FACT SHEET...

The Biologic Cause of the Abortion Breast Cancer Link: The Physiology of the Breast

The physiology of the breast provides the strongest evidence of the causal link between abortion and breast cancer. The same biology that accounts for 90% of all risk factors for breast cancer accounts for the ABC link.

Simply stated, the biology rests on two principles.

1. *The more estrogen a woman is exposed to in her lifetime, the higher her risk for breast cancer.* It is well established that estrogen is implicated in the formation of three cancers: uterine, germ cell and breast.[1] Estrogen can induce cancers to form in two ways, as a *genotoxin* and a *mitogen*. A *genotoxin* or *mutagen* directly damages the DNA (causes mutations), initiating a process that leads to the formation of cancer cells. Certain natural metabolites of estrogen have been shown to cause mutations[2]. A *mitogen* causes cells to proliferate, that is, to multiply through division (mitosis). Each a time a cell divides to form two cells, it must replicate its DNA. During replication, mutations in the form of copying errors and/or chromosomal translocations can occur, causing abnormal cells to form. These abnormal cells can go on to become cancerous. The stimulation of proliferation (mitogenesis) that estrogen causes, increases the chances that abnormal cells will grow into malignant tumors. Estrogen is so potent that it is measured in parts per trillion.

   If a woman starts her menstrual cycles early, say, age nine and continues to menstruate into her late 50's, she is at higher risk for breast cancer, as she has been exposed to monthly estrogen elevations for a long period of time. This too is the science behind a recent, well-publicized study that shows that estrogen-based hormone replacement therapy increases the risk of breast cancer.[3] In a similar way, birth control pills can elevate breast cancer risk.

2. *The earlier a woman's breast matures from prepubescent (Type 1) and pubescent (Type 2) lobules to reproductive (Type 3) and lactation (Type 4) lobules (see diagrams), the lower her risk of breast cancer.*[4] Type 1 and 2 lobules are known to be where cancers arise. Type 1 lobules are also known as the TDLUs (terminal ductal lobular units)[5] where 80% of all breast cancers are formed; i.e., the in-situ and invasive ductal cancers. Type 3 and 4 lobules are mature and resistant to carcinogens. When a female child is born, she has only a small number of primitive Type 1 lobules. At puberty, when estrogen levels rise, the breast forms Type 2 lobules. It is only through the hormonal environment and length of a full term (or to at least 32 weeks) pregnancy that there is full maturation of Type 3 and 4 lobules in the breast. This maturation protects a woman and lowers her risk of breast cancer.

   This is why women who undergo full term pregnancies have lower risk of breast cancer and why women who remain childless have higher risk of breast cancer. Women who give birth after age 30 are also at increased risk of breast cancer as their immature Type 1 and 2 lobules are exposed to estrogen for the many years between the time of their first menstruation until their first full term pregnancy. Abortion in women under 18 and over 30 years old carries the greatest risk: these women have the highest percentage of Type 1 lobules in their breasts.[6]

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[1] Henderson BE, Ross R, Bernstein L. Estrogen is a cause of human cancer: The Richard and Hilda Rosenthal Foundation Award Lecture. Cancer Research 1988;48:246-53.

[2] Miller, Katherine. Estrogen and DNA damage: The silent source of breast cancer? *J Natl Cancer Inst* 2003: 95:100-102.

[3] Writing group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women. *JAMA* 2002;288:321-33.

[4] Diagrams taken from eds. Schwartz, Shires, Spencer, *Principles of Surgery* (McGraw Hill)

[5] Russo J, Tay LK, Russo IH. Differentiation of the mammary gland and susceptibility to carcinogenesis. *Breast Cancer Res & Treatment* 1982;2:5-73.

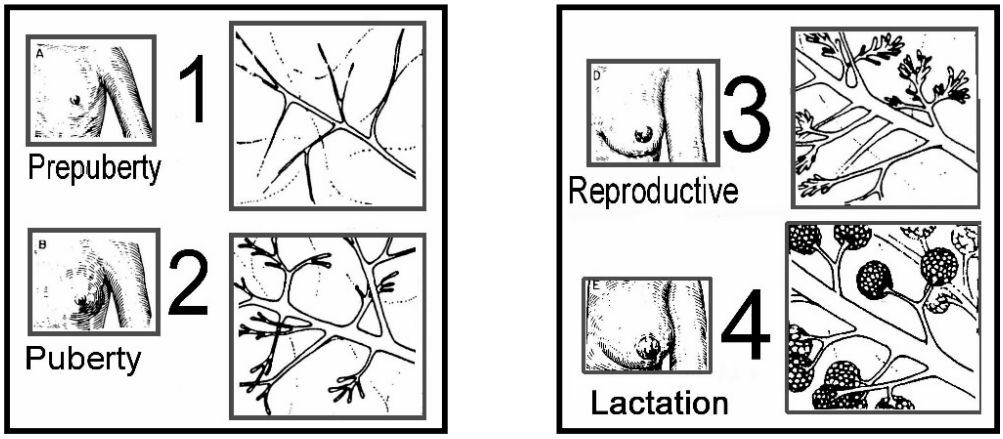
[6] Daling JR, Malone DE, Voigt LF, White E, Weiss NS. Risk of breast cancer among young women: relationship to induced abortion. *J Natl Cancer Inst* 1994;86:1584-92.

It is the interplay of these two principles, estrogen exposure and breast lobule maturation, which accounts for the fact that abortion can lead to breast cancer. Within a few days of conception, a woman's estrogen level rises. By the end of the first trimester, estrogen levels have increased by 2000%. The estrogen stimulation that causes the multiplication of Type 1 and 2 lobules, results in sore and tender breasts early on in pregnancy. It is only after 32 weeks that a woman's breasts stop growing larger and mature into Type 3 and 4 lobules in preparation for breast-feeding.

If abortion ends a woman's pregnancy before full maturation of her breasts, she is left with an increased number of the immature Type 1 and 2 lobules. She now has a greater number of the breast lobules where cancers can arise. This causes her to be at greater risk for breast cancer. It is through this same biologic mechanism that *any* premature birth before 32 weeks more than doubles breast cancer risk.[7]

Induced abortion thus increases breast cancer risk by two mechanisms. First, abortion leaves the breast with increased numbers of Type 1 and Type 2 lobules, those lobules in which cancer cells are formed, which are then exposed to more estrogen through menstrual cycles. These lobules would otherwise have been protected from cancer by maturation to Type 3 and 4 lobules, if pregnancy had gone to term. Second, the breast is exposed to high levels of estrogen during pregnancy, which can induce cancer cells to form.

Types of Breast Lobules



Diagrams taken from Schwartz, Shires, Spencer, eds., Principles of Surgery 5th Ed. (McGraw Hill, 1989)

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[7] Melbye M, *et al*. Preterm delivery and risk of breast cancer. *Brit J Cancer* 1999;80:609. Pregnancies that result in first trimester spontaneous abortions produce subnormal estrogen concentrations and generally *do not* increase breast cancer risk. That is, there is little estrogen stimulation of the breast. Stewart DR, Overstreet JW, Nakajima ST, Lasley BL. Enhanced ovarian steroid secretion before implantation in early human pregnancy. *J Clin Endocrinol Metab* 1993;76:1470-6. Kunz J, Keller PJ. HCG, HPL, oestradiol, progesterone and AFP in serum in patients with threatened abortion. *Br J Obstet Gynaecol* 1976;83:640-4. Witt BR, Wolf GC, Wainwright CJ, Johnston PD, Thorneycroft IH. Relaxin, CA-125, progesterone, estradiol, Schwangerschaft protein, and human chorionic gonadotropin as predictors of outcome in threatened and non-threatened pregnancies. *Fertil Steril* 1990;53:1029-36. Norman RJ, McLoughlin JW, Borthwick GM, Yohkaichiya T, Matthews CD, MacLennan AH, de Kretser DM. Inhibin and relaxin concentrations in early singleton, multiple, and failing pregnancy: relationship to gonadotropin and steroid profiles. *Fertil Steril* 1993;59:130-7