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IN THE NEWS

Green Tea Extract Helps Prevent Prostate Cancer



Compounds in green tea help prevent progression to prostate cancer in men at high risk for the disease, according to recent research findings.* Prostate cancer is the second leading cause of cancer-related death among American men.

In their study, Italian re-searchers enrolled 62 men with high-grade prostatic intraepithelial neoplasia, a pre-cancerous condition. Thirty-two of the men were given 200 mg of green tea catechins three times daily for one year, while 30 men received a placebo. Repeat prostate biopsies were performed on all participants six and 12 months after beginning the study.

Approximately one third of men with high-grade prostatic intraepithelial neoplasia can be expected to progress to cancer over the course of one year. This statistic corresponds with results seen in the placebo group, in which nine men (30%) developed prostate cancer during the one-year study period. However, only one (3%) of the men receiving green tea catechins developed prostate cancer—a 90% reduction in the risk of progressing to prostate cancer over one year.*

The supplemented group consumed 600 mg of green tea catechins daily, with the polyphenol epigallocatechin-3 gallate, or EGCG, accounting for approximately one half of the catechins. Previous studies have shown that green tea catechins and other polyphenols inhibit certain cancer cell lines, while epidemiological studies suggest that men who consume high levels of dietary polyphenols experience a decreased risk of prostate cancer.

This study is the first to demonstrate in-vivo protection by green tea catechins against prostate cancer. While additional studies are needed, the researchers recommend considering green tea for the prevention of prostate cancer, especially in high-risk populations such as the elderly, African-Americans, and men with a family history of the disease. Green tea catechins are inexpensive, well-tolerated, and non-toxic to normal cells.

—Linda M. Smith, RN

Reference

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Melatonin May Protect Against Cell Phone Radiation

Brief exposure to radiation emitted by cell phones in-duces pathological changes in the skin and kidneys that may be attenuated by melatonin, report Turkish researchers.^{1,2}

Cell phones emit and receive radio frequency radiation in the ultra-high frequency range (824-894 megahertz, or MHz). The biological impact of exposure to this radiation is a function of the time exposed and the power of the cell phone signal. Increased oxidative stress induced by radio frequency radiation may contribute to DNA changes and increased risk of certain cancers.

The Turkish researchers investigated the effects of cell phone radiation on the skin and kidneys. The skin, they theorized, serves as the body's protective layer and may be more vulnerable to radiation damage than internal organs. The kidneys may also be vulnerable to radiation damage from cell phones, which are frequently worn on belts.

Thirty laboratory rats were equally divided into three groups. Group 1 rats served as non-treated controls, Group 2 rats received

30 minutes of 900-MHz whole-body irradiation daily for 10 days, and Group 3 rats received melatonin at a dose of 10 mg/kg of body weight each day for 10 days before receiving 30 minutes of 900-MHz whole-body irradiation.^{1,2} The scientists then examined skin sections for radiation injury and analyzed blood and urine markers of lipid peroxidation, kidney damage, and oxidative stress.

The skin of Group 2 rats exhibited diverse signs consistent with acute injury, and the rats also demonstrated increased lipid peroxidation, kidney impairment, and oxidative stress. As expected, no such changes occurred in the Group 1 controls. In the Group 3 rats, melatonin prevented nearly all skin changes and other signs of radiation-induced damage.^{1,2}

The study authors concluded that the antioxidant and free radical-scavenging properties of melatonin offer significant protection from the damaging effects of routine cell phone use, particularly on the skin and kidneys.

—Linda M. Smith, RN

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2. Oktem F, Ozguner F, Mollaoglu H, Koyu A, Uz E. Oxidative damage in the kidney induced by 900-MHz-emitted mobile phone: protection by melatonin. *Arch Med Res*. 2005 Jul-Aug;36(4):350-5.

Bromelain Reduces Bowel Inflammation



Oral supplementation with bromelain decreases inflammation of the colon in animals with active inflammatory bowel disease (IBD) while reducing the incidence and severity of spontaneous colitis, report researchers at Duke University Medical Center.*

Earlier anecdotal reports indicated that the protein-digesting activity of bromelain, an enzyme fraction derived from pineapple stem, may have contributed to the remission of ulcerative colitis in two patients who had been unsuccessfully treated with conventional medicine.

Treatment with up to 1000 mg a day of high-quality bromelain led to a decreased incidence and severity of spontaneous colitis in genetically susceptible mice after 18 weeks. Bromelain supplementation also significantly reduced inflammation in test animals with established IBD.

IBD comprises two conditions: ulcerative colitis, characterized by inflammation of the lining of the large bowel or rectum, and Crohn's disease, an inflammation affecting the full thickness of the bowel that may involve any part of the gastrointestinal tract. Inflammation of these areas may hamper nutrient and water absorption, and lead to blood loss from the inflamed bowel walls. IBD can cause significant pain and diminished quality of life, while contributing to nutritional deficiencies and additional health problems.

The encouraging results of this study may lead to additional clinical trials to confirm the anti-inflammatory benefits of supplemental bromelain.

—Christie C. Yerby, ND

Reference

- * Hale LP, Greer PK, Trinh CT, Gottfried MR. Treatment with oral bromelain decreases colonic inflammation in the IL-10-deficient murine model of inflammatory bowel disease. *Clin Immunol*. 2005 Aug;116(2):135-42.

Fish Oil Enhances Heart Rate Variability

Omega-3 fatty acids from fish oil may help protect the cardiovascular system by promoting heart rate variability, according to researchers from Mexico and the United States.*

Omega-3 fatty acids are known to help prevent heart disease and decrease the risk of sudden death in those with established

heart disease. This recent finding demonstrates a new physiological basis for the protective effects of omega-3 fatty acids.

Heart rate is a function of the autonomic nervous system, and variation in heart rate from beat to beat and minute to minute is a sign of good health. Clinically, a decrease in heart rate variability is associated with arrhythmias and portends poor survival after myocardial infarction.

In this study, 52 nursing home patients were randomly assigned to receive either 2 grams a day of omega-3 fatty acids from fish oil or alpha-linolenic acid, a plant-derived omega-3 fatty acid from soy oil capsules. Heart rate variability was measured for six minutes every other day over a six-month period using standardized, non-invasive methods. After just one month, those supplementing with fish oil exhibited significant improvements in various measures of heart rate variability. The soy oil recipients saw lesser but still significant increases in heart rate variability.

The researchers concluded that omega-3 fatty acids—particularly those from fish oil—appear to exert their lifesaving effects in part by promoting heart rate variability. Until now, omega-3 fatty acids' anti-inflammatory and anti-thrombotic properties have been thought to account for their protective effects. This study links omega-3 fatty acids to a measurable effect on cardiovascular physiology. Regular exercise, weight loss, stress reduction, and restoration of normal sleep have also been shown to improve heart rate variability.

—Linda M. Smith, RN

Reference

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IN THE NEWS

DHEA Improves Insulin Response



Dehydroepiandrosterone (DHEA) administered to women with adrenal insufficiency improves insulin sensitivity, according to Mayo Clinic investigators.¹

DHEA, a steroid released from the adrenal gland, has been shown benefit some medical conditions coincident with aging, such as heart disease and diabetes. Adrenal insufficiency and diminished DHEA levels become more prevalent with advancing age.

In the Mayo Clinic study, 28 women with adrenal insufficiency received 50 mg of micronized DHEA or placebo daily for 12 weeks, underwent a two-week washout period, and then received the alternative preparation for 12 weeks. After each of the two 12-week study periods, the participants underwent metabolic testing.

In as little as 12 weeks, replacement doses of DHEA produced a clinically significant decline in fasting insulin levels. Fasting plasma glucose levels were likewise lower with DHEA supplementation than with placebo. Improved “efficiency” of insulin during DHEA replacement was demonstrated by the greater amount of dextrose infusion required to maintain equivalent blood glucose levels in the two groups of patients.¹

This study strongly suggests that DHEA replacement increases insulin sensitivity and improves glucose metabolism, and that DHEA may play an important role in preventing type II diabetes.¹ These findings also support a recent study in which DHEA supplementation helped improve insulin sensitivity in otherwise healthy elderly men and women.²

DHEA may exert its effects by helping to decrease the abdominal fat that often increases with age and is associated with lowered tissue sensitivity to insulin. Abdominal obesity is associated with an increased risk of metabolic syndrome and type II diabetes.²

—Linda M. Smith, RN

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Blood Vessel Drugs Once Called Failures Halt Cancer Growth

Nobody believed Judah Folkman when, in the 1960s, he claimed that the growth of cancers could be stopped, even reversed, by blocking the tiny vessels that feed them blood. Over the years, however, he has survived peer rejection of his theory, and gone on to develop drugs that did what he predicted they would do.

In 1998, Endostatin™, one of several anti-blood-vessel-growth drugs developed in his lab, was hyped by the media as a “cure” for many different cancers. A scant seven years later, *Fortune* magazine derided it as a “failure.” Both statements turn out to be high exaggerations.

A professor of pediatric surgery and cell biology at Harvard Medical School and Children’s Hospital in Boston, Folkman today is excited by what he sees in the scientific, if not the public, press. Endostatin™, the drug *Fortune* called a failure, was used to treat 486 patients with lung cancer in China. At Dana-Farber Cancer Institute in Boston, it has given new lives to four adults who have been taking it, and it is helping children with brain and other tumors.



A related drug, called Avastin™, was approved for use in the United States in February 2004. Since then, 27 other countries have OK'd it for treating colon cancer. Avastin™ is also being tested on patients with kidney, breast, and ovarian cancers. In addition, another blood-vessel-growth blocker, Tarceva®, has been approved for treatment of lung cancer in the United States.

Researchers have also associated overgrowth of blood vessels with macular degeneration, the leading cause of blindness in elderly people. Convinced that blood-vessel-growth blockers can help these people, the Food and Drug Administration approved a drug called Macugen™ for this purpose in December 2004. Avastin™, which is made by Genentech Inc., also shows promise for treating macular degeneration.

A common antibiotic, doxycycline, was found to possess anti-blood-vessel-growth properties. So was the popular painkiller Celebrex®.

Thirty to 40 other blood-vessel-growth inhibitors are now undergoing tests in patients with different types of cancers. And additional varieties of the drugs are under development to treat other diseases. Every week, Folkman sees encouraging scientific reports on the drugs he has grandfathered, but he's not the type to gloat or scream for vindication. "It's a large, fast-moving field," he says modestly. "And I'm excited to see the high rate of progress."

UPS, DOWNS, AND UPS AGAIN

Folkman showed signs that he would revolutionize medicine in high school when, in the basement of his family's Ohio home, he made a crude pump to keep a rat's heart pumping blood. During medical training in the 1950s, Folkman and another student built an implantable pacemaker to shock weakened hearts back into a normal rhythm. But it was in 1961 when he made the discovery that now dominates his life.

Folkman and a colleague noticed that malignant mouse tumors implanted into isolated organs never grow beyond the size of a pinhead. But replant those tumors into the bodies of live animals, and they expand rapidly. From this, Folkman came up with the then-radical idea that tumors secrete proteins able to stimulate the growth of hair-thin blood vessels that bring them nutrients and carry away their wastes. He applied the name "angiogenesis," meaning "birth of blood vessels," to this process.

In 2005, no scientist or physician doubts the existence of angiogenesis or its major role in cancer, but in the early 1970s Folkman's idea was heavily criticized. "Fantasy," some experts labeled it.

By 1997, Folkman and his colleagues at Boston's Children's Hospital found a natural compound they called Endostatin™, which blocks the growth of blood vessels and shrinks tumors without the usual harsh side effects of chemotherapy.

The battle over Endostatin's efficacy as a drug, however, still rages. In 1998, the stock of the company that made the drug, EntrezMed, rocketed 381%. But the company went broke and had to stop making Endostatin™.

A major problem still exists, Folkman notes, over the way doctors measure the success or failure of anti-cancer drugs. If a tumor shrinks at least 50%, they call it a "partial response" to the medication. Anything less becomes a "no response." Endostatin™ has stabilized cancer growth, or produced slow tumor regression, over a period of three years in some patients. Although that's less than a 50% response, some of the patients have gone back to work and to playing golf. Four who have been on the drug for at least 3 1/2 years are enjoying normal lives. "They were very disappointed with media reports calling Endostatin™ a failure," Folkman points out.

Doctors at Harvard-affiliated Children's Hospital and Dana-Farber Cancer Institute can't get as much of the drug as they want. Mark Kieran of Children's Hospital is now testing Endostatin™ against all types of solid tumors in children, including those of the brain, bone, and muscle.

THE BOOM IS ON

Avastin™ (whose generic name is bevacizumab) enjoys good press and proves that Folkman has been on the right track for the past 33 years. “Avastin™ vindicates the idea that tumors can be effectively controlled by targeting the network of blood vessels that feed them,” says Joseph Paul Eder, a Harvard associate professor of medicine who treats patients at Dana-Farber Cancer Institute.

The drug has won approval from governments all over the world for its success in treating colon cancer. Once that door opened, researchers began trying it on other malignancies. Leonard Appleman is testing Avastin™ on kidney cancer patients at Dana-Farber. Harold Burstein, working at the same institute, has almost finished his tests of it on women with breast cancer, and says he will report his results in December. Others are treating ovarian cancer with Avastin™.

No results have been formally announced, but word from people close to these trials indicates that Avastin™, used by itself and in combination with other drugs, shows that the angiogenesis blocker boom is on. One scientist, who did not want to be identified but who has been involved for years with testing the drugs, says: “If I had cancer, I’d want Endostatin™ or Avastin™, or both.”

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