INTRODUCTION AND AIM OF THE WORK

Asthma is a complex inflammatory disease of the lung characterized by variable airflow obstruction, airway hyperresponsiveness and airway inflammation (***Yssel and Groux., 2000).*** Numerous studies have demonstrated that CD4+T lymphocytes, via the release of specific cytokines, orchestrate the events that initiate and maintain the inflammatory response observed in asthma ***(Naseer et al., 1997).***

Allergic asthma is associated with up-regulation of Th2 cytokines, notably IL-4, IL-5 and IL-10 which have been implicated in the pathogenesis of allergic inflammation by their ability to promote IgE synthesis ***(Hamelmann and Gelfand.,2001).*** A recently described human cytokine, interleukin-13 (IL-13) which shares a receptor component and signaling pathways with IL-4, was found to be necessary and sufficient for the expression of allergic asthma ***(Wills-Karp-M., 2001).***

Kroegel et al (1996) demonstrated that IL-13 is actively secreted during the late asthmatic response in mild to moderate atopic asthmatics.

IL-13 is a product of activated CD4+ Th2 lymphocytes ***(Zhu et al., 1999),*** which shares a number of biologic properties with IL-4 ***(de Vries, 1998)***. As such it has been shown that IL-13 will promote IgE synthesis by B cells and downregulates IL-12 and IFN-γ production thereby favoring a Th2-type response ***(Van Der Pouw Kraan et al.,1998).*** IL-13 may also contribute to the recruitment of eosinophils into the airways by enhancing the expression of vascular cell adhesion molecule-1 (VCAM-

1) ***(Bochner et al., 1995)***. On the basis of these properties, IL-13 may have a considerable role to play in the pathogenesis of allergic inflammation such as bronchial asthma ***(Naseer et al., 1997)***.

Recently it has been described in steroid sensitive asthmatics that corticosteroid therapy modulates cytokine expression to favor a Th1-type cytokine profile, which may be one of its beneficial actions in the treatment of allergic diseases ***(Robinson et al., 1993b).*** Further in chronic asthmatics it is found that after steroid therapy the expression of IL-13mRNA is decreased compared with base line value ***(Naseer et al., 1997)***.