

Scientists trace the path from pollutants in food to a heightened allergic response

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Exposure to the heavy metal cadmium is known to irritate the stomach and lungs or cause kidney disease, but new research links another health issue to inadvertently ingesting low doses of the pollutant: High



activation of the antibodies that cause an allergic response.

Researchers traced this link in mice to gut bacteria, that after exposure to ingested cadmium, over-produced an enzyme that degrades vitamin D—effectively creating conditions that mimic vitamin D deficiency. In terms of clinical effects, the mice sensitized to a specific allergen that consumed cadmium produced high levels of antibodies against the allergen as well as immune cells that increased their respiratory symptoms.

Separate epidemiological research has shown an association in children between vitamin D deficiency and higher susceptibility to asthma and other allergy symptoms. And a Congressional <u>report</u> released on Sept. 29 disclosed an unexpected source of cadmium in kids, announcing that dangerous levels of toxic heavy metals, including cadmium, had been detected in several brands of baby food.

"The problem is, because cadmium doesn't degrade easily—it has a half-life in the body of at least 15 years—if you are chronically exposed to low doses, it accumulates over time," said Prosper Boyaka, professor and chair of veterinary biosciences at The Ohio State University and senior author of the study. "It's also not something we can easily avoid being exposed to because it can remain in air, soil and water."

Most people ingest the natural element cadmium, a heavy metal used for batteries and making pigments, by eating plant and animal foods that have absorbed the pollutant or drinking contaminated water. The Environmental Protection Agency lists cadmium among eight metals considered extremely toxic at small concentrations.

Boyaka and colleagues found that an experimental compound that inhibits the activated enzymes reduced the allergic response in mice that ingested cadmium.



"We proposed two possible strategies in the paper," Boyaka said. "One is vitamin D supplementation, but that has to happen before cadmium exposure has caused a heightened allergic reaction, so the question would be when to use a supplement. We also propose targeting those enzymes as a way to prevent the heightened allergic response."

The research was published recently in the journal *Mucosal Immunology*.

Environmental pollutants such as cadmium and lead are considered contributors—along with genetic predisposition and overuse of antibiotics—to the growing number of people with allergies, but the mechanisms behind that association aren't well-understood, Boyaka said.

In this study, researchers added what is considered a "subtoxic" dose of cadmium to <u>drinking water</u> consumed by groups of mice for 28 days. Using a mouse model that simulates human <u>genetic predisposition</u> to an egg allergy, the team exposed the mice to an egg protein to test their allergic response.

Mice that drank water containing subtoxic doses of cadmium and then were exposed to the allergen had a stronger allergic reaction—in the form of internal inflammatory actions and allergy symptoms—than the response in control mice.

"Our hypothesis was that cadmium would change the microbe population in the gut because we know that dysbiosis, or a change in the microbiota, can drive allergic responses. And yes, giving those tiny, tiny doses of cadmium in the drinking water did change allergic sensitization," Boyaka said.

The effect was known to occur in the gut, as expected, because germ-free mice—which lack intestinal microbes—that were orally exposed to cadmium did not over-produce the antibody that causes an allergic



response. But the researchers determined the cadmium wasn't killing cells or making the intestines leaky. A series of experiments linked cadmium in the gut to production of inflammatory molecules—as well as to stimulation of the two enzymes that degrade vitamin D.

"That's the main finding—after exposure to subtoxic doses of heavy metals, the pollutants remain in soft tissue, including in the gut. And what they do is make cells more reactive. In the gut, specifically, bacteria will make certain cells produce more of the enzyme that degrades vitamin D," Boyaka said. "That's a connection that we did not know before."

Boyaka's lab is now investigating potential compounds that could be used to block the vitamin D-degrading enzymes.

Co-authors, all from Ohio State, include Eunsoo Kim, Astrid Bonnegarde-Bernard, Stephen Opiyo, Marisa Joldrichsen, Zayed Attia, Brian Ahmer and Estelle Cormet-Boyaka.

More information: Eunsoo Kim et al, Pollutants enhance IgE sensitization in the gut via local alteration of vitamin D-metabolizing enzymes, *Mucosal Immunology* (2021). DOI: 10.1038/s41385-021-00440-4

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