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IN THE NEWS

New research shows how broccoli beats cancer

There's an abundance of scientific evidence today to suggest that fruits and vegetables can aid in cancer prevention. In the last decade, researchers uncovered one compound in particular that's a potent anti-cancer agent. Sulforaphane is most highly concentrated in broccoli, as well as in other cruciferous vegetables, such as Brussels sprouts, cabbage and cauliflower. According to the American Institute of Cancer Research, animal studies to date have shown that sulforaphane can dramatically reduce the number of malignant tumors, reproduction, growth rate and size, as well as delay cancer onset. Some research findings suggest that sulforaphane might exert its anti-cancer effects by activating detoxifying proteins in the body known as 'Phase 2 detoxifying enzymes' [Proc Natl Acad Sci USA 1997 Sep 16;94(19): 10367-10372]. The role of these special enzymes appears to be scavenging for cancer-causing molecules.



Now a new study adds to these positive reports about sulforaphane by proposing that the compound may additionally play a more direct role in cancer prevention by inducing cell cycle arrest and apoptosis [Cancer Res 2000 Mar 1;60(5):1426-1433]. After treating human colon cancer cells with sulforaphane, the researchers found a number of chemical and structural cellular changes that indicated cancer cells were being killed off by the compound. The study authors also go on to explain that broccoli contains a stable sulforaphane precursor (isothiocyanate), which causes broccoli to release its sulforaphane content when the vegetable is chewed or chopped.

Earlier research at Johns Hopkins University found that sulforaphane blocked the formation of mammary tumors in rodents treated with a potent carcinogen [Proc Natl Acad Sci USA 1994;91:3147-3150]. Results showed that the number of rats that developed tumors was reduced by 60%, the number of tumors they developed dropped by 80%, and tumor size decreased by 75%. Other research by the American Health Foundation showed that sulforaphane inhibited the appearance of premalignant lesions of colon cancer in rats [Proc Natl Acad Sci USA 2000;41:660]. And, almost a decade ago, scientists had found that sulforaphane boosted the activity of two detoxifying enzymes—quinone reductase and glutathione transferase—in experimental mice [Proc Natl Acad Sci USA 1992 Mar 15;89(6):2399-2403]. As a result, scientists speculated that sulforaphane may regulate the metabolism of carcinogens, possibly by helping to neutralize harmful molecules and minimizing the oxidative damage that predisposes cells to cancerous growth. Now the latest research adds another piece to the puzzle that explains how sulforaphane goes about subduing cancerous growth and development.

—Angela Pirisi

Folic acid and vitamin B12 save lives and money

An eye-opening study published in the August 22, 2001 edition of the Journal of American Medical Association concluded that supplementation with folic acid and vitamin B12 would result in a tremendous increase in health and longevity of the American population at a low cost, while saving 24 billion dollars in healthcare costs over the coming decade.

Researchers at the University of California at San Francisco studied available data on U.S. homocysteine levels. Even modestly elevated homocysteine levels are associated with higher incidence of stroke and heart attack, as well as with increased mortality from cardiovascular disease. A previous study showed that folic acid could lower homocysteine by 25%; the addition of vitamin B12 led to a further 7% drop. The potent homocysteine-lowering effect of folic acid was also demonstrated by a decrease in homocysteine levels in the U.S. population since 1998, when the FDA mandated folic acid enrichment of cereal products. This enrichment alone should produce a 13% decrease in heart disease and mortality in men, and 8% in women.



This study projects that if people diagnosed with heart disease and/or high homocysteine use a 1 mg supplement of folic acid and .5 mg of B12, there should be 310,000 fewer cardiovascular disease-related deaths in 2001 to 2011. The cost of vitamin therapy would be very low compared to the cost of other treatments.

Note that the authors of the study recommend, at least for individuals at increased risk, a dose of folic acid much higher than the official recommended dosage. It should also be noted that folic acid is better absorbed from supplements than from food.

“Folic acid, vitamin B12 show potential as heart disease treatments.” EurekAlert 21-08-2001.

—Ivy Greenwell

Lutein helps prevent atherosclerosis

Lutein, the yellow carotenoid found in egg yolks and green leafy vegetables such as spinach and kale, is best known as a nutrient that helps protect against macular degeneration. Now lutein has also been shown to be effective against atherosclerosis. A recent University of Southern California study on the cardiovascular effects of lutein consisted of three parts. The epidemiological portion of the study measured levels of serum lutein at baseline and 18 months later in a group of 480 men and women aged 40 to 60, enrolled in the Los Angeles Atherosclerosis Study. The subjects had no history of heart disease. Those with the highest serum lutein (the upper 20%) had the least increase in carotid artery thickness: 0.004 millimeters versus 0.021 millimeters for those in the lowest 20% of serum lutein, a fivefold difference. This finding was confirmed in the mouse model. The atherosclerotic lesions in mice supplemented with lutein were 43% smaller than in controls.

The in-vitro portion of the study compared tissue culture of cells from the inner wall of human carotid arteries pretreated with various doses of lutein in terms of LDL-induced migration of monocytes, a type of large white blood cell that plays a major role in the progression of atherosclerosis. Lutein inhibited monocyte migration in a dose-dependent manner, higher doses being most effective in preventing monocyte recruitment.

This study suggests that lutein may be of crucial importance in preventing atherosclerosis.

—IG

Dwyer JH et al. Oxygenated carotenoid lutein and progression of early atherosclerosis: the Los Angeles atherosclerosis study. *Circulation* 2001; 103:2922-7.

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