IL-32 expression upregulated in chronic rhinosinusitis
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Interleukin-32 mRNA expression is significantly higher in biopsies obtained from patients with chronic rhinosinusitis, compared to levels found in biopsies obtained from individuals without the condition, according to a study published online April 9 in Allergy.

Michael B. Soyka, M.D., of the Swiss Institute of Allergy and Asthma Research in Davos, and colleagues isolated human primary sinonasal epithelial cells (HSECs) from biopsies of patients with CRS and from healthy individuals and stimulated them with different cytokines. IL-32 mRNA expression was investigated and protein levels were determined.

The researchers found that tumor necrosis factor-? (TNF-?) and interferon-? (IFN-?) upregulated IL-32 mRNA expression in primary epithelial cells, but other interleukins tested did not influence IL-32 mRNA expression. In HSECs co-cultured with T-helper 1 cells (Th1), IL-32 was significantly higher than in cells co-cultured with Th0 or Th2 cells. Compared with healthy subjects, IL-32 mRNA expression was significantly higher in biopsies obtained from CRS patients with nasal polyps (CRSwNP). Compared with control tissues, in patients with CRS, IL-32 was detected in biopsies.

"In conclusion, our study demonstrates that IL-32 might be involved in the pathogenesis of CRS," the authors write. "The induction of IL-32 in HSECs and sinonasal biopsies by Th1 cells, TNF-?, and IFN-? and the upregulation of IL-32 in CRSwNP indicate a potentially important role of this cytokine."

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