

## Laurelhurst Integrative Health

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## Frequently Asked Questions about Hormones & Breast Cancer

### What is breast cancer?

Breast cancer occurs when cells in the breast divide and grow uncontrolled. The cell cycle is the natural mechanism that regulates the growth and death of cells. When normal cell regulators malfunction and cells don't die at the proper rate, cell growth goes unchecked and cancer can develop.

Breast cancer tumors usually grow slowly. By the time a tumor is large enough to be felt as a lump, it may have been growing for 10 years and spread of tumor cells (metastasis) may have already occurred. Therefore, screening methods (via mammography, ultrasound, MRI, or thermography) are very important. In addition, preventive measures such as a healthy diet and lifestyle, nutritional supplementation, and exercise are crucial.

### What are the risk factors for breast cancer?

It's helpful to categorize modifiable risk factors from those that cannot be changed. Most breast cancer risk factors are modifiable, meaning they can be changed based on daily choices regarding diet, exercise, lifestyle habits, and stress management.

#### Non-modifiable risk factors:

- Female
- Advancing age
- Family history (BRCA1 or 2)
- Early age menarche (first menstrual period)
- Late menopause
- Diethylstilbestrol (DES) use by mother

#### Modifiable risk factors:

- Obesity (nearly triples risk)
- Lack of exercise
- Synthetic HRT, especially progestins
- Birth control pill use
- High animal and trans fats/low fiber/low fruit and vegetable diet
- Smoking
- Breast trauma
- Late age pregnancy, never having been pregnant, lack of breast feeding
- High alcohol intake (>1 drink per day)
- Cigarette smoking
- Working the "graveyard" shift  
Environmental toxins (radiation, xenoestrogens, secondhand smoke)
- Benign breast disease (fibrocystic breast changes/disease may or may not increase risk)

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**Can you explain the affects of various hormones on breast tissue?**

**Estrogens**, especially estradiol and estrone, stimulate proliferation of breast tissue and breast cancer cells. There is much confusion about the use of estrogen replacement therapy (ERT) and breast cancer risk; this is true for the use of human identical (bio-identical) estrogens as well as synthetic forms, such as Premarin. It's helpful to review the results from 3 very large, well-conducted studies over the past several years – the Women's Health Initiative, the Million Women Study, and the Nurses' Health Study – to clarify the relationship between estrogen replacement and breast cancer risk. The Women's Health Initiative study (JAMA 2002) found an increased risk of breast cancer in women using Premarin along with the synthetic progestin Provera, but not in women using Premarin alone. The Million Women Study (Lancet 2003) did show an increased risk with ERT (all forms, except intravaginal estriol) and an even higher risk with estrogen plus synthetic progestins. Lastly, the Nurses' Health Study (NEJM 1995) found the same results, higher breast cancer risk with ERT, and much higher when synthetic progestins were added.

It's important to note that several studies have shown no increased risk with the use of bio-identical **estriol**, especially when used intravaginally (Bergkvist 1989, Million Women Study 2003, Fournier 2004, Rosenberg 2006, Lyytinen 2006). In addition, vaginal estriol use in breast cancer survivors does not increase the risk of recurrence or death (Dew 2003)

The data is irrefutable that **synthetic progestins** (such as Provera) significantly increase the risk for breast cancer. However, human identical (bio-identical) **progesterone** is a different molecule than synthetic progestins and has a different effect on breast cancer risk. Progesterone deficiency (in women who don't ovulate or who don't make enough progesterone) has been shown to increase the risk for breast cancer. In one study (Cowan 1981) progesterone deficient women had a 5.4 fold risk of premenopausal breast cancer, and a 10 fold increase risk of death from all malignant cancers. Another study (Sturgeon 2004) evaluated hormone levels in women under age 45 who developed premenopausal breast cancer. There was no association between serum SHBG (sex hormone binding globulin), estradiol, testosterone or androstenedione and premenopausal breast cancer risk. The only link was between luteal phase progesterone levels – the highest level was associated with the lowest risk. **Cyclic progesterone supplementation** (oral micronized progesterone, such as Prometrium, topical progesterone cream, or intravaginal progesterone) does not increase breast cancer risk (Plu-Bureau 1999, Fournier 2004)

The role of **testosterone** in breast cancer is often confusing, due to the use of synthetic (methyl) testosterone v. bio-identical testosterone in many studies. Clinical evidence supports that bio-identical testosterone is breast protective. Androgens are known to inhibit breast cancer in almost every breast cell line via the androgen receptor (Hackenberg 1956, Szelei 1997, Ortmann 2002). Adrenal androgens counteract the growth stimulatory affects of estrogen on breast cancer cells (Bocuzzi 1994). Non-oral testosterone supplementation, including testosterone delivered by pellet implant, has been shown to prevent breast proliferation, decrease estrogen receptor alpha and prevent the stimulation of breast tissue from estrogen/progestin therapy (Zhou 2000, Dimitrakakis 2003, Slagter 2006, Hoffling 2007). Bio-identical testosterone, delivered by pellet implant has also been shown to lower the risk of breast

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cancer when given in combinations with estrogen and estrogen/progestin therapy (Dimitrakakis 2004, Gambrell 2006) Testosterone and other androgens have been used to successfully treat breast cancer patients (Testosterone Monograph, Clin Pharm 2000, Greenblatt 1949).

Testosterone's action is anti-proliferative and pro-apoptotic and is mediated through the androgen receptor (Szelei 1997, Kandouz 1999, Lapointe 1999, Ando 2002, Ortmann 2002). Interestingly, synthetic progestins may act as endocrine disruptors that increase the risk of breast cancer by blocking the androgen receptor and the negating the protective effects of testosterone on breast tissue (Birrell 2007).

### **I've heard doctors claim that there is no difference between bio- identical hormones and synthetic hormones. Do you agree?**

No – that statement is not based on available data. For example, synthetic progestins and bio-identical progesterone do not have the same risk for breast cancer. The same is true for synthetic methyl-testosterone and bio-identical testosterone (see above). It may also be helpful to review Dr. Retzler's "Bio-identical Hormone Position Paper".

### **The FDA recently claimed that there was no data on the safety of estriol. Is that a valid statement?**

That is not true. No trial has shown an increased risk of breast cancer with vaginal estrogen (especially estriol) (Bergkvist 1989, Million Women Study 2003, Fournier 2004, Rosenberg 2006, Lyytinen 2006). Vaginal estriol use in breast cancer survivors does not increase the risk of recurrence or death (Dew 2003). Vaginal estriol does not increase the risk of endometrial hyperplasia or uterine cancer (Bachmann 1977). There is no accumulation of hormones or metabolites with vaginal estrogen or progesterone therapy (Keller 1981, Trevoux 1982, Nahoul 1993, Levy 2000, Kuhl 2005).

### **Do you treat patients with a history of breast cancer with hormones? How do you treat them?**

Women with a history of breast cancer deserve to be adequately informed about each individual hormone and risk of recurrence. I recommend breast cancer survivors do not use estradiol replacement, unless safer forms of estrogen (estriol) have been unsuccessful at alleviating symptoms. Therefore, I usually prescribe intravaginal estrogens (especially estriol) which does not increase the risk of breast cancer or recurrence. In addition, testosterone supplementation, especially via pellet implant, is excellent at managing many symptoms.

Breast cancer patients also receive individualized exercise, dietary, and stress management counseling, as well as supplement recommendations. All women are encouraged to take part in my yearly Transformational Health & Detox group, to support healthy detoxification and elimination, as well as fine-tune diet, exercise, and stress management habits. Treatment recommendations are evidence-based when possible, and based on physiology when research isn't extensive.

### **What else do you recommend for patients for prevention?**

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- Drink alcohol only in moderation or not at all (<2 drinks per day – 1 drink increases breast cancer risk by 10%, and 2 drinks increase the risk by 20%)
- Exercise – 30-45 minutes every 24-48 hrs, has been shown to decrease risk up to 60%
- Early childbearing and breast feeding, when feasible
- Maintain normal weight
- No smoking
- Empty “stress bucket” daily
- Maintain vitamin D levels in optimal range: 40-70 ng/mL
- Yearly detox

### Do you think stress plays a role in breast cancer?

Absolutely! Chronic, prolonged stress suppresses natural killer (NK) cell function. Natural killer cells are a type of white blood cell that seek out and destroy cancer cells. Chronic stress has been shown to impair the cell's ability to repair DNA damage, leading to increased possibility of defective cell division.

### Are there any natural supplements that can help decrease risk?

- **Indol-3-carbinol** – 200 to 600 mg per day. I3C enhances estrogen metabolism and has been shown to lower breast cancer risk (see handout on I3C).
- **Green tea** contains EGCG, which helps block vascular endothelial growth factor (VEGF), preventing angiogenesis (formation of blood vessels feeding a tumor). Green tea has also been shown to inhibit tumor growth and increase apoptosis (tumor cell death). Suggested dosage is 300-1500 mg of green tea capsules per day. For breast cancer prevention, drinking 3 cups of green tea per day is recommended.
- **Melatonin** inhibits breast cancer cell growth and reduces tumor spread. A high percentage of breast cancer patients have low melatonin. In addition, women who work at night have a higher risk. Recommended dosage depends on prevention (3-5 mg) or breast cancer treatment (20-50 mg)
- **Fish oil** – 2,000 to 6,000 mg EPA and DHA per day. Higher omega-3 to omega-6 ratio may reduce the risk of breast cancer, especially in pre-menopausal women (Goodstine 2003). Fish oil has been shown to retard the growth of breast cancer in vitro, and inhibit tumor development and metastasis in animal studies.
- **High antioxidants** – carotenes, C, E, selenium, zinc