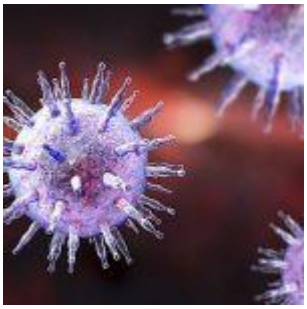
What drives allergic asthma? New insights



The most prevalent chronic illness in children is allergic asthma, which is marked by wheezing and respiratory problems brought on by inhaled allergens like pollen, mould, and pet hair. Allergic asthma can also last into adulthood. According to a recent study, the interaction of immune cells and nerves in the lungs can add to the emergence of this disease.

Researchers created novel newborn rodent models of allergen exposure for the research, replicating the development of allergic asthma from childhood to maturity. Tracking allergen-specific immune cells, also known as T helper 2 resident memory cells (Th2-TRMs), which are known to be the main cause of recurring allergic inflammation in the airways, was the focus of the research.

After infants are exposed to allergens, experiments showed that sympathetic neurons in the lungs generate dopamine and are located close to specific T helper 2 cells. These T helper 2 cells are more likely to change into Th2-TRMs and are given the order to create immune response-stimulating molecules, or cytokines, when dopamine binds to DRD4 receptors on these cells. When the same allergen was encountered as an adult after being exposed to it as a neonate, lung inflammation and T helper 2 cell change were decreased by blocking this dopamine binding after exposure.

One of the many age-related variables that controls Th2-TRMs

in the immature lung is probably dopamine transmission. New therapeutic options for the therapy of allergic asthma may be discovered with an improved knowledge of the early life Th2-TRM program's mediators.

Journal article: Wang et al., W., 2023. Lung dopaminergic nerves facilitate the establishment of T helper 2 resident memory cells in early life. *Journal of Allergy & Clinical Immunology*.

Summary by Stefan Botha