molecule CD1d, and their outstanding ability to produce both Th1- and Th2-cytokines. In this review, we will overview current understanding of the pathophysiological roles of iNKT cells in asthma. We would also discuss on possible therapeutic approaches to bronchial asthma employing glycolipid ligands for iNKT cells.

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