

# Researchers identify source of molecule linked to nasal polyps, asthma attacks

May 23 2018

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A new discovery about how the immune system responds to common sinus infections and asthma could explain why patients develop these issues in the first place and ultimately may lead to improved targeted

therapies. Researchers from the Perelman School of Medicine at the University of Pennsylvania have identified the source of the inflammatory cytokine Interleukin-25 (IL-25), an immune molecule that recruits a subset of inflammatory cells. Common respiratory disorders like chronic rhinosinusitis and asthma have recently been linked to elevated levels of IL-25, but the cellular source of it in the respiratory system was unknown until now. The *Journal of Allergy and Clinical Immunology* published the findings today.

The source of IL-25 is the solitary chemosensory cell (SCC), a highly-specialized type of cell similar to cells found in taste buds as well as cells in the gut. In non-inflamed tissue, SCCs make up about one percent of [human cells](#) lining the sinuses. However, when Penn researchers looked at human nasal polyps, the number of SCCs lining the polyp tissue was massively increased. Furthermore, treating these cells in a petri dish with the inflammatory molecule Interleukin-13 (IL-13), which has been implicated in driving nasal polyp formation and asthma, stimulated expansion of the SCCs and increased production of IL-25.

"The more of these cells are present, the more likely the body will mount an inappropriate, exaggerated immune response due to elevated levels of IL-25. The body ends up in a vicious cycle, and so it never goes back to its baseline," said the study's senior author Noam A. Cohen, MD, Ph.D., a professor of Otorhinolaryngology and director of Rhinology Research at Penn.

Surprisingly, the researchers also found that IL-25 is secreted into mucus, not tissue, meaning an extra step is apparently required before the cells can go to work. The IL-25 has to get from the mucus into the tissue to have its effect, otherwise the IL-25 is simply sneezed out or blown out in a tissue. However, if the linings of the sinuses have been breached by infection or irritants in the environment, then the IL-25 can access the underlying tissue, resulting in recruitment of additional

immune [cells](#) and local inflammation.

"We can measure levels of IL-25 in the mucus, so it's possible this can be an indicator of who will develop conditions like polyps or asthma," said the study's lead author Michael Kohanski, MD, Ph.D., a Rhinology Fellow at Penn. "Also, if we can bind up IL-25 before it reaches the [tissue](#), we may be able to prevent the inflammation altogether."

Cohen pointed out that the process of SCC's secreting IL-25 is normal, and thus is most likely not something that should be completely prevented.

"Rather, we want to control the excessive concentrations found in polyps in a targeted way, perhaps with a nasal spray," Cohen said. Cohen added that this approach may reduce clinical reliance on steroids. He and his team say they are planning further research to examine this approach.

**More information:** Michael A. Kohanski et al, Solitary Chemosensory Cells are a Primary Epithelial Source of Interleukin-25 in Chronic Rhinosinusitis with Nasal Polyps, *Journal of Allergy and Clinical Immunology* (2018). [DOI: 10.1016/j.jaci.2018.03.019](https://doi.org/10.1016/j.jaci.2018.03.019)

Provided by Perelman School of Medicine at the University of Pennsylvania

Citation: Researchers identify source of molecule linked to nasal polyps, asthma attacks (2018, May 23) retrieved 2 May 2024 from <https://medicalxpress.com/news/2018-05-source-molecule-linked-nasal-polyps.html>

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