

# Researchers study the link between vitamin D and inflammation

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Scientists recently gained insights into how vitamin D functions to reduce inflammation caused by immune cells that might be relevant to the responses during severe COVID-19. In a study jointly published by Purdue University and the National Institutes of Health, scientists do just that.

Majid Kazemian, assistant professor in the departments of Computer Science and Biochemistry at Purdue University, was co-lead author of the highly collaborative study, along with Dr. Behdad Afzali, chief of the Immunoregulation Section of the National Institutes of Health's National Institute of Diabetes and Digestive and Kidney Diseases.

"Our work demonstrates a mechanism by which vitamin D reduces inflammation caused by T cells. These are important cells of the immune system and implicated as part of the immune response to the infection causing COVID-19. Further research, especially clinical trials, and testing in patients, are necessary before this can be adopted as a [treatment option](#)," Kazemian said. "We do not recommend the use of normal vitamin D off the shelf at the pharmacy. No one should be taking more than the recommended doses of vitamin D in an attempt to prevent or combat COVID infections."

Previous studies have shown vitamin D's ability to reduce the inflammation caused by T cells— inflamed cells in the lung characteristic of the most severe and dangerous cases of COVID-19. But as important as understanding that a drug works is understanding the how and the why. This is both to maximize benefit and minimize harm (such as preventing people from eating livestock dewormer or injecting household cleaners into their veins) as well as to pave the way for future treatments.

If scientists understand how vitamin D works to combat inflammation, they understand more about how both the drug and related diseases work, paving the way for new, even more effective drugs.

Kazemian and his team began by studying how viruses affect lung cells in a previous study. Finding that viruses can trigger a biochemical pathway, known as the immune complement system, the researchers started looking for ways to disrupt that pathway and ameliorate the

subsequent inflammation.

The team studied and analyzed individual lung cells from eight people with COVID—something only possible because of Kazemian's experience with gene sequencing and data mining. They found that in the lung cells of people with COVID, part of the immune response was going into overdrive, exacerbating lung inflammation.

"In normal infections, Th1 cells, a subset of T cells, go through a pro-inflammatory phase," Kazemian said. "The pro-inflammatory phase clears the infection, and then the system shuts down and goes to anti-inflammatory phase. Vitamin D helps to speed up this transition from pro-inflammatory to the anti-inflammatory phase of the T cells. We don't know definitively, but theorize the vitamin could potentially help patients with severe inflammation caused by Th1 cells."

In patients with COVID-19, the pro-inflammatory phase of the Th1 cells seems not switched off, possibly because the patients didn't have enough vitamin D in their system or because something about the cell's response to vitamin D was abnormal. In that case, the researchers posit, adding vitamin D to existing treatments in the form of a prescribed highly concentrated intravenous metabolite may further help people recovery from COVID infections, though they have not tested this theory.

"We found that vitamin D—a specialized form of it, not the form you can get at the drugstore—has the potential to reduce inflammation in the test tube, and we figured out how and why it does that," Kazemian said. However, it's important to understand that we did not carry out a clinical study, and the results of our experiments in the [test tube](#) need to be tested in [clinical trials](#) in actual patients."

The research was published in *Nature Immunology*.

**More information:** Daniel Chauss et al, Autocrine vitamin D signaling switches off pro-inflammatory programs of TH1 cells, *Nature Immunology* (2021). [DOI: 10.1038/s41590-021-01080-3](https://doi.org/10.1038/s41590-021-01080-3)

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