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REPORT

CLA INHIBITS PROSTATE CANCER GROWTH AND METASTASIS

Conjugated linoleic acid (CLA) has been shown in physiological concentrations (i.e. low doses) to inhibit proliferation of human tumor cell lines including glioblastoma, melanoma, colorectal, lung, breast, prostatic carcinoma and leukemia.

In a study published in *Anticancer Research* (1998 May-Jun; 18(3A): 1429-34), mice were injected with an aggressive human prostate cancer cell line (DU-145). Within two weeks, palpable tumor masses grew at the site of injection, and metastatic lesions to the lungs appeared in eight weeks. At the end of the 14th week, euthanasia was employed as the tumor burden completely overwhelmed the animal. Because the described tumor growth pattern was consistent and reproducible, this model was chosen to investigate the effects of linoleic acid, a common polyunsaturated fat found and consumed in high amounts in the Western diet, and CLA, a fatty acid that is deficient in most diets.

A fresh group of mice was fed either a standard lab chow diet (the control group), a diet spiked with 1% linoleic acid, or a diet spiked with 1% CLA for two weeks. The mice were then injected with the same aggressive human prostate cancer cell line that proved so lethally effective to the test model group.

After eight weeks, the mice fed linoleic acid showed faster tumor growth compared to the control and CLA supplemented group. At necropsy, the CLA-fed group displayed a significantly smaller tumor burden compared to both the control and linoleic acid-fed mice. Notably, in 33% of the CLA-fed mice, the primary tumor progressed for five weeks, and then "fell out," leaving behind only a necrotic scar. Most of the other tumors in the CLA-fed mice began to show necrosis after six weeks. This necrosis of the primary tumor is the type of "cure" human cancer patients often hope for.

The CLA-fed mice displayed significantly reduced rates of lung metastasis. The findings showed that lung metastasis occurred in 80% of the control group and 100% of the linoleic acid-fed group. By contrast, only 10% of animals receiving the CLA supplemented diet had lung metastasis.

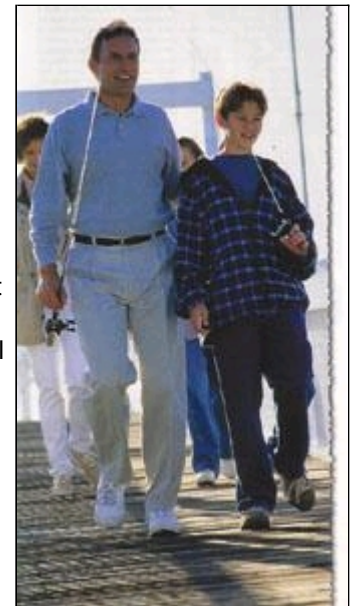
Finally, measurements of serum levels of ICAM-1 (a good indicator of tumor burden) showed significantly higher levels in the control and linoleic acid-fed groups compared to the CLA-fed mice.

In a discussion of these results, the scientists pointed out that adding only 1% of CLA to the diet (dry weight) has been shown to produce dramatic anti-cancer results and that adding more CLA does not confer additional benefits. This confirmed previous studies showing 1% of CLA to be highly effective in cancer prevention and treatment.

What this means to people seeking to use CLA for cancer prevention or therapy is that they may only need to take three 1000 mg capsules a day of 70% CLA to obtain the effects seen in the published studies. Assuming the average person eats 2.2 pounds (1,000,000 mg) of food a day, and further assuming that average diets are 80% water, that would leave 200,000 mg of dry weight food to measure the ideal percentage of CLA against. For most people, the amount of CLA needed to equate to 1% of their diet (dry weight) would be three 1000 mg capsules per day of 70% CLA.

The FDA has been studying CLA as a possible supplement or food additive for the purposes of preventing cancer. Indeed, the researchers who conducted this study concluded that, "there is the possibility that a CLA-enriched food product may be used in the chemo-prevention of prostatic cancer."

Foundation members have not had to wait for the bureaucratic FDA, as they have had access to low-cost CLA capsules since May 1996. Based on the published studies, it appears that the ideal dose of CLA for cancer prevention or treatment is three 1000 mg capsules of CLA per day. If you eat more than 2.2 pounds of food a day, then one or more additional capsules of CLA would be needed. CLA may thus represent one of the more practical and affordable ways of using a safe dietary supplement as a cancer preventive and as an adjuvant cancer treatment.



Modified citrus pectin slows PSA doubling time

The results of a pilot clinical trial were presented at the International Conference on Diet and Prevention of Cancer (May 28- June 2, 1999) in Tampere, Finland. Modified citrus pectin was shown to slow the PSA doubling time in prostate cancer patients with low levels of PSA. The doctors stated that more research involving larger numbers of patients is needed to fully define the role of modified citrus pectin in prostate cancer treatment. The doctors noted, "All study participants are still alive and evaluable for long-term followup almost three years after completion of this study." This pilot study was conducted by Stephen Strum MD, Mark Scholz MD, Jon McDermed Pharm D, Michael McCulloch BA and Isaac Eliaz MD.

Plant fiber derived from citrus fruit contains "citrus pectin" that interferes with cancer cell and cancer cell-metastatic target site interactions. For prostate cancer to spread, research indicates that a clump of cells may be required rather than a single cell or a few individual cells together.

In a study by Pienta et al, two groups of rats serving as an animal model for human prostate cancer were injected with a particularly virulent form of prostate adenocarcinoma cells that grow more rapidly and have a greater tendency to metastasize throughout the body. Half of the rats were "controls" and were given plain drinking water, while the others were given modified citrus pectin in their water at a 0.1% concentration. Results from this study showed that 15 out of 16 of control animals developed lung metastases, compared to only 7 out of 14 in the group receiving modified citrus pectin. The number of metastatic colonies in the lungs of the modified citrus pectin group was significantly lower than in the control group.

The same authors later showed that modified citrus pectin also reduced the ability of rat prostate cancer cells to bind to rat endothelial cells (the type of cells that form the inside lining of blood vessels) in a dose dependent manner. In other words, with higher doses of modified citrus pectin, proportionately greater reductions in cancer cell binding to endothelial cells were observed. (Pienta KJ et al, *J Natl Cancer Inst.* 87:348-353,1995).

The Life Extension Foundation introduced modified citrus pectin in 1995-now of the brand Pecta-Sol-as an adjuvant prostate cancer therapy. The dose used in the most recent human study was 15 grams a day. Based on this new study, prostate cancer patients may consider incorporating modified citrus pectin into their daily regimen.

Fathers with early prostate cancer pass on the risk

Previous studies suggest that prostate cancer is an inherited disease. In a study published in the journal *Cancer* (1999;86:477-483), men from families with a history of the disease, especially if a close family member was diagnosed before age 70, were at a significantly higher risk.

A team of researchers from Umea University in Sweden obtained the medical histories of 5,595 men whose fathers had been diagnosed with prostate cancer in Sweden between 1959 and 1963. The researchers found that the younger a father was when his prostate cancer was diagnosed, the greater the chance of the son developing it by age 70. Among sons whose fathers were diagnosed before age 70, the risk of prostate cancer at age 70 was nearly 9% compared with about 3% among members of the general population-more than 2.5 times greater. When fathers were diagnosed during their 70s, their sons' risk was less than twice that of the general population. Among sons of fathers diagnosed after age 80, the risk was only 1.5 times greater.

Risk of prostate cancer among sons rose to 3 times that of the Swedish population when at least two close family members had the disease, and if one of these relatives had been diagnosed before age 70, the risk to the son of being diagnosed by age 70 was 43%-14 times that of the general population.

Because early prostate cancer can be treated easily, the researchers recommend that men in this high-risk group "undergo annual prostate specific antigen (PSA) screening and prostate examination between the ages of 50 and 70."

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