

Vitamin D may prevent diabetes



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Do you take vitamin D?

Vitamin D deficiency has been linked to type 2 diabetes and heart disease, two illnesses that commonly occur together and are the most common cause of illness and death in Western countries.

Both disorders are rooted in chronic inflammation,



which leads to insulin resistance and the buildup of artery-clogging plaque.

Now new research in mice at Washington University School of Medicine finds that vitamin D plays a major role in preventing

the inflammation that leads to type 2 diabetes and atherosclerosis.

Studying mice that lacked the ability to process vitamin D in immune cells involved in inflammation, the researchers found that the animals made excess glucose, became resistant to insulin action and accumulated plaques in their blood vessels.

“The finding that vitamin D helps regulate glucose metabolism may explain previous epidemiological studies identifying an increased risk of diabetes in patients with vitamin D deficiency,” said senior investigator Carlos Bernal-Mizrachi.

In the study, inactivation of the vitamin D receptor induced diabetes and atherosclerosis, so normalizing vitamin D levels may have the opposite effect, said the associate professor of medicine and of cell biology and physiology.

Inadequate vitamin D turned immune cells into transporters of fat. The findings suggest that getting enough vitamin D may reduce those properties in immune cells, decreasing inflammation and reducing the onset of a combination of heart disease and diabetes.

Bernal-Mizrachi and his team are conducting clinical studies in people who have type 2 diabetes, treating them with vitamin D to see whether it can prevent some of the complications of diabetes and inflammation in humans, too.

Reference:

“Deletion of macrophage vitamin D receptor promotes insulin resistance and monocyte cholesterol transport to accelerate atherosclerosis in mice,” Oh J, Riek AE, Darwech I, Funai K, Shao JS, Chin K, Sierra OL, Carmeliet G, Ostlund RE, Bernal-Mizrachi C, *Cell Reports March 19, 2015*