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## REPORT

**Prostate Cancer**

and Cruciferous Vegetables

By Terri Mitchell

A new study in the Journal of the National Cancer Institute shows that three or more servings of cruciferous vegetables a week slashes prostate cancer risk almost in half. The study, which involved more than 600 men with prostate cancer, was conducted in the Seattle area. This confirms data from a Canadian study showing that cruciferous vegetables, tomatoes, green vegetables and beans/lentils/nuts all substantially reduce the risk of prostate cancer.

Cruciferous vegetables such as cabbage, brussels sprouts and cauliflower contain cancer-fighting substances, including indole-3-carbinol (I3C). I3C changes the way estrogen is metabolized, and may prevent estrogen-driven cancers such as breast cancer. It's presently unknown whether I3C is the reason cruciferous vegetables protect against prostate cancer.

Unlike breast cancer, which often grows under the influence of estrogen, prostate cancer is not so straightforward. Some prostate cancers will stop growing when estrogen is blocked with the drug tamoxifen, but others will not. In most studies, androgen (male) hormones are the culprit, not estrogen (female) hormones. And when estrogen is given, these androgen-driven cancers will stop growing. When it comes to prostate cancer prevention, data suggests that it is the balance of hormones that's important rather than the level of any single hormone. However, this still leaves open the question of whether I3C-which alters estrogen metabolism-might be effective in preventing or retarding prostate cancer. It may, because I3C has other anticancer properties.

**I3C stops cancer growth**

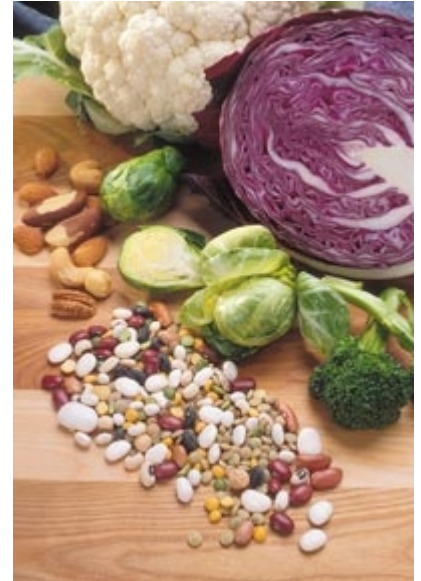
One of I3C's most important actions is to interfere with cancer cells' ability to grow. A normal cell goes through "checkpoints" to ensure that everything is okay before it replicates. Cancer cells override the checkpoints, and grow at break neck speed. I3C restores the brakes. By forcing the cancer cells to stop at the checkpoint, the body has an opportunity to destroy deviant cells and stop the growth of cancer. Chemotherapeutic drugs such as doxorubicin work the same way.

I3C is also potentially important in restoring communication between testosterone, estrogen and progesterone. This can be critical because these hormones have a direct effect on the growth of prostate cancer cells. Sex hormones are essentially messengers that go to cells and tell them to do, or not do something-like grow. In prostate cancer, the ability of normal cells to "get the message" is disrupted. The messenger hormones are sending the wrong messages. I3C and certain other substances, such as flavanoids, may have the power to restore communication through something called the "Ah receptor" (aryl hydrocarbon). The Ah receptor is a very powerful "grow" switch which controls estrogen. If you can get control of the Ah receptor, you can control estrogen.

The Ah receptor is notorious. This is where the highly toxic chemical, dioxin, and related chemicals enter cells. When dioxin and its relatives get into the Ah receptor, immunosuppression and carcinogenesis can occur. When plant substances like I3C get in, the opposite can happen. It remains to be seen whether I3C's ability to control Ah, and put the brakes on estrogen, will contribute to its potential as an anti-prostate cancer supplement. Evidence suggests it will.

**What causes prostate cancer?**

No single cause of prostate cancer has ever been determined, but the single biggest risk factor for prostate cancer is eating products that contain animal fat. In one study involving over 6,000 men, those who routinely ate milk, cheese, eggs and meat (all of them) had a 3.6 times greater risk of fatal prostate cancer than those who did not. A more recent study shows that the more foods with animal fat a man eats, the greater the risk of turning up with advanced prostate cancer. This study, from the National Cancer Institute, puts African Americans at particular risk. It is not fat per se that is the problem; it is animal fat. Studies on vegetable fat don't show an association to prostate cancer. And studies on heterocyclic amines (carcinogens created when meat is cooked)



don't show a strong association. A study from Harvard indicates that red meat is the worst culprit. Environmental chemicals tend to accumulate in meat fat. (Is it a coincidence that when bulls destined for slaughter are implanted with synthetic estrogen, their prostates show cancer-like changes? Although it is illegal in this country to sell animal products containing hormones and drugs or chemicals, it happens routinely. For more on this, see the FDA's "Enforcement Reports" and "Warning Letters.") In a study from the Netherlands, however, only cured meat and milk were associated with prostate cancer; fresh meat, fish, cheese and eggs were not.

Seventy-five percent of all prostate cancers are diagnosed in men over 65. But this is no magical number, and it's probable that many of these cancers were present years before they were diagnosed. Prostate cancer is a slow-growing cancer. The five-year survival rate for localized cancer is good news-100%. However, after five years, the survival rate gradually declines to 52% at 10 years. This argues for prevention. Don't just sit around thinking it won't happen to you. Act now for the future. Prostate cancer is one of the few cancers where a strong link exists between eating one type of food (meat) and getting the disease. Even the conservative American Cancer Society advises that men at risk curtail their intake of meat and eat plant-based foods instead. Based on new studies coming out, one of those vegetables should be cruciferous. Or-if you're a broccoli hater like the recent former president-I3C capsules may provide prostate protection. Prostate cancer is the second most prevalent cancer in men. If you're a male over age 50, that is something to think about.

Note: Signs and symptoms of prostate cancer mostly involve urinary problems. However, in most early-stage cases, there are no symptoms at all. If in doubt, get checked. Annual PSA blood test and digital rectal exam are strongly advised.

For more information on prostate cancer, see the American Cancer Society's Cancer Resource Center at [www.cancer.org](http://www.cancer.org). They have excellent information on testing procedures and the cancer itself. The National Cancer Institute has approximately 200 prostate cancer trials underway. See [www.ClinicalTrials.gov](http://www.ClinicalTrials.gov). One of them is looking at how vitamin E, green tea, soy, fruits, vegetables, fiber and fat may affect levels of prostate specific antigen (the marker for prostate problems). To access the FDA's "Enforcement Reports" and "Warning Letters", go to [www.fda.gov/foi/electr.htm](http://www.fda.gov/foi/electr.htm) and search under "dairy" and "cattle/food".

## References

- Chang YC, et al. 1999. Cytostatic and antiestrogenic effects of 2-(indol-3-ylmethyl)-3,3'-diindolylmethane, a major in vivo product of dietary indole-3-carbinol. *Biochem Pharmacol* 58:825-34.
- Clinton SK, et al. 1997. Dietary fat and protein intake differ in modulation of prostate tumor growth, prolactin secretion and metabolism, and prostate gland prolactin binding capacity in rats. *J Nutr* 127:225-7.
- Cohen JH, et al. 2000. Fruit and vegetable intakes and prostate cancer risk. *J Natl Cancer Inst* 92:61-8.
- Giovannucci E, et al. 1993. A prospective study of dietary fat and risk of prostate cancer [see comments]. *J Natl Cancer Inst* 85:1571-9.
- Groot MJ, et al. 1998. Combinations of growth promoters in veal calves: consequences for screening and confirmation methods. *Zentralbl Veterinarmed A* 45:425-40.
- Hayes RB, et al. 1999. Dietary factors and risks for prostate cancer among blacks and whites in the United States. *Cancer Epidemiol Biomarkers Prev* 8:25-34.
- Ip MM, et al. 1980. Functionality of estrogen receptor and tamoxifen treatment of R3327 Dunning rat prostate adenocarcinoma. *Cancer Res* 40:2188-93.
- Jain MG, et al. 1999. Plant foods, antioxidants, and prostate cancer risk: findings from case-control studies in Canada. *Nutr Cancer* 34:173-84.
- Jana NR, et al. 1999. Cross-talk between 2,3,7,8-tetrachlorodibenzo-p-dioxin and testosterone signal transduction pathways in LNCaP prostate cancer cells. *Biochem Biophys Res Commun* 256:462-8.
- Klinge CM, et al. 1999. The aryl hydrocarbon receptor (AHR)/AHR nuclear translocator (ARNT) heterodimer interacts with naturally occurring estrogen response elements. *Mol Cell Endocrinol* 157:105-19.
- Noble RL. 1980. Production of Nb rat carcinoma of the dorsal prostate and response of estrogen-dependent transplants to sex hormones and tamoxifen. *Cancer Res* 40:3547-50.
- Norrish AE, et al. 1999. Heterocyclic amine content of cooked meat and risk of prostate cancer. *J Natl Cancer Inst* 91:2038-44.
- Schilt R, et al. 1998. Pour-on application of growth promoters in veal calves: analytical and histological results. *Analyst* 123:2665-70.
- Schuurman AG, et al. 1999. Animal products, calcium and protein and prostate cancer risk in the Netherlands Cohort Study. *Br J Cancer* 80:1107-13.
- Snowdon DA, et al. 1984. Diet, obesity, and risk of fatal prostate cancer. *Am J Epidemiol* 120:244-50.
- Verbeke R, et al. 1976. Quality of the meat after the application of anabolic agents in young calves. *Environ Qual Saf Suppl* 5:123-30.

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[Back to the Magazine Forum](#)

treatment. You should consult with a healthcare professional before starting any diet, exercise or supplementation program, before taking any medication, or if you have or suspect you might have a health problem. You should not stop taking any medication without first consulting your physician.