

We can learn something from the Greeks and Italians, as well as from the Israelis. For example, when it comes to cancer prevention, it isn't necessarily how much fat you consume but the type of fat that may be most important.

The Mediterranean diet, traditionally consumed in Greece and regions of Italy, emphasizes whole grains, fruits, vegetables, seafood, and olive and walnut oils with limited meat and dairy. It is not a particularly low-fat diet, however. In fact, the average daily intake of overall fat for Greek women is forty percent of total calories, a figure roughly equivalent to the American diet. Yet, Greek women have much lower breast cancer rates than their American counterparts. Together with a higher intake of vegetables, whole grains, and fruits, a high intake of neutral or beneficial fats appears to be protective, observes researcher Emanuela Taioli.

Similarly, the low breast cancer rates in southern Italy are thought to be due to diets that avoid dangerous saturated fats. Whereas rates are higher in the north where French cooking, rich in butterfat, predominates. The absence of margarine, the source for trans-fatty acids which are another type of dangerous fat, from the southern diet is also important. Japanese researchers have found a high incidence of breast cancers in rodents fed a diet high in margarine.

Meanwhile, Israel has one of the highest intakes of polyunsaturated and saturated fats in the world. The consumption of omega-6 polyunsaturated fatty acids, which are found in safflower, corn and other highly processed commercial cooking oils, is about 8% higher than in the United States and 10 to 12% higher than in most European countries.

In fact, Israeli Jews may be regarded

Pancreatic, Colon & Breast Cancer Risk Reduced by Flax & Seafood



as a population-based dietary experiment of the effect of a high omega-6 and saturated fat diet. Not surprisingly, there is an extremely high prevalence of cardiovascular diseases, hypertension, non-insulin-dependent diabetes mellitus and obesity among Israeli Jews. There is also an increased cancer incidence and mortality rate, especially in women, compared with western countries. Studies suggest that high omega-6 fatty acid consumption might be the cause.

Profound Health Implications

We must take these findings very seriously if we are to maintain great health. For American women and men, emphasizing the good fats such as those from seafood (e.g., **salmon, tuna, and trout**), **flax** and **walnut oils**, is critical for maintaining healthy cell growth and reproduction.

Each of these sources is rich in fats called **omega-3 fatty acids**, which have shown suggestive evidence that they can reduce our risk for several types of cancers.

Flax Gains Respect as Cancer Protector

In particular, flax holds a special role for the prevention of pancreatic, colon,

and breast cancers, as well as prevention of the spread of melanoma.

Use of flax as a cancer prophylactic is "an area that I think has a lot of promise," notes Lilian U. Thompson, Ph.D., of the University of Toronto, one of a handful of researchers investigating the relationship between flax and cancer inhibition.

One of the keys to flax's anti-cancer properties is that it is rich in compounds known as lignan precursors. These are converted by flora in the colon to the lignans enterolactone and enterodiol. Lignans are one of the important phytochemicals that people can consume daily to minimize their risk of cancer and, also, if they do get cancer, to minimize its spread (metastases).

Thompson and her colleagues began their scientific quest into the powers of lignans by seeking the richest source of their precursors, screening for the production of enterolactone and enterodiol from about 70 common foods. Finding that flaxseed produced 75 to 800 times more of these substances than any other foods led them to their intensive study of flax.

Breast Cancer Prevention & Treatment

Today, Thompson is one of the world's leading authorities on flax's human health benefits, especially in the area of its use as part of cancer prevention and treatment.

In one of her early studies, Dr. Thompson already knew flaxseed lignans had been shown to be protective at the early promotional stage when cancers have not quite formed. Now she wanted to determine whether supplementation with flaxseed, beginning 13 weeks after carcinogen administration, would reduce the size of already established mammary tumors present at the start of treatment, as well as appearance of new tumors. **After seven weeks of treat-**

ment, established tumor volume was over 50% smaller in all treatment groups while there was no change in the placebo group. The correlation between established tumor volume and urinary lignan excretion "indicates that the reduction in tumor size is due in part to the lignans derived from . . . flaxseed."

A woman's cumulative exposure to estrogen, including the length of her estrous cycle, plays an important role in her lifetime breast cancer risk; the more estrogen to which her tissues are exposed, the greater her risk. Because flax lignans are weakly estrogenic, it has been thought that they might displace from the receptors of breast cells more toxic forms of estrogen that are likely to increase women's risk of cancer. In this sense, because they are weak estrogens, flax's lignans might have a beneficial anti-estrogenic effect much like the drug tamoxifen—but without its risks.

Thus, the antiestrogenic effects of flaxseed were compared with tamoxifen by monitoring estrous cycles. Four-week supplementation of a high-fat diet with flaxseed produced a dose-related cessation or lengthening of the cycle in about two-thirds of animals. With tamoxifen, 83% of the animals had irregular cycles. Thus, both compounds were antiestrogenic; however, flax performed its activities without tamoxifen's gross tissue toxicity (including uterine cancer risks).

Meanwhile, in another controlled experimental study, spontaneous breast cancers were significantly inhibited in the high alpha-linolenic acid group (flax is one of the richest sources of this fatty acid).

Comparative biological research also suggests an anti-breast cancer role for lignans. Researchers from the Department of Biological Sciences, Clark Atlanta University, Georgia, compared levels of urinary lignans among cancer-resistant primates with those of humans.

It was found that primates consuming their regular food excreted large amounts of the lignans, enterolactone and enterodiol. When fed a high fat diet, excretion levels were reduced by

more than 90% to a level observed in women with breast cancer.

Finally, we have convincing human evidence that high levels of flax's fatty acids in adipose breast tissue can prevent the emergence of aggressive metastases. In this study, published in the *British Journal of Cancer*, 121 women patients with an initially localized breast cancer were studied. Their adipose breast tissue was obtained at the time of initial surgery, and its fatty acid content analyzed. A low level of alpha-linolenic acid was strongly associated with the presence of vascular invasion, indicating the cancer was likely to spread. After an average 31 months of follow-up, 21 patients developed metastases. Large tumor size, high cell-division rates, presence of vascular invasion **and low levels of alpha-linolenic acid** were single factors significantly associated with an increased risk of metastasis.

Melanoma Prevention & Treatment

Researchers from the Department of Biomedical Sciences, Creighton University School of Medicine, Omaha, Nebraska, investigated the effect of dietary supplementation of flaxseed, the richest source of lignans, on experimental melanoma cells. **Flax reduced tumor occurrence by up to 63%**. The addition of flaxseed to the diet also caused a dose-dependent decrease in tumor area and volume, implying that it could be beneficial both in prevention and treatment.

"These results provide the first experimental evidence that flaxseed reduces metastasis and inhibits the growth of the metastatic secondary tumors in animals. It is concluded that flaxseed may be a useful nutritional adjuvant to prevent metastasis in cancer patients."

Colon Cancer

Most recently, Thompson and other researchers from the Department of Nutritional Sciences, University of Toronto, Ontario, Canada, found that lignans significantly reduced the proliferation of four different types of human colon tumor cell lines, even if

they were incubated with various levels of cancer promoters.

Pancreatic Cancer

Because a number of polyunsaturated fatty acids have been shown to inhibit the growth of malignant cells in the test tube, researchers investigated whether fatty acids modify the growth of human pancreatic cancer. To do so, they studied alpha-linolenic, gamma-linolenic, docosahexaenoic and eicosapentaenoic (EPA) acids and the effect of each fatty acid on cancer cell growth.

Each had an inhibitory effect, with EPA (found in seafood and formed from alpha-linolenic acid in flax) being the most potent.

"The ability of certain fatty acids to inhibit significantly the growth of three human pancreatic cancer cell lines *in vitro* at concentrations which could be achieved *in vivo* suggests that administration of such fatty acids may be of therapeutic benefit in patients with pancreatic cancer."

Varied Sources Best

It is probably wise to combine omega-3 fatty acids from various sources. A diet rich in seafood such as salmon, tuna, trout and sardines, combined with the use of lignan-rich flax (e.g., **Barlean's Lignan-Rich Flax Oil**), is a great approach. There are many ways to put more flax into your diet. Use on bread, mixed with yogurt, as a salad dressing and in many other recipes not involving heat (which destroys the beneficial compounds in flax). The reason we have recommended **Barlean's Lignan-Rich Flax** is in its name: it's rich in what we call lignan precursors, which we now know are powerful anti-cancer agents. Visit our website at freedompressonline.com for a variety of flax-based recipes. ♦

REFERENCES

- Bougnoux, P., et al. *Br J Cancer*. 1994; 70: 330-334.
 Falconer, J.S., et al. *Br J Cancer*. 1994; 69:826-832.
 Ingram, D., et al. *Lancet*, 1997; 350(9083): 990-994.
 Jenab, M. & Thompson, L.U. *Carcinogenesis*. 1996;17: 1343-1348.
 Kamano, K., et al. *Anticancer Res*. 1989; 9: 1903-1908.
 Musey, P.I., et al. *Life Sci*, 1995; 57(7): 655-664.
 Orcheson, L.J., et al. *Cancer Lett*, 1998; 125(1-2): 69-76.
 Sung, M.K., et al. *Anticancer Res*, 1998; 18(3A): 1405-1408.
 Taioli, E., et al. *Nutrition and Cancer*, 1991; 16: 259-265.
 Thompson, L.U., et al. *Carcinogenesis*, 1996; 17(6): 1373-1376.
 Trichopoulou, A., et al. *Journal of the National Cancer Institute*, 1995; 87(2): 110-116.
 Yam, D., et al. *Israeli J Med Sci*.1996; 32: 1134-1143.
 Yan, L., et al. *Cancer Lett*, 1998; 124(2): 181-186.
 Yanagi, S., et al. *Oncology*, 1993; 50(4): 201-204.