

Rachel's Environment & Health News

#630 - The News of 1998 -- Part 1

December 23, 1998

It's time for our 1998 wrap-up.

Evidence continued to accumulate during 1998 that your diet can drastically alter your chances of getting heart disease and cancer, including breast cancer.

The good news is that eating monounsaturated fats (the kind found in olive oil, canola oil, and nuts) seems to have a protective effect against these major diseases.[1] The nuts highest in monounsaturated fats are hazelnuts, macadamias, pecans, almonds, pistachios, Brazil nuts, walnuts, and peanuts.

The bad news is that hydrogenated vegetable oil and partially hydrogenated vegetable oil can have major harmful effects, increasing your chances of heart attack and cancer, including breast cancer. It is the trans-fatty acids in hydrogenated vegetable oils that seem to be the culprits.[2]

Hydrogenated vegetable oils are mainly found in margarine and vegetable shortening, which in turn are common ingredients of bread, cookies, crackers, chips, candy bars, and many baked goods such as doughnuts. Many french fries are now cooked in hydrogenated vegetable oils. If you eat a normal American diet, it is hard to avoid large doses of hydrogenated or partially-hydrogenated vegetable oils, but the evidence is mounting that they are really bad news and should be avoided whenever possible.

Alberto Ascherio at the Harvard School of Public Health estimates that trans-fatty acids are now killing at least 30,000 Americans every year. [3] Read the label and purchase wisely.

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Breast Cancer Prevention

During 1998, evidence continued to accumulate indicating that a significant portion of female breast cancer is preventable because it is caused by exposure to cancer-causing agents (chemicals and radiation -- including hydrogenated vegetable oils) added intentionally or unintentionally to the environment and food. (See REHW #571, #572, #573, #574, #575.)

About 182,000 new cases of breast cancer occur in American women each year, and 46,000 deaths occur annually from the disease. In the U.S., the occurrence of breast cancer has increased steadily at the rate of one percent each year for the past 40 years.

The "cancer establishment" -- the cluster of government agencies and private corporations that controls the flow of cancer research dollars (see REHW #571, #572) -- is feeling tremendous pressure to demonstrate a preventive approach to breast cancer.

Accordingly, the National Cancer Institute announced in April that a drug called tamoxifen had cut the occurrence of new breast cancers by 45% in a group of 13,388 women who were thought to have a high probability of getting the disease.[4] Government regulators acted swiftly and the news media trumpeted the story. A committee of the U.S. Food and Drug Administration (FDA) announced in September that it was recommending that the FDA approve tamoxifen as a drug for "reducing the risk" of breast cancer. A spokesperson for the FDA told the NEW YORK TIMES that "potentially tens of millions of women" could be candidates for tamoxifen treatments at a cost of \$80 to \$100 per month per person. Tamoxifen is marketed under the name Nolvadex by Zeneca, the chemical company that sponsors Breast Cancer Awareness Month each year. Tamoxifen has been used for breast cancer chemotherapy for two decades.

The FDA committee carefully avoided using the words "prevent" or "prevention" because it said tamoxifen may merely delay the onset of cancers and not actually prevent them; it is too early to tell. Still, the message from the cancer establishment was unmistakably one of

prevention. The NEW YORK TIMES ran a front-page story saying tamoxifen's approval by FDA "would be a milestone in efforts to prevent cancer." [5] Even before the tamoxifen study was published, the TIMES wrote an editorial about it, calling tamoxifen "a breast cancer breakthrough." "For the first time, scientists have demonstrated that breast cancer can not only be treated but actually prevented," the TIMES editorial said. [6]

Unfortunately, it is not clear that tamoxifen represents a real victory for most women. The TIMES acknowledged in its editorial that, if 1000 women took tamoxifen for 5 years, 17 breast cancers would be avoided, and bone fractures from osteoporosis would be reduced; however in the same 1000 women during the 5 years tamoxifen would cause an additional 12 endometrial cancers (cancers of the lining of the uterus) and at least 10 potentially-fatal blood clots. The published study also reported an increase in strokes and eye cataracts among those treated with tamoxifen, compared to a control group.

In its news story, the TIMES reported that the FDA committee "said it did not yet have enough information to determine which women were at high enough risk of breast cancer to make the drug's hazards, including potentially fatal blood clots as well as cancer of the uterine lining, worth its benefits."

The National Cancer Institute has released a computer "risk disk," a diskette containing a program intended to help women judge their risk of getting breast cancer. The diskette is available in both PC and Macintosh formats; telephone 1-800-4-CANCER or sign up to receive the diskette by mail at <http://cancertrials.nci.nih.gov>.

Of course, no one should rely on a computer program -- or on information they read in the news media -- to make decisions about their health without consulting a qualified medical specialist.

Two smaller studies of tamoxifen and breast cancer were published in September and neither of them showed any benefits from tamoxifen treatments. [7,8,9] Differences in criteria for recruiting women into the studies may have produced the contradictory results. Nevertheless, definitive evidence of tamoxifen's benefits and dangers must await further study.

In late April, the NEW YORK TIMES reported on two unpublished studies of a drug called raloxifene. According to the TIMES, both studies show that raloxifene can reduce a woman's chances of getting breast cancer without increasing her chances of getting endometrial cancer. A study is now under way to compare the effects of raloxifene vs. tamoxifen. [10]

To us, the tamoxifen and raloxifene studies reveal a curious shift in the cancer establishment's view of "prevention." To most people, cancer prevention means preventing exposures to cancer-causing agents. Instead cancer "prevention" is coming to mean treating a woman with a potent drug year after year, in an attempt to counteract the effects of her lifelong exposure to carcinogens. The eagerness of the NEW YORK TIMES to promote this new view of prevention on page 1, and in its editorial columns (often relying on preliminary data from unpublished studies), is, itself, curious and worrisome. It is as if the cancer establishment has abandoned the struggle to get carcinogens out of the environment and the nation's food supply, relying instead on drug treatments. It occurs to us that there is simply no money to be made in old-style prevention. It is hard to make a living by reducing women's exposures to radiation and carcinogenic chemicals. But getting FDA approval for a new drug can be extremely lucrative even if its benefits hardly outweigh its dangers.

To us, the most interesting study of 1998 was never reported in the NEW YORK TIMES or any other of the mass media. In September, researchers at the University of Birmingham in England reported exposing pregnant rats to small amounts of dioxin on the 15th day of pregnancy. [11] [Dioxin is a highly-toxic, chlorinated byproduct

of combustion, incineration, metal smelting, and the manufacture of many chemicals, including pesticides. All Americans carry amounts of dioxin in their bodies that the U.S. Environmental Protection Agency considers dangerous. (See REHW #390, #391.)

The female offspring of the dioxin-exposed pregnant rats were born normal, but by the time they were 7 weeks old, their mammary glands had developed an unusually high number of "terminal end buds" -- the places in a breast where breast cancers develop. Four studies have shown that there is a direct correlation between the number of terminal ends buds in a breast and its susceptibility to breast cancer.

The Birmingham researchers went on to expose these young rats (and a control group) to a well-known carcinogenic chemical (dimethylbenz[a] anthracene). Sure enough, the dioxin-exposed young rats developed many more breast cancers than did the control group.

This elegant study shows that (a) timing of exposure to dioxin (and presumably to other toxicants) is critical; (b) exposure to a chemical before birth can predispose an animal to breast cancer later in life even if the chemical itself is known to INHIBIT breast cancer when exposure occurs later in life, as is the case with dioxin; (c) present methods of testing chemicals for their cancer potential are missing the boat, failing to ask the right questions about the dangers of the cancer-causing chemicals we are all legally exposed to year after year.

There are important opportunities to prevent breast cancers, and other cancers, in this world, and they do not require us to expose tens of millions of women to powerful chemotherapy drugs year after year. They simply require us to develop the political will to clamp down on the murderous practices of industrial polluters and the food industry.

-Peter Montague (National Writers Union, UAW Local 1981/AFL-CIO)

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[1] On the protective effect of nuts, see Janet Raloff, "High-Fat and Healthful," SCIENCE NEWS Vol. 154 (November 21, 1998), pgs. 328-330. On the protective effect of olive oil, see J.M. Martin-Moreno and others, "Dietary fat, olive oil intake, and breast cancer risk," INTERNATIONAL JOURNAL OF CANCER Vol. 58, No. 8 (September 15, 1998), pgs. 774-780, and: N.R. Somonsen and others, "Tissue stores of individual monounsaturated fatty acids and breast cancer: the EURAMIC study. European Community Multicenter Study of Antioxidants, Myocardial Infarction, and Breast Cancer," AMERICAN JOURNAL OF CLINICAL NUTRITION Vol. 68, No. 1 (July 1998), pgs. 134-141. On the protective effect of monounsaturated fats in general, see A. Wolk and others, "A prospective study of association of monounsaturated fat and other types of fat with risk of breast cancer," ARCHIVES OF INTERNAL MEDICINE Vol. 158, No. 1 (January 12, 1998), pgs. 41-45.

[2] Alberto Ascherio and others, "TRANS-Fatty Acids Intake and Risk of Myocardial Infarction," CIRCULATION Vol. 89, No. 1 (January 1994), pgs. 94-101. And P. Pietinen and others, "Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men. The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study," AMERICAN JOURNAL OF EPIDEMIOLOGY Vol. 145, No. 10 (May 15, 1997), pgs. 876-887.

[3] A. Ashcerio and W.C. Willett, "Health effects of trans fatty acids," AMERICAN JOURNAL OF CLINICAL NUTRITION Vol. 66 [4 Supplement] (October 1997), pgs. 1006S-1010S.

[4] The study was published in September. See B. Fisher and others, "Tamoxifen for Prevention of Breast Cancer: Report of the National Surgical Adjuvant Breast and Bowel Project P-1 Study," JOURNAL OF THE NATIONAL CANCER INSTITUTE Vol. 90, No. 18 (September 16, 1998), pgs. 1371-1388.

[5] Gina Kolata and Lawrence M. Fisher, "Drugs to Fight Breast Cancer Near Approval," NEW YORK TIMES September 3, 1998, pgs. A1, A18.

[6] "Breast Cancer Breakthrough [editorial]," NEW YORK TIMES April 8, 1998, pg. A24.

[7] Trevor Powles and others, "Interim analysis of the incidence of breast cancer in the Royal Marsden Hospital tamoxifen randomised chemoprevention trial," LANCET Vol. 352 (July 11, 1998), pgs. 98-101.

[8] U. Veronesi and others, "Prevention of breast cancer with tamoxifen: preliminary findings from the Italian randomised trial among hysterectomised women," LANCET Vol. 352 (July 11, 1998), pgs. 93-97.

[9] Kathleen I. Pritchard, "Is tamoxifen effective in prevention of breast cancer?" LANCET Vol. 352 (July 11, 1998), pgs. 80-81.

[10] Lawrence K. Altman, "Studies Show Another Drug Can Prevent Breast Cancer," NEW YORK TIMES April 21, 1998, pg. A16.

[11] Nadine M. Brown and others, "Prenatal TCDD and predisposition to mammary cancer in rats," CARCINOGENESIS Vol. 19, No. 9 (1998), pgs. 1623-1629.

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