

# A treatment for obesity-associated asthma

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(Medical Xpress)—Research conducted at Boston Children's Hospital indicates that obesity might cause asthma via factors in the immune system and suggests a new way of treating asthma in obese people—who often respond poorly to standard asthma medications. The study, conducted in a mouse model of asthma and in human lung fluid, was published online Dec. 15 by *Nature Medicine*.

A growing body of literature links asthma with [obesity](#), but the reason for the link has been unknown. Both conditions have become more common over the last several decades. The new study, led by Dale Umetsu, MD, PhD, and Hye Young Kim, PhD, of the Division of Allergy and Immunology at Boston Children's, explored obesity's effects on the immune system.

"Since it's been a puzzle to understand why obesity predisposes people to asthma, our goal was to find the connection between these two problems, which occur in both children and adults and to explore possible new treatments," says Umetsu.

The researchers studied mice that were fed a high-fat diet causing them to become obese. Unlike mice fed normal diets, the obese mice developed airway hyper-reactivity or constricted and twitchy airways, the predominant feature of asthma.

In the study, obesity appeared to alter the innate immune system—the body's first responder to infection—in several ways to cause lung inflammation. The lungs of the obese, asthmatic mice had several

differences from those of non-obese mice:

High levels of the protein interleukin 17A (IL17A), a cytokine (signaling molecule) associated with several inflammatory conditions

Increased numbers of the immune cells that produce IL17A, known as type3 innate lymphoid cells (ILC3 cells)

Activation of an inflammatory protein known as NLRP3 inside lung cells

Increased production of the cytokine IL-1 $\beta$ , a stimulator of ILC3 cells.

When the obese mice were genetically altered in that they could not produce IL17A or NLRP3, they did not develop asthma. But when the same mice had IL-1 $\beta$  or ILC3 cells placed directly in their lungs, either was enough to spur production of IL7A and to cause [asthma symptoms](#).

"We have discovered an inflammatory pathway to asthma that previously had not been recognized," says Umetsu "This pathway may be resistant to standard [asthma medications](#) such as corticosteroids."

In addition, when IL-1 $\beta$  production was blocked with the drug anakinra (Kineret, Swedish Orphan Biovitrum), used to treat rheumatoid arthritis, the [obese mice](#) did not develop asthma.

Although the researchers were unable to study human patients directly, they did examine lung fluid from 10 patients with lung disease who underwent bronchoalveolar lavage, a diagnostic test. Those with [severe asthma](#) had increased numbers of ILC3 cells producing IL-17 as compared with those with [mild asthma](#) or no asthma—circumstantial evidence suggesting that the [mouse model](#) may mirror what occurs in [obese people](#) who develop asthma.

"Obese people have been noted to have elevated systemic levels of IL-17 and IL-1 $\beta$ , particularly those who have non-allergic [asthma](#)," notes Umetsu.

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**More information:** Paper: [dx.doi.org/10.1038/nm.3423](https://doi.org/10.1038/nm.3423)

Provided by Children's Hospital Boston

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