Nutrition *News and Views*

September/October 2012 **Vol.16, No.5** **For health professionals**

PREVENTING BREAST CANCER – PART 1

by Judith A DeCava, CNC, LNC

Breast cancer is the second leading cause of cancer deaths in US women today. A woman's lifetime risk has nearly tripled during the past 4 decades. Every year now about 1.3 million women will be diagnosed with breast cancer. Prevention is a high priority. But how? Many conflicting reports plus long-held 'facts' that turn out to be erroneous cause dismay and confusion. Yet some evidence holds true, more is being learned and the potential for prevention is realistic. For example, it's known that breast cancer incidence varies greatly from country to country. Within the US it varies from area to area. There have to be factors that account for these differences.

We often hear that 1 in 8 women will develop breast cancer, a misleading way to put it. Actually, women who live to age 90 have a 1-in-8 **lifetime** risk of being diagnosed with breast cancer. Although only about half of all women live that long, 1 in 8 will have developed breast cancer at some point in her life. The **risk** of developing breast cancer starts low in young women and grows with age. At age 30, a woman has a 0.4% change of developing breast cancer in the next 10 years (4 out of 1,000 women); at age 40 a 1.5% chance, at age 50 a 2.7% chance, at age 60 a 3.8% chance and at age 70 a 4.1% chance. Family history is a factor and genes have been accused. Early onset of menstruation (before age 12), late menopause, having no children or waiting to have children after age 30, and never breastfeeding have been blamed. Dense breast tissue is on the list—the fewer fat cells in the breast, the denser the tissue. Age, health history, and lifetime accumulation of estrogen have been considerations. But there are no absolute answers. So far radiation is the only proven cause of breast cancer. Environmental toxins, hormone drugs and certain other drugs are certain culprits. More men are developing breast cancer (1 man for every 100 women). Being a woman is not a prerequisite. [1]

Breast cancer is classified into **two** types. 1) **Noninvasive** (in situ) breast cancer, meaning cancer cells have not spread to adjacent areas of the breast; they remain in their place of origin. The most common type is ductal carcinoma in situ (**DCIS**) which occurs in the lining of the milk ducts. Noninvasive breast cancer may be called 'stage 0' cancer. 2) **Invasive** breast cancer, meaning cancer cells infiltrate or spread outside the membrane that lines a duct or lobule into surrounding tissues. Cancer may then affect other parts of the body. Invasive breast cancer can be stage 1, 2, 3, or 4, depending on how advanced it is. Breast cancer can be **further** classified according to what type of tissue it arises from. 1) Milk **ducts**. Ductal carcinoma is the most common type of breast cancer. 2) Milk-producing **lobules**. Lobular carcinoma originates in the lobules, subdivisions of mammary glands where breast milk is produced. 3) Connective **tissues** (muscles, fat and blood vessels). Rarely, breast cancer can originate from these breast tissues; in this case, it's called sarcoma.

Inflammatory breast cancer (IBC) is rare, of sudden onset (weeks to months) and can be confused with mastitis. The affected area is red, swollen, warm and tender because cancer cells have blocked the lymphatic vessels in the breast. There may or may not be a lump. IBC accounts for only 1-5% of all breast cancer cases in the US and is more common among younger women and African American women; it infrequently occurs in men. IBC is the most aggressive form of breast cancer with survival rates lower than for other types. Lobular carcinoma in situ (**LCIS**) was considered an early stage of breast cancer. But it's now known that LCIS may just indicate a risk for developing breast cancer. Doctors are more likely to take a wait-and-see approach.

DCIS, the most common form of breast cancer (previously considered pre-cancerous), is the type especially on the increase, possibly because it's found more often due to expanded screening. DCIS is contained in the milk ducts, hasn't spread out to the fatty breast tissue or any other part of the body. It's not large enough to be palpable, but is only picked up on mammograms where it shows up as little specks of calcium (micro-calcification). Then it's usually confirmed by biopsy. Frequently at the center of these microcalcifications is a 'foci' of a cancer that is basically debilitated or defunct. All that's left is a remnant which the body has effectively contained—an abnormal milk duct or a calcified mass surrounded by fibroid tissue. Before prevalent mammography screening, this diagnosis represented less than 5% of all new cases of breast cancer. Now between 30-50% of new breast cancer diagnoses obtained through mammography are classified as DCIS. Since most cases are treated with either breast removal or radiation, it's not known how many would have

remained dormant without treatment. Mastectomies are often performed as 'just-in-case' measures. There is evidence that these little abnormalities are something most women's bodies are able to extinguish themselves. A noninvasive cancer like DCIS does not necessarily have to be treated with surgery, radiation, and chemotherapy. Women can choose watchful waiting. Further, they can improve their lifestyle (nutrition, physical activity, lower exposure to toxins). How many claims of cancer 'cures' by surgery, radiation, or chemo involve DCIS—a condition which may have regressed on its own and was already contained? [2]

CAUSES. A number of risk factors can contribute to breast cancer. The more risk factors involved, the more the chance of developing breast cancer. The more we know, the better we can plan strategies for preventing it.

Many studies have concluded that breast cancer is tied to **estrogen** exposure. The body produces three major types of estrogen—estriol, estrone and estradiol. Estrogen travels though the bloodstream largely as estradiol. Women with higher circulating estradiol levels appear to have a higher risk of breast cancer. Some breast tumors are believed to be estrogen-responsive—that estradiol increases growth rates in breast cells already cancerous. But isn't a woman's body **supposed** to produce estradiol, an important, needed female hormone? Why would it stimulate breast cancer? One reason given is cumulative exposure—starting menstruation at an earlier age, not getting pregnant or not breastfeeding (times when minimal estradiol is produced), having short and more frequent menstrual cycles, experiencing menopause past age 55. Yet most women with these experiences don't get breast cancer. Various estrone and estradiol metabolites have been shown to **either** inhibit cellular proliferation or enhance proliferation—in the laboratory. There are no definite answers.

There is some evidence indicating that one aspect is the **liver**'s inability to properly break down (metabolize) estradiol. That's why nutritional support (with cruciferous vegetables, for example) to the liver can enhance its detoxifying cytochrome 450 enzymes and lower risk. Nowadays the livers of most people are overburdened with toxins. If the liver cannot handle this onslaught, it cannot perform its other functions properly such as processing and breaking down hormones. Liver dysfunction can set in motion biochemical imbalances that tip the scales towards breast cancer. Second of all, estrogens and progesterone need to be **balanced**. Women with relatively low progesterone may be more prone to estrogen dominance, an imbalance that may contribute to breast cancer. Yet women with higher-than-normal progesterone levels can have an increased rate of breast cancer. Overweight can result in excess estrogen or excess progesterone. Normal healthy production of estrogens, progesterone and testosterone in proper amounts with proper balance does not cause cancer.

Lack of full development and differentiation of breast tissues makes cells more susceptible to damage from toxins, radiation and hormone imbalance. Full development occurs with a full-term pregnancy. Risk of breast cancer is about halved among women who have had at least one full-term pregnancy. Women who breastfeed their infants have a lower risk. According to one study, risk drops by 7% for each child a woman has and decreases by 4.3% for every year of breastfeeding. In another study, women with a history of breast cancer in their immediate families and who had breastfed had a 59% lower risk than women who had not breastfed.

Hormone **drugs** (hormone replacement therapy, contraceptives), hormone-mimicking **chemicals** and hormone disrupting chemicals may account for many cases of breast cancer. Hormone replacement therapy (**HRT**) consists of horse-urine or synthetic estrogens and synthetic progestins (progesterone). These are laboratory-produced hormones, foreign to the human body. Plus the dosage of estrogen is often greater than needed. Bodily-made progesterone tends to **protect** against breast cancer; synthetic progestins do **not** protect and may increase risk. "Estrogen-progestin use, both sequential and continuous, appears to be strongly associated with the risk of breast cancer compared with the use of estrogen alone." Short-term use of estrogen therapy by women who have had a hysterectomy appears to **not** increase the risk of breast cancer, but longer term use may increase risk. HRT increases breast density (linked to increased cancer risk) with initiation and decreases it with discontinuation. Numerous studies show an increased incidence of advanced or invasive breast cancer due to HRT. Some forms of **birth control**, also made of synthetic estrogens and progestins, may increase risk. Oral contraceptives have been implicated in several studies. Women using estrogen and testosterone drugs (for low libido) or long-term use of certain infertility drugs have an increased risk of invasive breast cancer. There is concern that prolonged intake of **DHEA** (dehydroepiandrosterone) as a drug could lead to breast cancer as it can be metabolized to estrogen. But some studies indicate that in women, DHEA increases serum

testosterone rather than estrogen. In men the tendency is to more estrogen than testosterone. Concern also exists because the DHEA drug can result in increased serum concentrations of free insulin-like growth factor-1 (IGF-1) which may stimulate promotion of breast cancer, particularly in postmenopausal or obese women. Elevated IGF-1 increases breast density (linked to breast cancer). Our bodies normally produce DHEA (an adrenal hormone), but taking DHEA as a drug may imbalance other hormones and biochemicals. Circulating levels of IGF-1 are higher in meat eaters, probably not due to eating meat per se, but eating meat containing hormone residues from animals raised on industrial farms. Synthetic hormones are routinely fed to livestock to increase fat content and hasten growth. There are at least 6 estrogen-like compounds approved to promote growth in farm animals and one for dairy cows to increase milk production. Residues are in the meat and milk.

Women who work predominantly at **night** have an increased risk of breast cancer. Prolonged exposure to light at night when melatonin levels are typically the highest is what increases the risk. Melatonin is secreted in response to darkness; it causes sleepiness but also affects many other parts of the body, including levels of other hormones. Insufficient melatonin production may be a contributor to the development of breast cancer. [3]

Overweight and inactivity are two predominant risk factors for breast cancer. Losing weight—and keeping it off—decreases risk. One study found that breast cancer risk rises 40% in adult women who gain 21 to 30 pounds after age 18. In another study, a comparison was made between women who gained only 20 pounds or less after age 18 with those who gained 60 pounds or more. The women gaining the higher amount of weight had an elevated risk for every breast tumor type, stage and grade; they were nearly twice as likely to have ductal type tumors and three times as likely to have cancer that spread beyond the breast. Risks for over-weight women are particularly elevated after menopause. Among other things, obese women tend to have much higher levels of insulin; their cells eventually become insulin resistant. Among participants in the Women's Health Initiative Observational Study, those with high insulin levels had nearly 2½ times the risk of breast cancer of those with low insulin levels. World Health Organization experts claim that weight **loss** and **exercise** could prevent 25-30% of breast cancer cases. Regular fitness activities reduce breast cancer risk for both pre- and post-menopausal women by 20-30% compared with inactive women. Even women at increased risk of breast cancer lower their risk substantially by regularly participating in physical activities. [4]

Women with certain **genetic** mutations are said to be at increased risk of developing breast (or ovarian) cancer. A number of genes (at least 12) have been blamed, but BRCA1 and BRCA2 are the two whose mutations are most implicated. BRCA stands for BReast CAncer. All women carry BRCA1 and BRCA2 genes, which are involved in normal cell growth. Only women who have **mutations** in these genes are believed to be at higher risk for breast or ovarian cancer. BRCA genes **without** mutations are thought to keep cancer at bay. It's estimated that only 1 in 300 to 500 women in the US carries the BRCA mutations. The mutations are found in less than 1-5% of women diagnosed with breast cancer. Such mutations are also thought to increase risk of prostate and breast cancer in men. Some BRCA mutations are more common in women of Ashkenzzi Jewish heritage. The US Preventive Services Task Force recommends AGAINST routine genetic screening. They say only about 2% of women have family history patterns that would warrant testing: a) 3 relatives with breast cancer. b) 2 relatives with breast cancer if at least 1 was diagnosed at age 50 or younger. c) Cancer diagnosed in both breasts. d) Ovarian cancer, especially more than 1 relative had it or there is also breast cancer in the family. e) Breast cancer in a male relative. By the way, genetic screening can miss mutations.

In reality there "exists broad variation in breast cancer risk among carriers of BRCA1 and BRCA2 mutations." Women with neither of these genetic mutations often have a family history of the disease. And 90% of breast cancers occur in women with no family history and no BRCA mutations. Variations occur in different racial and ethnic groups. Questions remain. For example, "Why do these mutations predominantly affect hormone-responsive tissues when the mutant gene is widely expressed throughout the body?" It has been suggested that signaling by progesterone may be involved, but so far no one knows for sure. Alterations in the balance of any number of hormones may affect risk—in carriers of BRCA mutations and those without mutations. It appears that genetic mutations may **not** be as clear a cause of breast cancer as originally thought. An article in *The Lancet* admits that the lifetime risk of developing breast cancer in individuals who have the BRCA1 and BRCA2 mutations "might have been exaggerated," the overestimation occurring "because a woman's risk is associated not only with the genetic mutations, but also with other risk factors."

Genetic mutations may be a predisposing condition, but not the cause. Your genes don't dictate your health; instead it's the **expression** of your genes. Your lifestyle and emotional state turn genes on and off. It's called epigenetics—changes in gene expression caused by mechanisms other than changes in the underlying DNA sequence. Having 'bad genes' doesn't necessarily mean an individual is doomed to something like cancer. Genes are not self-regulating. They're blueprints, largely activated and controlled by their environment including diet, physical activity, toxic exposures, thoughts, emotions, and more. These can create tens of thousands of different variations from a genetic blueprint, allowing for a huge amount of leeway in modifying the expression or 'read-out' of each gene. A study reported in *Genes & Nutrition* says that epigenetic regulation "constitutes an important mechanism by which dietary components can selectively activate or inactivate gene expression." Healthy lifestyle habits, including natural foods, regular physical activity, and maintenance of normal weight, "may help reduce DNA damage and enhance DNA repair." Various food enzymes prevent cancers in experimental animals with specific cancer genes. A number of nutrients (including Co-Q10 and B vitamins) increase the activity of a gene-repair enzyme in women with breast cancer. Cruciferous vegetables may ameliorate the effects of certain genotypes and reduce breast cancer risk. *UC Berkeley Wellness Letter* says that separating the effects of genes, environment, and lifestyle behaviors is difficult, if not impossible. A study in *Breast Cancer Research* analyzed data from more than 85,000 post-menopausal women. Women who did three things—maintained a healthy weight, exercised, and limited alcohol—had a 15-25% reduced risk of breast cancer over a 5-year period, compared to women who did none of them. Women with a **family** history, though still at higher risk, benefited as much as those with no family history. According to articles in *Science*, a healthy lifestyle is protective against breast cancer in women with BRCA1 or BRCA2 mutations. But BRCA mutation carriers who used oral contraceptives for at least 5 years, those who had used them before age 30 years, or those who first used them before 1975 (with a higher estrogen content) had a 29% to 42% increased risk of breast cancer compared with BRCA mutation carriers who had never taken birth control pills.

Sadly, some women—many young—who learn that they carry gene mutations have had their breasts removed in an attempt to avoid breast cancer. Removing a man's prostate may prevent prostate cancer. But these are not answers. A 2010 study found that women who had their breasts removed as a preventive measure did not get breast cancer—they couldn't, of course, because they didn't have breasts. But only 7% of those who decided to forgo breast removal developed breast cancer. The other 93% did NOT develop breast cancer. [5]

Mounting evidence links a wide array of **toxic** environmental chemicals to breast cancer. Included are various pesticides, polycyclic aromatic hydrocarbons, solvents, DDT, polyvinyl chloride, bisphenol-A, polychlorinated biphenyls, dioxin, flame retardants, ethylene oxide, parabens, phthalates, food additives, methyl mercury, smoking and second-hand smoke. Living near a nuclear facility, incinerator, leaching landfill, superfund site, paper mill, gas station, chemical factory, industrial agriculture, airport, mining operation, electrical substation, or golf course can expose you to cancer-causing pollutants. Nitrogen dioxide, polycyclic aromatic hydro-carbons and benzene produced by vehicular traffic can increase risk. Women who consistently eat well-done steak, hamburgers and bacon have a 4.62-fold increased risk of breast cancer. Cooking foods at **high** temperatures causes the formation of heterocyclic amines (HAs) which are linked to breast cancer. Grilled salmon contains enough HAs to cause gene mutation. Parabens are synthetic anti-fungal preservatives used in more than 13,000 cosmetics and personal care products including deodorant. Parabens, organochlorine pesticides and PCBs, just to mention a few, are among environmental estrogenic chemicals that accumulate in breast tissue. Air fresheners, products for mold and mildew control, and many cleaning products contain hormone-disrupting ingredients. Organochlorine pesticides used in homes, construction materials, furnishings, plastic computer housings, on golf courses, lawns, and more, are found in much higher amounts in cancerous breast tissues than in normal breast tissues. Hormone-disrupting plasticizers are absorbed from auto and industrial exhaust, furnishings like carpets and sofas, new car interiors, construction materials, and all common plastic, polyvinyl, acrylic, Styrofoam and styrene items. Chemical pollutants in waterways are piped into our homes. The fact that more men are also developing breast cancer (up 25% in 25 years) seems to indict environmental exposures and lifestyle factors. Toxic chemicals interfere with normal liver function and disrupt the body's balance of hormones. Residues accumulate over time in fatty tissues like the breast. It's plausible that, more than a woman's own hormones, cancer risk comes from toxic chemicals and hormone drugs. These can harm directly by disrupting hormones, or indirectly by interfering with the body's ability to produce and metabolize needed hormones.

We can't avoid all toxins, but we can avoid **many**. As much as possible, obtain organic foods raised without synthetic fertilizers and pesticides. Get meats and milk products animals raised without pesticide-laden feeds, hormones and drugs. Eat only small ocean fish. Avoid dairy products from cows given recombinant bovine growth hormone (rBGH or rBST). Avoid trans fats and other altered fats and oils. Eat whole real foods; avoid processed, prepackaged foods which can contain chemicals like BPA or phthalates. Store beverages and food in glass instead of plastic. Use glass baby bottles and BPA-free sippy cups and toys for little ones. Avoid cosmetics containing parabens. Use truly natural toiletries such as shampoo, toothpaste, deodorant. Don't use pesticides around the home, commercial cleaning products (use non-toxic cleaners), plastic food wrappers and storage bags, plastic cups and bottles, foods in cans with plastic linings. Opt for chemical-free rugs, avoid additives like "stain-resistant," choose wood instead of vinyl and plastic. Avoid vinyl shower curtains; use cotton duck instead. Allow new products to off-gas outside before bringing them inside. Make sure to drink and cook with clean water. If possible, avoid living areas with high air pollution. If you live in a polluted area, don't exercise outdoors when air-quality is unhealthy. Don't smoke and stay away from others who do smoke. Don't have unnecessary x-rays. Avoid artificial air fresheners, dryer sheets, fabric softeners or other synthetic products that can disrupt hormone balance. Replace non-stick pots and pans with ceramic, glass, or stainless steel. When redoing your home, look for toxin-free alternatives in lieu of chemically-treated materials. A good **detoxification** program periodically can help eliminate some toxic compounds and potentially lower risk.

In 2000 Finnish scientists found that women who used antibiotics to treat urinary tract infections increased their risk of breast cancer. In 2004 researchers reported that women who had breast cancer tended to have a history of heavier **antibiotic** use than cancer-free women. Risk was greater with longer duration of antibiotic use and every class of antibiotics was linked to increased risk. Antibiotics alter intestinal flora and reduce the ability to metabolize nutrients and other food components that may help protect against cancer. The drugs may kill off intestinal bacteria that neutralize potentially cancer-causing substances. Healthy bacteria are a crucial part of the immune system. Antibiotics disrupt the processing of estrogen by intestinal microflora, alter estrogen byproducts, and adversely affect immune function, thus lowering the body's defense and repair capacities. Women taking antibiotics may already have weakened immune systems, leading to ills and anti-biotic prescriptions that continue to weaken and damage their immune systems. Risk of cancer is boosted. [6]

PREVENTIVES? Some drugs are being used to prevent breast cancer. **Tamoxifen** (Nolvadex), a selective estrogen receptor modulator (SERM) is prescribed mostly to women at high risk for estrogen-receptor positive breast cancers or who have cancer to prevent a recurrence. A SERM stimulates estrogen-receptor action in bone but counteracts action of estrogen in the uterus and breast. Studies have had varied results, collectively unimpressive. Tamoxifen has risks, the reason why in one study, only 1% of participants actually took the drug. Side effects include increased risk of uterine cancer, cataracts, stroke, and blood clots in the lungs. Taken longer than 5 years, it may increase the risk of an aggressive and difficult-to-treat breast cancer. The World Health Organization classifies tamoxifen as a carcinogen. Many women who take the drug (up to 95% in some studies) derive no benefit at all. Seven-year follow-up data on healthy but high-risk women indicate tamoxifen has not been proven to reduce breast cancer mortality. **Raloxifene** (Evista), an osteoporosis drug and another SERM, is being used for high risk women. One study claimed a 50% reduction in breast cancer incidence. Of the 9700-plus women in each of 2 drug groups (tamoxifen and raloxifene), 167 got breast cancer. This translates to 1.7%, whereas 3.4% would be 'expected' to develop cancer—hence the supposed 50% reduction. Raloxifene increases risk of venous blood clots and fatal stroke. Leg cramps and hot flashes are among the side effects of both drugs. A 2011 study used **exemestane** (Aromasin), an aromatase inhibitor which lessens the amount of estrogen made in the body, as a preventive. Results: 1.4% of untreated women developed breast cancer compared with about a half of 1% in the drug-treated women. One invasive breast cancer may be avoided for every 94 women taking the drug daily for 3 years. Side effects included hot flashes, fatigue, sweating, insomnia, joint pain, and as other aromatase inhibitors can cause blood clots and stroke, this one probably does too. **Aspirin** is touted as a preventive though study results have not really proved this. Generally epidemiological studies have been used, not clinical trials. So no true cause and effect can be claimed. Gastric ulcers and other complications are known to be caused by aspirin. And since aspirin interferes with the natural process of inflammation—the body's attempt to repair tissues that have been insulted or injured—the immune system may be compromised. Researchers in a 2004 study reported that women who took 7 or more aspirin tablets per day had the greatest breast cancer risk reduction. Yet the Nurses' Health Study, involving over

88,000 women, showed a 58% increase in pancreatic cancer risk in people who took just 2 aspirins per week. There is evidence that aspirin increases risk of estrogen- and progesterone-receptor negative breast cancer. [7]

To support a woman's hormonal **balance**, the following can be considered:

Before menopause: Just Before Two Meals:	After menopause: Just Before Two Meals:
1 Symplex F (chew)	1 Symplex F (chew)
1 Ovex (chew)	1 Drenatrophin PMG (chew)
1 Wheat Germ Oil	2 Cataplex C (chew)
1 Chlorophyll Complex	2 Chlorophyll Complex
1 Black Currant Seed Oil	1 Evening Primrose Oil (MediHerb)

---

[1] MH Forouzanfar, KJ Foreman, et al, *Lancet*, 22 Oct 2011**,** 378(9801):1461-84; *Health News*, Jun 2001, 7(6):8; J Swartzberg, *UC Berkeley Wellness Lttr*, Mar 2006, 22(6):3; Dj Biau, R Porcher, *JAMA*, 16 May 2012, 307(19):2023; *UC Berkeley Wellness Lttr*, Feb 2009, 25(5):2-3; RM Williams, *Townsend Lttr*, Oct 1999, 195:158-9; U Beronesi, P Boyle, et al, *Lancet*, 14 May 2005, 365(9472):1727-41.

[2] M Napoli, *Health Facts*, Aug 2006, 31(8):3, 8; NIH State-of-the-Science Conference, Sept 2009, http://consensus.nih.gov/2009/deisstatement.htm; http://brest-cancer-research.com/content/6/S1/P23; *Breast Cancer Res*, 11 Nov 2005, PMID:1657703; L McTaggart, *What Doctors Don't Tell You*, Nov 2002, 13(8):5; J Mercola, http://articles.mercola.com/sites/articles/archive/2011/02/14/beating-breast-cancer....

[3] S Loft, et al, *Cancer Epidemiol Biomarkers Prev*, Sept 2005, 14(9):2137-42; P Bronson, *Townsend Lttr*, Nov 2008, 304:85-7; T Key, et al, *J Natl Cancer Inst,* 2003, 95:1218-26???; *John R Lee MD Med Lttr*, Feb 2001:1 & June 2001:3, 8 & Jan 2003: 2-8; C Rutter, M Mandelson, et al, *JAMA*, 10 Jan 2001, 285(2):171-6; P Newcomb, L Titus-Ernstoff, et al, *Cancer Epidemiol Biomarkers Prev*, 2001, 11:593-600; K Johnson, *Fam Practice News*, 15 Jul 2003, 33(14):1, 5; T Lagro-Janssen, W Rosser, et al, *Lancet*, 9 Aug 2003, 362:414-15; V Beral, et al, *Lancet*, 9 Aug 2003, 362:419-27; H Kuhl, *Climacteric*, 2004, 7:319-23; L Rosenberg, J Palmer, et all, *Arch Intern Med*, 2006, 166:760-5; W Chen, J Manson, et al, *Arch Intern Med*, 2006, 166(9):1027-32; A LaCroix, R Chlebowski, et al, *JAMA*, 2011, 305:1305-14; Presentation at Assn for Cancer Research Annual Meeting, Chicago, Ill, 4 Apr 2012; *Tufts Univ Hlth & Nutr Lttr*, Feb 2007, 24(12):3; R Moss, *Townsend Lttr*, Apr 2007, 285:154-6; M Kumle, *Lancet*, 23 Aug 2008, 372(9639):608-10; *Hlth Facts*, Apr 2009, 34(4):5; E Grant, *JAMA,* 8 May 2002, 287(19):2360-1; R Tamimi, S Hankinson, et al, *Arch Intern Med*, 2006, 166:1483-9; R Burkman, et al, *Fertility & Sterility*, 2003, 79:844-51; *BMJ*, 2001, 322:586-7; B Stoll, *Eur J Clin Nutr*, 1999, 53:771-5; C Diorio, M Pollak, et al, *Cancer Epidemiol Biomarkers Prev*, May 2005, 14(5):1065-73; N Allen, P Appleby, et al, *Cancer Epidemiol Biomarkers Prev*, Nov 2002, 11:1441-8; J Hansen, *Epidemiology*, 2001, 12:74-7; S Davis, D Mirick, et al, *J Natl Cancer Inst*, 17 Oct 2001, 93:1557-62; E Schernhammer, F Laden, et al, *J Natl Cancer Inst*, 17 Oct 2001, 93(20):1563-8; J Kliukiene, et al, *Brit J Cancer*, Jun 2001, 84:397-9; A Gaby, *Townsend Lttr*, May 2001, 214:20; preg & lactation: K Greene, *Science News*, 12 Nov 2005, 168(20):307-8; P Newcomb, B Storer, et al, *N Engl J Med*, 1994, 330:81-7; Collaborative Group on Hormonal Factors in Breast Cancer, *Lancet,* 20 Jul 2002, 360(9328):187-95; T Zheng, L Duan, et al, *Am J Epidemiol*, 2000, 152(12):1129-35.

[4] A Eliassen, G Colditz, et al, *JAMA*, 12 Jul 2006, 296(2):193-201; HS Feigelson, AV Patel et al, *Cancer*, 1 Jul 2006, cited in *Tufts Univ Hlth & Nutr Lttr*, Aug 2006, 24(6):8; K Micels, K Terry, et al, *Arch Intern Med*, 2006, 166(21):2395-402; R MacInnis, D English, et al, *Cancer Epidemiol Biomarkers Prev*, Dec 2004, 13(12):2117-25; J Barnett, *Nutr Rev*, Feb 2003, 61(2):73-6; Z Huane, S Hankinson, et al, *JAMA*, 5 Nov 1997, 278(17):1407-11; *Hlth & Healing*, Jul 2010, 20(7):5; *Tufts Univ Hlth & Nutr Lttr*, May 2004, 22(3):2; S Maruti, et al, *J Natl Cancer Inst*, 2008, 100:728-37; B Tehard, C Friedenreich, et al, *Cancer Epidemiol Biomarkers Prev*, 2006, 15(1):57-64; A Eliassen, S Hankinson, et al, *Arch Intern med*, 2010, 170(19):1758-64; A Bardia, L Hartmann, et al, *Arch Intern Med*, 2006, 166:2478-83; A McTiernan, C Kooperberg, et al, *JAMA*, 10 Sept 2003, 290(10):1331-6; R Segal, *Altern Ther Women's Hlth*, May 2002, 4(5):33-6; E John, P Horm-Ross, et al, *Cancer Epidemiol Biomarkers Prev*, Nov 2003, 12:1143-52; A Patel, M Press, et al, *Cancer*, 2003, 98:2161-9; *UC Berkeley Wellness Lttr*, May 2009, 25(8):1; B Liebman, *Nutr Action Hlthlttr*, Jul/Aug 2010, 37(6):1-7.

[5] E Ziv, J Cauley, et al, *JAMA*, 13 Jun 2001, 285(22):2859-63; T Walsh, S Casadei, et al, *JAMA*, 22/29 Mar 2006, 295(12):1379-88; *UC Berkeley Wellness Lttr*, May 2006, 22(8):7 & Oct 2011, 28(1):5; R Pluta, *JAMA*, 1 Jun 2011, 305(21):2244; R Shakya, L Reid, et al, *Science* 28 Oct 2011, 334 (6055):525-8; R Travis, G Reeves, et al, *Lancet*, 19 Jun 2010, 375(9732):2143-51; P Barry, *Sci News*, 16 Jun 2007, 171(24):371-2; N Seppa, *Sci News*, 2 Dec 2006, 170(23):355 & 25 Sept 2010, 178(7):12; C Begg, R Haile, et al, *JAMA*, 9/16 Jan 2008, 299(2):194-201; E John, A Miron, et al, *JAMA*, 26 Dec 2007, 298(24):2869-76; E Stokstad, *Science*, 31 Mar 2006, 311(5769):1847; M Mayer, *Breast Cancer Action Newslttr*, Feb/Mar 2004, #80; A Poole, Y Li, et al, *Science*, 1 Dec 2006, 314(5804):1467-8; Collaborative Grp on Hormonal Factors in Breast Cancer, *Lancet*, 27 Oct 2001, 358(9291):1389-99; L Shi, *Science*, 2 Jul 2010, 329(5987):32; J Couzin, *Science*, 2 Feb 2007, 315(5812):592-4; A Hynes, *Nat Health*, Oct 2005, 35(9):56-65; O Johannsson, N Loman, et al, *Lancet*, 24 Oct 1998, 352(9137):1359-60; H Frankish, *Lancet*, 24 Aug 2002, 360(9333):625; *Genes & Nutr*, May 2011, 6(2):93-108; J Kotsopoulos, SA Narod, *Cancer Causes Control*, 2005, 16:125-38; V Premkumar, S Yuvaraj, et al, *Br J Nutr*, 2008, 100:1179-82; J Benson, www.naturalnews.com/z030041_breast_cancer_genes.html, 14 Oct 2010; S Lee, J Fowke, et al, *Am J Clin Nutr*, Mar 2008, 87(3):753-60; Couzin, Yu et al, Manke et al, King et al, Levy-Lehad, *Science*, 24 Oct 2003, 302(5645):574-5, 591-3, 636-46; J Stephenson, *JAMA*, 8 Jan 2003, 289(2):164.

[6] T Hudson, *Townsend Lttr*, May 2004, 250:162-3 & Jul 2011, 336:104-5; R Williams, *Townsend Lttr*, Jun 2002, 227:39-41 & Nov 2004, 256:45-7; S Rogers, *Total Wellness*, Sept 2004:1-3 & Nov 2004:3-5 & Aug 2005:4-5; S Toland, *Nat Solutions*, Oct 2010, 128:41-7; *Pesticides & You*, Summ 2007, 27(2):8 & Fall 2007, 27(3):7; http://sciencereview.silentspring....; *UC Berkeley Wellness Lttr*, Aug 2001, 17(11):1 & Mar 2010, 26(6):1 & Dec 2011, 28(3)8; P Band, N Le, et al, *Lancet*, 5 Oct 2002, 360(9339):1044-9; F van Leeuwen, et al, *J Natl Cancer Inst*, 2003, 95:971-80; NCAMP *Tech Rpt*, Aug-Sept 2007, 22(8-9):6-8 & Feb 2007, 22(2):3 & May 2007, 22(5):2-3; C Holden, *Science*, 1 Jun 2007, 316(5829):1261; M Warner, et al, *Environmental Hlth Perspec*, Jul 2000, 110:625-8; A Heyer, P Grandjean, et al, *Lancet*, 5 Dec 1998, 352(9143):1816-20; C Charlier, A Albert, et al, *Occup Environ Med*, 2003, 60:348-51; *J Appl Toxicol*, May 2012, 32(5):305-9 & Mar 2012, 32(3):219-32; G Vince, *NewScientist.com*., 12 Jan 2004; J Whelan, *New Scientist.com.* 24 Jan 2004; *J Applied Toxicol*, Apr 2011, 31(3):262-9 & 12 Jan 2012, 32(3):219-32 & 1 Feb 2012, 32(5):305-9; C Velicer, S Heckbert, et al, *JAMA*, 18 Feb 2004, 291(7):827-35; R Ness, J Cauley, *JAMA*, 18 Feb 2004, 291(7):880-1; *UC Berkeley Wellness Lttr*, May 2004, 20(8):2-4.

[7] S Wolfe, *Worst Pills, Best Pills News*, Aug 2002, 8(8):60-1 & Jul 2006, 12(7):49-51; R O'Regan, *Lancet*, 29 Apr 2006, 367(9520):1382-3; *Cancer Res*, 2009, 69:6865-70, cited in *What Doctors Don't Tell You*, Oct 2009, 20(7):5; G Caine, A Blann, et al, *Lancet*, 11 Jan 2003, 361(9352):177-8; M Napoli, *HealthFacts*, Aug 2002, 27(8):3 & May 2006, 31(5):4; J Bradbury, *Lancet*, 6 Jul 2002, 360(9326):63; L Speroff, *OB/GYN Clin Alert*, Jun 2006, 23(2):9-11; *Duke Med Health News*, Sept 2008, 14(9):2; VG Vogel, J Costantino, et al, *JAMA*, 21 Jun 2006, 295(23):2727-41; W Gradishar, D Cella, *JAMA*, 21 Jun 2006, 205(23):2784-6; M Napoli, medconsumers.org/2011/06/06/can-this-drug-prevent-breast-cancer/…; N Seppa, *Sci News*, 2 Jul 2011, 180(1):16; *UC Berkeley Wellness Lttr*, Aug 2004, 20(11):1; M Terry, *JAMA*, 26 May 2004, 291(20):2433-40; T Hudson, *Townsend Lttr*, Aug/Sept 2009, 313/314:132; S Wolloshin, L Schwartz, *JAMA*, 22/29 Sept 2004, 292(12):1426; J Wright, *Nutr & Healing*, Aug 2004, 11(7):6; mercola.com, archive, 17 Jun 2012.