



The neuropeptide NMU amplifies ILC2-driven allergic asthma

Type 2 innate lymphoid cells (ILC2s) both contribute to mucosal homeostasis and initiate pathologic inflammation in allergic asthma. However, the signals that direct ILC2s to promote homeostasis versus inflammation are unclear.

To identify such molecular cues, Dr. Vijay Kuchroo and his colleagues from Brigham and Women's Hospital and the Broad Institute of MIT and Harvard closely examined lung-resident ILCs. Using single-cell RNA sequencing, the team explored more than 65,000 individual cells that existed under normal or inflammatory conditions, looking for genes that were more active in one state or subpopulation versus another.

Among many distinguishing genes they found, one in particular that stood out: *Nmur1*, a receptor for the neuropeptide NMU. Utilizing both *in vitro* and *in vivo* experiments, the team confirmed that NMU signaling can significantly amplify allergic inflammation when high levels of alarmins, molecules known to trigger immune responses, are present. The team also observed that ILCs co-located with nerve fibers in the lung. Neurons in the lung can induce smooth muscle contractions that manifest themselves as coughing and wheezing, two central symptoms of asthma.

This study demonstrates a novel neuro-immune pathway that exacerbates mucosal allergic inflammation *in vivo*. These findings may identify a way of blocking allergic lung inflammation by controlling neuropeptide receptors, which may lead to the development of a new therapeutic approach for preventing asthma.

The authors used Bio X Cell's anti-mouse CD3 (clone 145-2C11), anti-mouse CD28 (clone 37.51), and anti-mouse IFN γ (clone XMG1.2) antibodies to generate T_H2 cells for *in vitro* experiments.

Read the full article in Nature:

<http://www.nature.com/nature/journal/v549/n7672/full/nature24029.html>