A protein that shuts down immune cells in the lungs could be key to a new treatment for asthma attacks, a new report says.
The naturally occurring protein, called Piezo1, prevents a type of immune cell called type 2 innate lymphoid cells (ILC2s) from becoming hyperactivated by allergens.

An experimental drug called Yoda1 that switches on Piezo1 reduced the activity of these immune cells in mice, alleviating asthma symptoms, researchers report.

"Given the importance of ILC2s in allergic asthma, there is an urgent need to develop novel mechanism-based approaches to target these critical drivers of inflammation in the lungs," researcher Omid Akbari, a professor at the University of Southern California's Keck School of Medicine, said in a news release.

Once triggered by an allergen, ILC2s drive the inflammatory cascade that cause airways to swell and tighten, making it tough for asthma patients to draw breath.

In mouse research, researchers found that activated ILC2s naturally produce a protein called Piezo1 that limits their activity.

In the absence of Piezo1, mouse ILC2s became more responsive to allergy signals and promoted even more airway inflammation.

On the other hand, Yoda1 caused Piezo1 to kick into action, reducing the activity of ILC2s.

Human ILC2s also produce Piezo1, researchers say, and the drug Yoda1 also worked on lab-engineered mice with the human immune cells.

"Remarkably, treatment of these humanized mice with Yoda1 reduced airway hyperreactivity and lung inflammation, suggesting that Yoda1 may be used as a therapeutic tool to modulate ILC2 function and
alleviate the symptoms associated with ILC2-dependent airway inflammation in humans," Akbari said.

He said future research should focus on developing specific drugs to control Piezo1 in humans, which might help control or head off allergic asthma attacks.

The new study appears in the *Journal of Experimental Medicine*.


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