

# IL-32, a proinflammatory cytokine in aspirin sensitive asthma

Contribution to YSC Davos 2008

Thematic topic: Medicine

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*Key words:* IL-32; T-cells, epithelial cells, asthma

## INTRODUCTION/BACKGROUND

IL-32 is a proinflammatory cytokine, which is assumed to play a role in autoimmune diseases. It is produced by natural killer cells, T lymphocytes and epithelial cells. In epithelial cell lines, it can be secreted upon stimulation of some proinflammatory cytokines such as IL-1-beta, IFN-gamma and IL-12. IL-32 induces the production of TNF- alpha, IL-6 and IL-8 in monocytic cell lines. Furthermore, IL-32 up regulates the expression of cyclooxygenase-2 and influences the production of prostaglandin E2 in peripheral blood mononuclear cells. A special regulatory role for prostaglandin E2 has been postulated in aspirin-induced asthma. The aim of the study is to examine the role of IL-32 in asthmatic patients with and without aspirin sensitivity.

## MATERIAL & METHODS

The mRNA level of IL-32 in freshly isolated CD4+ positive T lymphocytes from healthy donors and asthmatic patients is analyzed by realtime PCR. The expression of IL-32 in T-cells is determined by FACS analysis and in primary bronchial epithelial cells by immunofluorescence stainings. Urinary levels of prostaglandin E2 and leukotrien E4 are measured in urine samples by ELISA.

## RESULTS

The relative expression of IL-32 in CD4+ T lymphocytes is higher in asthmatic patients with aspirin sensitivity than in healthy controls or asthmatic patients without aspirin sensitivity. Systemic treatment with glucocorticoids decreases the expression of IL-32 in T-cells in asthmatic subjects after 4 hours significantly. IL-32 mRNA is also expressed in primary bronchial and sinus epithelial cells and the expression of IL-32 can be stimulated by TNF-alpha, TGF-beta and IFN-gamma.

## CONCLUSIONS & OUTLOOK

The current study suggests that IL-32 is involved in the inflammatory process of the airways in asthma with higher expression in T-cells in aspirin sensitive subjects and can be suppressed by systemic glucocorticoid treatment.