Update of cytokines and genes in asthma and allergic rhinitis

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In this last issue of 2014, there are several papers related to the prevalence and pathogenesis mechanisms of asthma and allergic diseases. The first paper by Wanlapakorn et al.,[1] reports a cross-sectional descriptive study investigating asthma in Thai students aged between 6-12 years from 3 public schools and 3 private schools in Bangkok., Only 9% of the 1,428 students enrolled in this study were diagnosed as asthmatic, with only 10% of the cases reported not being under control. This result indicates that the prevalence of asthma in the student population from elementary schools in the Bangkok area is characterized by a relatively low incidence, which might be resulting from better asthma management strategies in the royal capital.

Two other papers investigate the role of IL-33 and IL-20 in pathogenesis of asthma. Although IL-33 is known to play a key role in the onset of asthma, Wu and colleagues,[2] demonstrated that it is also implicated in airway smooth muscle cells (ASMCs) proliferation in ovalbumin-sensitized mice and inflammation associated to bronchial asthma. IL-20, belonging to the interleukin family IL-10, has been found to be highly expressed in the airway epithelium of asthma patients where other cytokines such as Th2 cytokines, IL-4, IL-5 and IL-133 were also highly expressed. The IL-20 was also found to be significantly correlated with asthma severity, suggesting that it might be a good indicator of asthma risk. Biman Saikia and colleagues[4] discovered a novel mutation in the DNA binding domain of STAT3 in patients with Hyper IgE syndrome (HIES), a primary immunodeficiency disorder. This mutation induces the reduction of Th17 cell production and consequently increases patients susceptibility to Mycobacterial infection. Another interesting finding was reported by Shen and colleagues.[5] They found that patients with nasal polyposis (NP) have defective regulatory T cells and Foxp3 levels and present significantly decreased TGF-β levels in allergen stimulated PBMCs in vitro. The authors hypothesized that these finding may explain why NP patients have chronic inflammation that could not be controlled by regulatory T cells.

Finally, the last paper of the december 2014 issue investigate determinants of another important allergic disease: allergic rhinitis (AR). Yu Zhang et al.,[6] showed that the expression of H2-Eb1 (orthologous gene of human HLA-DRB1 in mice) gene accompanied by an increased GATA-3/T-bet ratio play an important role in AR model in mice, corroborating recent studies in the field.

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