Women and Cancer: Opportunities for Prevention

PHYSICIANS COMMITTEE FOR RESPONSIBLE MEDICINE

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Ask any doctor what women can do to prevent breast cancer, and the response will probably be to get an annual mammogram after age 50, or perhaps after age 40. Mammograms certainly are important. But they do not prevent cancer. They *find* cancer. Biopsy, surgery, or chemotherapy then follow.

What is largely unknown to the American public—and sadly underemphasized in medical schools—is that breast cancer is often a preventable illness. When I was a medical student, I was not taught that breast cancer had any relationship to dietary factors. At that time, breast cancer attacked 1 in every 11 women. When I was a resident in the early 1980s, most doctors remained ignorant of any risk factors that could be controlled, and the rate went up to one in ten. The failure to prevent cancer has exacted an increasing toll; today, the disease attacks one woman in eight.

It is not that scientists do not have the information. As long ago as 1982, the National Research Council published a report called *Diet, Nutrition, and Cancer*,¹ showing the mountain of evidence already available linking specific dietary factors to cancer of the breast and other organs. But brochures with watered-down recommendations have sat collecting dust at cancer research centers. There was never an organized effort to give women the information they need to make decisions about cancer prevention.

The dietary factors emerged in comparisons of different countries. In Japan, for example, breast cancer is rare. But Japanese women who move to the United States soon have the same risk of cancer as American women—at least 400 percent higher than in Japan. The differences in cancer risk between the U.S. and Japan are not due to genetics. Nor is it something in the air or water. The critical factor is the amount of fat, particularly animal fat, in the diet.^{2,3} In Japan, only about 15 percent of the calories in the diet come from fat. In the United States, the fat content of the diet has been more than two times more, around 35 percent. The more fat women consume, the greater their cancer risk. Similar findings have been made within countries.

When the link between fat and cancer was found,

researchers did not have to look far for reasons to explain it. Several possibilities presented themselves. First of all, it is known that many breast tumors are "fueled" by estrogens, the female sex hormones for both women and men. But the more estrogen there is, the greater the driving force behind some kinds of breast cancer. The principal estrogen is *estradiol*, and the amount of estradiol produced by the body is linked to the amount of fat in the diet. On high-fat diets, estradiol production increases. On low-fat diets, it decreases. When women begin low-fat diets, their estradiol levels drop noticeably in a very short time. Vegans (people who consume no animal products) have significantly lower estrogen levels than non-vegetarians, perhaps because of the lower fat content of the vegan diet.

In addition, estradiol is carried in the blood on special carrier molecules. On high-fat diets, more estradiol breaks free from its carrier molecules and becomes biologically active, like soldiers jumping off a jeep and starting their attack. So high-fat diets may promote cancer by increasing the amount in addition to the biological activity of estradiol in the body.

Another problem with high-fat diets is that the meat, poultry, fish, and dairy products that usually make up such diets are devoid of fiber. Fiber is the part of plant foods that resists digestion in the intestinal tract. Evidence suggests that fiber helps reduce estrogen levels by trapping it in the digestive tract. In addition, soybeans, which are a mainstay of Asian diets, contain *phytoestrogens*, which are very weak estrogens which can compete with and blunt the effect of normal estrogens. In the process, soybean products have been shown to reduce cancer risk.

So high-fat diets increase estrogen production, apparently increasing cancer risk. Low-fat, high-fiber diets reduce estrogens to a more biologically normal level. Since meats, poultry, fish, and dairy products contain no fiber, they increase the fat content and reduce the fiber content of the diet by displacing plant foods.

Since most Americans are on high-fat diets, one might ask if the resultant elevation of estrogen has other effects. The answer is almost certainly yes. During my medical education, I worked for a time at an inner-city clinic in Washington, D.C. There, girls of 12 and 13 would come in asking

for birth control pills. Many had already had their first child and did not want to become pregnant again. I wondered why nature designed the human body to become sexually mature at an age when girls are not old enough to care for a child or even to sustain a long-term relationship. It appears that nature is not to blame. In fact, evidence suggests that the boy is designed to reach puberty a bit later.

According to the World Health Organization, the average age of puberty in girls in western countries in 1840 was about 17 years of age. Today we take it as a matter of course that girls will reach puberty at 11, 12, or 13. One-hundred-fifty years ago, high-fat diets were limited to a small, wealthy portion of the population. Today, high-fat diets have spread to the entire population, and puberty has occurred earlier and earlier, possibly due to the estrogen increase caused by high-fat diets. Early puberty has been associated with increased risk of breast cancer. A comparison of different countries lends further support to this theory. In China, low-fat diets are still the rule. There, the age of puberty ranges between 15 and 19. Dr. T. Colin Campbell of Cornell University has studied the Chinese diet, which is centered on rice and vegetables, with little meat and no dairy products. The apparent effect is not just a higher age of puberty, but also phenomenally low rates of heart disease, obesity, and cancer.

High-fat diets may also encourage the absorption of carcinogens into the body. Researchers have observed, for example, that when the carcinogens in cigarette smoke are absorbed through the lung tissue, they tend to travel along with fats in the blood. It may be that on a low-fat diet, the body is less able to absorb and transport carcinogens.

Evidence suggests that other factors also play important roles. The mineral selenium, found in grains, helps prevent cancer, as does physical exercise and avoiding alcohol.

We can dramatically reduce the toll, not only of breast cancer, but of other cancers as well. As everyone now knows, a low-fat, high-fiber, plant-based diet helps protect against colon cancer.

And there is more: we now have clues to preventing cancer of the ovary. A complex and fascinating study by Dr. Daniel Cramer of Harvard University elucidated the relationship between cancer and diet. Dr. Cramer studied hundreds of women with ovarian cancer, and had them record in detail what they normally ate. He compared them to a group of women who were similar in age and other demographic variables, but who did not develop cancer. There was one thing that the women with cancer had eaten much more frequently than women without cancer: dairy products, especially the supposedly "healthy" products, such as yogurt.

The culprit may be a normal breakdown product of the milk sugar *lactose*. Lactose is broken down in the body to another sugar called *galactose*. In turn, galactose is broken down further by enzymes in the body. According to Dr. Cramer, when dairy product consumption exceeds the enzymes' capacity to break down galactose, there is a build-up of galactose in the blood, which may damage a woman's ovaries. Some women have particularly low levels of these enzymes, and when they consume dairy products on a regular basis, their risk of

ovarian cancer can be triple that of other women. The problem is the milk sugar, not the milk fat, so it is not solved by using non-fat products. In fact, yogurt and cottage cheese seem to be of most concern because the bacteria used in their production increase the production of galactose from lactose.

There is a great deal of evidence that dietary factors can help prevent cancer. But what about improving survival for those who have cancer? The evidence is not all in, but there is reason to believe that foods can have an important effect here, too. The immune system is our line of defense against both initial cancers and the spread of cancer. Substantial evidence shows that certain foods can bolster immune function, while others impair it. For example, natural killer cells are specialized white blood cells that seek out and destroy cancer cells. A recent German study showed that vegetarians have more than twice the natural killer cell activity of non-vegetarians. 9 It is not yet known whether the immune strength of vegetarians comes from their having double the number of natural killer cells or from each cell having double the killing power. Whichever it is, vegetarians have a defense against cancer cells that is far beyond that of their meat-eating fellows. The immune strength of a vegetarian diet probably comes from its low fat content and from vitamin-rich vegetables and fruits.

Low-fat diets strengthen the immune defenses against cancer cells. Researchers in New York tested the effect of low-fat diets on immunity. They put healthy volunteers on a diet which cut the fat content to 20 percent. Three months later, the researchers took blood samples from the volunteers and examined their natural killer cells. As in the German study, the natural killer cell activity was greatly increased, although not as much as on the vegetarian diet used by the German researchers. It appears that all fats and oils—animal or vegetable—can impair the immune system. 11,12 Even fish oils interfere with natural killer cells. 13,14

Certain vitamins can be immune boosters. Beta-carotene is naturally found in yellow and dark green vegetables. Several research studies at the National Cancer Institute have shown that those who consume generous amounts of beta-carotenerich vegetables cut their cancer risk substantially. Beta-carotene's power comes partly from its ability to neutralize free radicals, molecules which tend to form in the body and which can attack the cells and cause cancer. Vitamins C and E also have some of this ability. But beta-carotene also increases the number of natural killer cells and increases the number of another kind of white blood cell, called the T-helper cell, which helps direct the immune response. The minerals selenium, zinc, and iron are also important to immune function, although for zinc and iron, both too much and too little can spell problems.

A cancer-prevention diet has to be very different from even the diet recommended by the National Cancer Institute. NCI still recommends a 30-percent fat diet, in spite of strong evidence that a 30-percent fat limit is far too high. The Japanese and Chinese do not eat a 30-percent fat diet. Their diets have half the fat of the NCI-recommended diet. A study by Dr. Willett and his colleagues at Harvard showed that a diet drawing 30 percent of its calories from fat had no measurable effect on cancer incidence. ¹⁶ A cancer prevention diet should contain no more

than 10 to 15 percent fat and should be vegetarian.

All together, the new knowledge on prevention is powerful artillery in the war on cancer. According to the National Cancer Institute, as much as 80 percent of cancers can potentially be stopped before they ever start. Tobacco amounts for 30 percent of cancer cases. Dietary factors account for even more, from 35 to 50 percent. As the consumption of meat, dairy products, and fried foods has become a daily routine, the female body has been assaulted by altered hormonal function, an unnatural age of puberty, and a much greater risk of cancer. By eliminating unhealthful dietary factors and encouraging the diets that diminish risk, we can hope to turn the tide on this epidemic.

References

- National Research Council. Diet, Nutrition, and Cancer. Washington, D.C., 1982.
- Armstrong B, Doll R. Environmental factors and cancer incidence and mortality in different countries, with special reference to dietary practices. Int J Cancer 1975;15:617-31.
- 3. Hirayama T. Epidemiology of breast cancer with special reference to the role of diet. Prev Med 1978;7:173-95.
- Rose DP, Boyar AP, Cohen C, Strong LE. Effect of a low-fat diet on hormone levels in women with cystic breast disease. 1. Serum steroids and gonadotropins. J Natl Cancer Inst 1987;78(4):623-6.
- Ingram DM, Bennett FC, Willcox D, de Klerk N. Effect of low-fat diet on female sex hormone levels. J Natl Cancer Inst 1987;79(6):1225-9.

- Goldin BR, Gorbach SL. Effect of diet on the plasma levels, metabolism and excretion of estrogens. Am J Clin Nutr 1988;48:787-90.
- Kagawa Y. Impact of westernization on the nutrition of Japanese; changes in physique, cancer, longevity and centenarians. Prev Med 1978;7:205-17.
- Cramer DW, Harlow BL, Willett WC, et al. Galactose consumption and metabolism in relation to the risk of ovarian cancer. Lancet 1989:2:66-71.
- Malter M, Schriever G, Eilber U. Natural killer cells, vitamins, and other blood components of vegetarian and omnivorous men. Nutr Cancer 1989;12:271-8.
- Barone J, Hebert JR, Reddy MM. Dietary fat and natural killer cell activity. Am J Clin Nutr 1989;50:861-7.
- Nordenstrom J, Jarstrand C, Wiernik A. Decreased chemotactic and random migration of leukocytes during Intralipid infusion. Am J Clin Nutr 1979;32:2416-22.
- Hawley HP, Gordon GB. The effects of long chain free fatty acids on human neutrophil function and structure. Lab Invest 1976;34:216-22.
- Kelley DS, Branch LB, Love JE, Taylor PC, Rivera YM, Iacono JM. Dietary alpha-linoleic acid and immunocompetence in humans. Am J Clin Nutr 1991;53:40-6.
- 14. Endres S, Ghorbani R, Kelley VE, et al. The effect of dietary supplementation with n-3 polyunsaturated fatty acids on the synthesis of interleukin-1 and tumor necrosis factor by mononuclear cell. N Engl J Med 1989;320:265-71.
- Watson RR, Prabhala RH, Plezia PM, Alberts DS. Effect of beta-carotene on lymphocyte subpopulations in elderly humans; evidence for a dose-response relationship. Am J Clin Nutr 1991;53:90-4.
- Willet WC, Stampfer MJ, Colditz GA, Rosner BA, Hennekens CH, Speizer FE. Dietary fat and risk of breast cancer. N Engl J Med 1987;316:22-8.