New UTMB study reveals mechanism central to pollen-induced allergies

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A new study from The University of Texas Medical Branch at Galveston has uncovered a mechanism that is central to becoming allergic to ragweed pollen and developing allergic asthma or seasonal nasal allergies. The findings are currently available online in the *American Journal of Respiratory Cell and Molecular Biology*.

When people are exposed to airborne allergens such as pollens, their immune system responds by sending neutrophils to the airways. Neutrophils are a type of white blood cells that move quickly to a site of injury or invading infection and cause inflammation. Inflammation is an important mechanism for protecting the body from injury and infection. However, people suffering from asthma or seasonal nasal allergies have developed heightened reactions to airborne allergens with repeated exposures, resulting in narrowed airways or stuffy nose.

The goal of this study was to evaluate how neutrophils may control and regulate the allergic responses and inflammation seen in response to inhaled ragweed pollen.

The researchers show that exposing regular mice to ragweed pollen extract prompted the synthesis of proteins called chemokines that attract neutrophils into the airways. They also tested the airway response to ragweed pollen in mice that were genetically modified to lack a gene that triggers chemokine production. They found that these altered mice did not have the amplified levels of chemokines, neutrophils or allergic sensitization when they were exposed to ragweed pollen.

"The study reported a remarkable paradigm-shifting observation: repeated administration of neutrophils to the airways along with ragweed pollen recreates allergic sensitization in these genetically modified mice," said UTMB's Sanjiv Sur, professor in the department of internal medicine, division of allergy and immunology, and professor in pediatrics and microbiology and immunology. "These data suggest that when the body is forced to react to the presence of pollen in the airways, it recruits neutrophils that induce a state of continuous oxidative stress in the airways. This type of cellular stress from any cause can worsen allergic asthma."

"We suggest that inhibiting recruitment of neutrophils by blocking chemokines may be a unique strategy for preventing pollen-induced allergic disorders," said Sur.

UTMB's Koa Hosoki, lead author and research scientist in the department of internal medicine, division of allergy and immunology added, "Until now, neutrophil recruitment after exposure to pollens was considered to be a non-specific event. In this study, we demonstrated for the first time that this crucial event initiates allergic sensitization to pollens. Future studies will have to determine if neutrophil recruitment is a universal mechanism of allergic sensitization to all inhaled allergens."

Source:
University of Texas Medical Branch at Galveston
