Chlorine Triggers Protective Nerve Receptor

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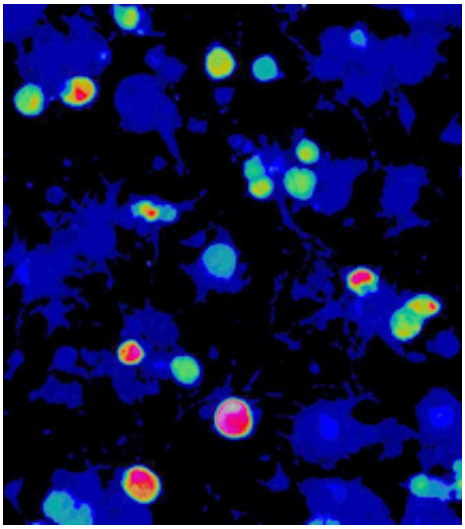

Airway nerve cells exposed to chlorine. Bright color indicates excitation of some nerve cells.

Inhaling chlorine triggers a nerve receptor that protects healthy people by inducing sneezing, coughing, and irritation, but can cause major problems for people with asthma and other respiratory problems, Yale School of Medicine researchers report today in the *Journal of Clinical Investigation.*

Prior to this study it was thought that chlorine and other oxidants induced pain and inflammation only through tissue injury. But the Yale team observed that mice lacking the receptor TRPA1 were insensitive to exposure to chlorine—which is used in industrial synthesis, for

disinfection of drinking water and swimming pools, and in household bleach. Interestingly, the receptor is the same one triggered by pungent mustard and noxious chemicals in cigarette smoke.

“We show that chlorine activates a specific receptor, TRPA1, in pain-sensing nerve endings in the airways,” said corresponding author, Sven-Eric Jordt, assistant professor of pharmacology. “We identified a population of neurons that fire in response to chlorine exposure, inducing pain and irritation, and narrowing airway passages—probably to protect the lung from chlorine damage.”

The problem with this response, he said, is that people whose respiratory systems already are compromised by asthma or congestion from colds and allergies then have a hypersensitive response to chemicals—their lungs are already doing what the brain is telling the respiratory system to do to protect itself.

“In these patients chlorine and other TRPA1 activators can trigger constriction of the bronchial pathways, and cause pain and discomfort in the airways,” Jordt said.

The silver lining, said Jordt, is that the study points to TRPA1 as a promising new target for the development of new drugs to suppress coughs and relieve pain and inflammation.

The lead authors are Bret Bessac and Michael Sivula of Yale. The study was conducted in collaboration with the laboratory of Lauren Cohn in the pulmonary section of Internal Medicine at Yale and funded by grants from the National Institute of Environmental Health Sciences of the National Institutes of Health.

Source: Yale University