

How Does the Independent Link Between Abortion and Breast Cancer Work?

Two principles account for the link:

- I. Estrogen Exposure
- 2. Breast Lobule Formation

Estrogen Exposure

As soon as conception occurs and even before implantation in the uterus, the embryo secretes a hormone, hCG (Human chorianic gonadotropin), which causes the mother's ovaries to produce more estrogen and progesterone. This causes the mother's breasts to become sore and tender.

In a viable pregnancy, estrogen levels increase 2,000% by the end of the 1st trimester. This surge in hormones causes the breasts to grow by making more Type I & 2 lobules where cancers can start. The breast doubles in volume by 20 weeks.

Breast Lobule Formation

- After 32 weeks of pregnancy, the Type I and 2 lobules mature into Type 3 and 4 lobules in preparation for breast feeding.
- If the pregnancy ends by elective abortion, the increase in numbers of Type I and 2 lobules formed in the first two trimesters provide more places for cancers to start, increasing risk.
- Women who never carry a pregnancy beyond 32 weeks never fully mature their breast tissue and have increased risk.
- Women who delay full-term pregnancy past age 30 have a 90% higher risk of breast cancer than those who carry a pregnancy to term by age 20.

What about Miscarriage?

If induced abortion is a problem, does spontaneous loss (miscarriage) in the first trimester carry the same risk?

Studies have shown dramatically lower levels of female hormone in those who miscarried.^{3,4} The less estrogen a woman produces, the lower her risk of breast cancer. Her breasts are not stimulated and remain unchanged.

Additionally, the overwhelming majority of epidemiological studies have shown that miscarriage is not associated with an increased risk of breast cancer. ^{5,6}

Epidemiologic Studies Support the ABC Link

Since 1957 there have been 67 studies done concerning induced abortion and breast cancer risk. Of these, 51 showed a positive association and 30 were statistically significant.⁷

A Woman's Choice

A woman who chooses induced abortion **before** she has had a child:

- Denies herself the risk reduction of a full-term pregnancy.
- May never have children—a risk for breast cancer.
- Or, delay a full-term pregnancy, which increases her risk of premenopausal breast cancer by 5% per year delayed after age 20.
- Risks preterm birth of subsequent pregnancies, which doubles her risk of breast cancer if she delivers before 32 weeks and cerebral palsy in those children born preterm.
- Raises her risk by the independent link.

A woman who chooses induced abortion **after** she has had a child:

- Denies herself a further 10% reduction in risk by another full-term pregnancy.
- Will have increased the number of Type I & Type 2 lobules where cancers start in her breast.
- Risks preterm birth of subsequent pregnancies, which doubles her risk of breast cancer if she delivers before 32 weeks and cerebral palsy in those children born preterm.
- Raises her risk by the independent link.



- ¹ Cogliano V, et al. Carcinogenicity of combined oestrogenprogestagen contraceptives and menopausal treatment. *Lancet Oncol* 2005; Aug 6(8):552-3.
- ² Brind J & Lanfranchi A. Breast cancer risks and prevention, Edition 4. Breast Cancer Prevention Institute; 2007. Found at: http://www.bcpinstitute.org/booklet4.htm#metabolism.
- ³ Witt BR, et al. 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.
- ⁴ Stewart DR, et al. Enhanced ovarian steroid secretion before implantation in early human pregnancy. *J Clin Endocrinol Metab* 1993;76:1470-6.
- ⁵ Daling J, et al. Risk of breast cancer among young women: relationship to induced abortion. *J Natl Cancer Inst* 1994;86:1584-92.
- ⁶ Brind J, et al. Induced abortion as an independent risk factor for breast cancer: A comprehensive review and meta-analysis. *J Epidemiol Community Health* 1996;50:481-96.
- ⁷ Epidemiologic Studies: Induced Abortion and Breast Cancer Risk. Breast Cancer Prevention Institute 2011;

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- ⁸ Shah P, Zao J on behalf of Knowledge Synthesis Group of Determinants of Preterm/LBW Births. Induced termination of pregnancy and low birthweight and preterm birth: a systematic review and meta-analyses. *BJOG* 2009;16:1425–1442.
- ⁹ H. Swingle et al., Abortion and risk of subsequent preterm birth: a systematic review and meta-analyses. *J Reprod Med* 2009; 54:95–108



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Abortion & Breast Cancer

Is there a Link?



What is the Truth behind the Controversy?



History and Background

- In 1970, breast cancer occurred in 1 out of 12 women.
- In the 1990's that number increased to 1 in 7 women.
- Breast cancer is the only major cancer that is on the rise.
- In 1973, abortion was legalized in this country.
 Since then, invasive breast cancer has increased by 40% and non-invasive (in situ) breast cancer by 400%.

Structure of the Breast

Breast tissue contains lobules, which are composed of a milk duct and some ductules (milk glands). There are four types of lobules.

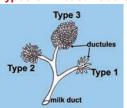
In general,

- Type I lobules—present at birth
- Type 2 lobules—form during puberty when estrogen levels rise and breasts develop
- Type 3 lobules—form after Type 4 lobules stop producing milk
- Type 4 lobules—contain colostrum (the early milk)

Before a full-term pregnancy, the breast is composed of 75% Type I and 25% Type 2 lobules. Type I lobules are where 85% of all breast cancers start as ductal cancers. Type 2 lobules form 10-15% of breast cancers which are called lobular cancers. Types 3 & 4 are resistant to cancer.

Induced abortion before 32 weeks leaves more breast tissue vulnerable to cancer because of increased exposure to estrogen hormones causing increased numbers of Type I & 2 lobules formed during the first half of pregnancy.

Types of Breast Lobules



Actual photomicrographs of human breast lobules





Type 1 Lobule

Type 3 Lobul

Factors Which INCREASE Breast Cancer Risk	
Factor	Mechanism
Alcohol	Increases estrogen exposure by impairing liver function
Benign proliferative breast disease	Result of increased estrogen exposure
BRCA genes	Inherited defects in cancer defense genes
Cigarette smoking	Benzopyrenes damage DNA
Contraceptive steroids (in pills, patches, vaginal rings, IUDs or injectable forms)	Increases estrogen exposure
Early menarche (age at first menstrual period)	Increases estrogen exposure
Female sex	Increased estrogen exposure
High socio-economic group	Delayed childbearing
Higher education	Delayed childbearing
Hormone replacement therapy (HRT)	Increases estrogen exposure
Increasing age	Premenopausal: Increases estrogen exposure Postmenopausal: Impairs immune function
Induced abortion	Leaves increased number of immature breast lobules and increases risk of premature births Increases estrogen exposure
Late childbirth (over 30 years old)	Increases exposure of Type 1 & 2 lobules to estrogen before first birth; long susceptibility window
Late menopause	Increases estrogen exposure
Nulliparity (never bearing children)	Maturity to cancer resistant breast lobules does not occur
Premature birth before 32 weeks	Leaves increased number of immature breast lobules Increases estrogen exposure
Postmenopausal obesity	Increases estrogen exposure
Radiation	Damages DNA
2nd trimester miscarriage	Leaves increased number of immature breast lobules (not associated with decreased estrogen)

Factors Which DECREASE Breast Cancer Risk		
Factor	Mechanism	
Breast feeding	Decreases estrogen by decreasing number of menstrual cycles and/or ovulation	
Cruciferous vegetables (e.g., broccoli, Brussels sprouts or DIM supplements)	Indole-3-carbinol decreases estrogen exposure by causing estrogen to be changed to an inactive metabolite of estrogen	
Early menopause	Decreases estrogen exposure	
Exercise	Unknown	
Having children (especially starting at a young age)	Decreases number of immature breast lobules	
Late menarche (age at first menstrual period)	Decreases estrogen exposure	
Omega-3 fatty acids (e.g., olive, flax seed, walnut oils)	Unknown	
Oophorectomy (removal of ovaries) before menopause	Decreases estrogen production	
Soy isoflavonoids (phytoestrogens)	May block estrogen receptors	

Factors Which HAVE NO EFFECT on Breast Cancer Risk		
Factor	Reason	
Saturated fat	Saturated fat intake not related to obesity	
Spontaneous abortions in first trimester (miscarriages)	No increased levels of estrogen as found in healthy pregnancies	

Breast Cancer and Hormone Replacement Therapy and the Pill

In June 2005, the World Health Organization concluded after review of all studies to date that estrogen-progestin combination drugs used in birth control pills and hormone replacement therapy (HRT) actually **cause** breast, cervical and liver cancer. This is a higher classification of risk than previously reported.¹

Increased breast cancer risk occurs whether these hormones are given orally, by injection, by absorption through the skin, or other means (e.g., birth control pills, Depo-Provera, the Patch, or vaginal rings). Even the newer lower-dose formulations, called "mini-pills," still increase breast cancer risk

Hormone replacement therapy (HRT), prescribed for the side effects of menopause, such as hot flashes and mood swings, also increases risk through the same mechanisms as birth control pills. The greater the number of years women take HRT, the higher the risk.

One potent synthetic estrogen, DES, has been found to increase risk in mothers and their daughters when taken during pregnancy.²

Like any medication, hormones used carefully and for short periods can be beneficial. Used for long periods of time, they can significantly increase breast cancer risk.

Alternatives to the use of these steroidal medications exist, which do not increase breast cancer risk.

