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## 8. Can Vitamin D Save Your Life?

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### New studies highlight the importance of the forgotten vitamin.

by Mariana Gosnell

For years doctors believed that vitamin D, sometimes called the “sunshine vitamin” because sunlight triggers the body to produce it, was important primarily in preventing rickets (a softening of the bones) in children. Once milk became fortified with vitamin D, rickets pretty much disappeared, and the problem of vitamin D deficiency seemed to have been solved. But according to Michael F. Holick, director of the Vitamin D, Skin, and Bone Research Laboratory at Boston University Medical Center, who has spent 30 years studying the vitamin, “rickets can be considered the tip of the vitamin D–deficiency iceberg.”

Today a lack of the vitamin has been linked to a host of other maladies, including cancers of the colon, prostate, and breast; tuberculosis; schizophrenia; multiple sclerosis; hip fractures; and chronic pain. How can one vitamin play a role in so many diverse illnesses? The answer seems to lie in the fact that most tissues and cells in the human body (and not just those in the intestine and bone that help fix calcium) have receptors for vitamin D, suggesting that the [vitamin is needed](#) for overall optimal health. In addition, some cells carry enzymes for converting the circulating form of vitamin D to the active form, making it available in high concentrations to the tissues locally.

A recent laboratory experiment at Boston University revealed that by activating the circulating form of the vitamin, prostate cells could regulate their own growth and possibly prevent the rise of cancer. Directly or indirectly, Holick points out, “the active form of vitamin D controls up to 200 different genes,” including ones responsible for cell proliferation, differentiation, and death.

Theories about vitamin D’s cancer-prevention qualities have begun to be validated. In June, Joan M. Lappe, professor of nursing and medicine at Creighton University School of Medicine in Omaha, and her colleagues [published](#) the results of a 4-year, double-blind, randomized trial in which nearly 1,200 healthy postmenopausal women took calcium alone, calcium with 1,100 international units (IU) of vitamin D a day, or a placebo. The women who took calcium with vitamin D had a 60 percent lower risk of developing cancers of any type than the placebo group; the calcium-only group’s risk didn’t significantly change.

Currently, the median vitamin D intake of adult Americans is only about 230 IU a day; Lappe was prompted by the study’s findings to recommend the dose be increased to 1,500 to 2,000 IU. “It’s low risk, with maybe a high payoff,” she [told a Canadian newspaper](#)

in June. Vitamin D comes from three sources: the sun’s ultraviolet (UVB) rays penetrating the skin, a few D-rich foods like fatty fish and some fortified foods, and supplements. The Canadian Paediatric Society has already recommended that pregnant or breast-feeding women get 2,000 IU of vitamin D daily.

Some clinicians have suggested that increased vitamin D intake might help ward off multiple sclerosis (MS), believed to be a progressive autoimmune disease. Last December, a team of researchers at the Harvard School of Public Health and other institutions [published](#) results from the first large-scale prospective study of the relationship between vitamin D levels and MS. After analyzing stored blood samples taken from 7 million military personnel and identifying those individuals who developed MS during a 12-year period, the team determined that the risk of getting MS was 62 percent lower for those whose blood concentration of vitamin D put them in the top quintile than for those in the bottom quintile. The study did not make clear, however, whether low vitamin D levels were a cause of MS or a marker of MS risk.

Vitamin D status may also affect vulnerability to infections. For example, African Americans need more sun exposure than Caucasians to make sufficient vitamin D; they also suffer from increased risk of tuberculosis. In a breakthrough study published in March, scientists from several institutions, including UCLA, discovered a possible link. On encountering the TB bacillus, receptors on immune-system scavenger cells known as macrophages stimulate the conversion of circulating vitamin D to its active form, which produces a peptide that destroys the bacillus. If circulating levels of D are low, macrophages can’t activate the vitamin D to initiate this response. A similar scenario could be operating with other infectious agents, maybe even the influenza virus.

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