Reproductive Choices of Young Women Affect Future Breast Cancer Risk

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Abstract

Breast cancer cases have risen dramatically in recent decades around the world. Many studies from multiple nations have found induced abortion (IA) to be linked to this rise. A plausible mechanism for that link is the interference by IA with normal breast cell maturation into cancer resistant cells. Influential medical organizations have relied on studies with important design flaws which obscure this link. Major risk factors for breast cancer include delayed childbearing and not breastfeeding, both of which result from IA early in a woman’s reproductive life. Choices to delay childbearing beyond 30 years of age, to limit or avoid it altogether, to abort a pregnancy rather than to carry it to term with the adoption option, and to not breastfeed, all put young women at increased risk for breast cancer.

Introduction

The incidence of breast cancer has increased dramatically during the past 40 years, so that 1 in 8 American women will face that diagnosis. In 1973, the incidence was 82.5/100,000 women, increasing to an incidence of 110.6/100,000 women in 1992.¹ That increase may have been partially due to improved and increased screening. Although there was a decrease in the breast cancer rate following the 2003 warning by the U.S Food and Drug Administration regarding the risks of Hormone Replacement Therapy, the incidence in 2014 had reached 125/100,000 women, an increase of 52% from 1973.²

This paper will describe breast development, the biology of breast cancer, the risk factors for breast cancer, and the relationship between induced abortion (IA) and breast cancer. Induced abortion prior to 32 weeks gestation, especially earlier in a woman’s reproductive years, is a modifiable risk factor for the development of breast cancer. It is important that women have accurate risk information to shape their reproductive health decisions.

Anatomy and Physiology of Breast Development

In order to understand why abortion contributes to an increased risk of breast cancer, it is crucial to understand the anatomy and physiology of breast development. The breast is composed of three primary tissues – fat, connective, and duct/glandular tissue. When a female is born, she has little glandular tissue; specifically, only a small number of immature alveolar buds and Type 1 lobules are present. A lobule is the term used to identify the milk duct and its surrounding mammary glands composed of cells that can produce milk. These Type 1 lobules have only a rudimentary duct system – think of a tree trunk with very few branches. During puberty, young women will develop additional Type 1 lobules, and some of the
lobules will mature to pubescent or Type 2 lobules with a slight increase in the duct system – so the tree trunk has more branches. The increase in size of the breast during puberty is caused mainly by increased fat cells and connective tissue. At the end of puberty, a female’s breast will contain a mixture of approximately 75 percent Type 1 lobules and 25 percent Type 2 lobules.³

During pregnancy a woman’s breasts undergo significant maturational changes at the cellular level in preparation for breastfeeding. Beginning at conception, the embryo secretes human Chorionic Gonadotropin (hCG) that stimulates maternal ovaries to increase production of estrogen and progesterone. Within just a few days of conception, a woman’s levels of estrogen rapidly increase. By the end of her first trimester, estrogen levels have increased by 2000 percent, and Type 1 and Type 2 lobules increase in number due to the estrogenic influence. This is termed the “proliferative phase,” and by 20 weeks gestation, the breast has doubled in volume. After 20 weeks of pregnancy, during the “differentiation phase,” the immature Type 1 and Type 2 lobules begin to differentiate or mature into Type 4 lobules that are capable of producing milk. Human placental lactogen (hPL), a hormone secreted by the fetal-placental unit, is the major factor influencing the maturation of cells into Type 4 lobules. After 32 weeks of pregnancy, most lobules have matured to Type 4, and by 40 weeks, 70 – 90 percent of a female’s lobules are the mature Type 4 lobules.⁴

Type 3 lobules are formed after Type 4 lobules stop producing milk. A woman who has stopped breastfeeding, or who gave birth and never breastfed her child, will have a breast that is composed mainly of Type 3 and 4 lobules.⁴

During menopause, the breasts’ lobules involute and revert to Type 1 lobules regardless of the woman’s parity status. However, despite their similar cellular appearance, Type 1 lobules after menopause demonstrate differences in their epigenetics and cellular receptors between women who have and have not given birth. Cells from the breasts of women who have previously given birth are more likely to be in the resting phase (see below) and maintain genetic differences conferred upon them during pregnancy and lactation, so benefits protecting against breast cancer are retained.⁴

**Biology of Breast Cancer**

Cells replicate through mitosis which requires cells to make a complete copy of their DNA. After new DNA is made, there is a resting phase in the cell cycle that allows any errors which might have occurred during DNA duplication to be repaired. Breast lobules differ in how they proliferate and how fast they replicate their DNA. Types 1, 2, 3 and 4 lobules differ in proliferative activity, the number of hormonal receptors on the cells, as well as the time spent in resting phase for DNA error repairs. The highest level of proliferation and cell duplication is seen in the Type 1 lobule. As the lobules differentiate into Type 2 and 3 lobules, there is less proliferative activity.⁵ - ⁷

This cell proliferation is influenced by the cells’ hormonal receptors. All cells in the breast lobules have receptors for estrogen and progesterone in their nuclei, and these hormones affect breast growth during monthly menstrual cycles and pregnancy. Type 1 lobules have more of these hormonal receptors than Type
2 lobules, and likewise Type 2 lobules have more receptors than Type 3 lobules. The more receptors a cell has, the more responsive it will be to hormonal levels – and the more affected it is by carcinogens.\(^8\)

In addition, Type 1 and Type 2 lobules copy their DNA more rapidly than Type 3 lobules, thus increasing the risk of mutations. The rapid DNA copying times also decreases the time spent in the cell’s resting phase when DNA mistakes are corrected. Both of these factors contribute to an increased vulnerability to mutation and cancer. Type 1 lobules, which have the most estrogen and progesterone receptors, give rise to 85% of breast (ductal) cancers, followed by 10-15% from Type 2 lobules of lobular cancers.\(^9(p.13),10\)

After a full-term pregnancy, Type 4 lobules predominate, with more fully differentiated (mature) cells and less stem cells, a decreased number of hormonal receptors, a slower DNA copying time, and a longer resting phase – all of which decrease the likelihood that breast cancer will develop in these lobules.\(^6\)

It is known that after a woman gives birth, with or without lactation (including when the baby is given up for adoption), the Type 4 lobules regress to Type 3, but importantly, via epigenetics, these cells maintain the genetic changes that protect them from susceptibility to cancer.\(^11-13\)

**Risk Factors for Breast Cancer**

A high-risk factor is having BRCA mutations – risk by age 70 for breast cancer up to 60% (and 83% for contralateral breast cancer).\(^14\) Yet among 1781 breast cancer cases referred for gene testing, only 9.3% were positive for BRCA 1 or 2 mutations.\(^15\)

An important variable for breast cancer is the amount of estrogen to which a woman is exposed without the differentiating effect of a full-term pregnancy.\(^16\) In a review article evaluating the role of estrogen in breast cancer, the authors state, “The weight of evidence indicates that exposure to estrogen is an important determinant of the risk of breast cancer.”\(^17\) Women who start their menstrual cycles at a younger age are at greater risk, as are those who have later menopause, and those who don’t have a full term pregnancy before age 30. This is because their immature Type 1 and 2 lobules have been exposed to higher levels of estrogen (and other potential carcinogens) for longer periods of time without the protection offered by a full-term pregnancy.\(^18,19\) In fact the risk for breast cancer is 90% lower if the first full term pregnancy is at 20 years of age as compared to 30 years of age.\(^9(p. 14)\)

A potential mechanism by which exposure to higher estrogen levels could contribute to an increased risk of breast cancer is that “oxidative metabolites of estrogen have genotoxic, mutagenic, transforming, and carcinogenic potential and thus could initiate or cause the progression of the carcinogenic process in humans.” Additional studies are needed to confirm the importance of these putative estrogen metabolite factors.\(^16\)

As early as 1713, Bernadino Ramazzini noted that nuns had a higher frequency of breast cancer than other women, and his observations were confirmed in 1842 by an Italian surgeon Domenico Rigoni-Stern.\(^20\) In 1970, the relationship between pregnancy and breast cancer was demonstrated as women who had their
first child before 18 years of age were noted to have approximately one-third the risk of breast cancer as those who gave birth at age 35 years or older. Other studies have shown risk for breast cancer to be related to parity number as well as time between the first birth and subsequent births. Each subsequent birth decreases breast cancer risk by 10%. An IA before the first pregnancy carries a higher risk than those that occur after the first full term birth. Breastfeeding less than 10 years after having an IA might significantly reduce one’s breast cancer risk compared to not breast feeding less than 10 years after an IA. Breast feeding reduces risk for breast cancer, at least partly, by reducing estrogen levels. Those factors, often ignored in study designs, could explain some of the incongruent findings of epidemiologic studies examining a link between IAs and breast cancer.

Other potential risk factors for breast cancer include alcohol (which increases estrogenic exposure by decreasing the liver’s ability to clear the estrogen), cigarette smoking (by damaging DNA) especially in younger nulliparous women, contraceptive steroids and hormonal replacement therapy, and postmenopausal obesity (fat cells make estrogen). If a woman has an IA or delivers prematurely prior to 32 weeks, her breasts have been exposed to the pregnancy’s higher levels of estrogen and have formed more Type 1 and 2 lobules. However, those lobules have not yet been allowed to fully develop and mature into Type 3 and 4 lobules. The cells have begun to rapidly multiply only to have the hCG and hPL hormones, which help mature them, drastically decreased when the pregnancy is terminated. This leaves more Type 1 and Type 2 lobules that have more cells in an undifferentiated state—cells that are more susceptible to carcinogen induced cancer. A 1997 Danish study of the association of breast cancer and abortion demonstrated increasing risk of breast cancer with increasing gestational age at abortion. Using 9-10 weeks of gestation as the reference, the relative risk for breast cancer rose from 0.81 for IA done at < 7 weeks to 1.89 for IA done at > 18 weeks (more than double). The multivariate relative risk of breast cancer was increased by 13% when the IA occurred at 13-14 weeks gestation, was increased by 23% when the IA occurred at 15-18 weeks gestation, and was increased by 89% when the IA occurred after 18 weeks gestation, compared to IA at 9-10 weeks gestation. Woman lose the protection they would otherwise have had with a full-term pregnancy. It is easy to understand from a biological standpoint how an IA may contribute to the risk of breast cancer.

Unlike IAs, spontaneous abortions (SA) or miscarriages (at least in the first trimester – which 90% are) are not generally associated with breast cancer. This is due to the fact that the levels of estrogen, progesterone, and other hormones are not as elevated in those pregnancies. As noted above, with a healthy pregnancy, the mother’s ovaries increase the production of estrogen and progesterone in response to fetal hCG. However, if the ovaries do not respond to fetal hCG, the mother’s estrogen levels will not rise and will be insufficient to maintain her pregnancy. An abnormality in the embryo or fetus that causes poor production of fetal hCG can also contribute to decreased maternal estrogen. Since there is not the same estrogen hormonal surge that occurs with a healthy pregnancy, the Type 1 and 2 lobules do not undergo the rapid proliferation that is normally seen in early pregnancy. So when the pregnancy ends in miscarriage in the first trimester, there are less undifferentiated lobules left than there are when a healthy pregnancy is ended via induced abortion.
Second trimester SAs and stillbirths, like IAs in either trimester, do increase the risk of breast cancer since these occur after the woman’s breasts have been exposed to the normally elevated levels of estrogen and progesterone and the cells have undergone proliferation but have not yet differentiated. Stillbirths after 32 weeks gestation are NOT associated with increased risk. Like IAs and SAs occurring between 13- and 31-weeks gestation, premature births at < 32 weeks gestation increase risk of breast cancer. Prematurity rates are higher after IAs using mechanical dilation of the cervix.\(^9(p.17-18)\) That translates to both the current increased risk associated with those kinds of abortions and the future increased risk associated with future premature deliveries at <32 weeks gestation.

To summarize, with the exception of an early SA, a pregnant woman’s breasts will undergo proliferative changes that will increase her risk of breast cancer until she reaches 32 weeks gestation. So, whether a pregnancy terminates before 32 weeks because of a premature birth, a second-trimester spontaneous miscarriage or an induced abortion, the woman’s risk of breast cancer is increased since her breast cells have been exposed to high levels of estrogen and progesterone with pregnancy. However, after 32 weeks gestation, her risk of breast cancer will decrease due to the molecular and epigenetic changes that occur as the cells differentiate and mature, and these changes remain protective during the woman’s life.

As previously stated, nulliparous women (like many nuns) are at increased risk of developing breast cancer due to lack of the protective effect of a normal pregnancy and breastfeeding. Women who get pregnant, have one or more IAs, and never carry a child to term are at an even greater risk for breast cancer. Studies that indicate a benefit or lack of risk from IAs in nulliparous women generally are comparing women who got pregnant and had an IA to women who never got pregnant, rather than comparing them to women who got pregnant and did not have an IA.\(^9(pp. 27,45,47)\) This is particularly pertinent because according to the Centers for Disease Control and Prevention, 40% percent of abortions were to women who had never given birth, placing them at greater risk for breast cancer than those who previously gave birth. The reproductive choice of not bearing live children can have grave consequences. Just carrying a pregnancy to term and giving up the baby for adoption, reduces the mother’s risk for future breast cancer, while breastfeeding the baby further reduces the mother’s risk.

**Abortion and Adolescents**

A study which purportedly did not find an overall link between IAs and breast cancer (with significant study design flaws) still found a significant 29% increased multivariate relative risk for women 12-19 years of age.\(^25\) In 1994, Daling, et al. demonstrated the increased risk facing adolescents who have IAs. In their case-controlled study, the highest risks for breast cancer occurred in women who aborted at ages younger than 18 years, especially if the abortion took place after 8 weeks gestation. These adolescents had an 800% increased risk of developing breast cancer. More significant was the even greater increased risk for adolescents less than 18 years of age who had an IA and who also had a family history of breast cancer.\(^21\)

**Dose-Effect Response between Abortion and Breast Cancer**
Given the physiology of cancer susceptibility of the breast, it would be expected that the greater the number of pregnancy-induced proliferation periods for Type 1 and 2 lobules unchecked by natural protective maturation, the greater the risk of breast cancer. There are studies on abortion and breast cancer that do support this expectation. Almost 45 percent of abortions in the United States are repeat abortions. Also, as discussed under Risk Factors, the greater the time (dose of time) for producing more cancer susceptible type 1 and type 2 lobules prior to an IA (the greater the gestation age at which an IA is performed), the greater the risk for breast cancer.

**Worldwide Epidemiologic Studies**

International studies confirm the link between abortion and breast cancer risk. In a study of nine European countries (England, Scotland, Wales, the Irish Republic, Northern Ireland, Sweden, the Czech Republic, Finland, Denmark), abortion rates were the best predictor of breast cancer rates. The nations with the highest abortion rates had the highest breast cancer rates. In the United Kingdom, after abortion was legalized in 1967, the incidence of breast cancer increased 70 percent between 1971 and 2002. A literature review examining the worldwide breast cancer epidemic, found IA rates to be significantly correlated with breast cancer rates around the world. The authors cited studies from Bangladesh, India, Iran, Russia, Sri Lanka, and Turkey, with some finding IAs to be the “most important” or “only significant” risk factor for breast cancer.

A meta-analysis of 20 studies involving women in South Asia found an increased risk of 151% in breast cancer following abortion. When induced abortions were differentiated from spontaneous abortions (miscarriages), there was a 291% increased risk of breast cancer following induced abortion. In addition, there was a dose response noted. This is of greater significance because South Asian women are normally at lower risk for breast cancer due to lower age at first pregnancy, higher rates of breastfeeding, and lower rates of alcohol consumption.

Researchers in India are also noting an increasing incidence of breast cancer. In evaluating 188 patients who presented to one hospital, it was noted that those women who had an induced abortion had a 6.38-fold increased risk of breast cancer. Another study from India confirmed that a history of abortions contributed to a more than six-fold increased incidence of breast cancer.

The enforcement of the one-child policy in China, which includes forced abortions, has led to an increased incidence of breast cancer rates in that country, with the incidence increasing 31 percent since 1983. Jiang, et al. found increased risk from IAs (with a dose response) without looking at timing of the IA with regard to the first full term pregnancy. A case-control study from Chengu, China of 794 cases and 805 controls was undertaken to understand the possible association of birth control methods with the increasing incidence of breast cancer in Chinese women (incidence rate of 23.37 per 100,000 in 2007 rose to 28.42 per 100,000 in 2013). Researchers found that women who had an induced abortion had an increased risk of breast cancer, and those who had experienced both a medical and surgical abortion had an even greater risk, with adjusted odds ratios of 6.8 to 17.0, meaning these women had a six to seventeen-fold increased risk of developing breast cancer.
The first article noting the link between abortion and breast cancer was published in Japan in 1957. Researchers evaluated records of cancer patients at nearly all the hospitals and 420 health centers in Japan between 1953 and 1955. Abortion had been legalized in Japan in 1948, and the researchers found there was an increased risk of breast cancer in women who had experienced an IA.36

**Conflicting Studies – Importance of Design**

With all of the evidence that IAs are likely a causal factor in the rise of breast cancer around the world, it is surprising that mainstream medicine has largely ignored or denied this important link. For example, the Guttmacher Institute claims, “Exhaustive reviews by panels convened by the U.S. and UK governments have concluded that there is no association between abortion and breast cancer. There is also no indication that abortion is a risk factor for other cancers.”37 The Susan G. Komen Breast Cancer Foundation states on its website, “Research clearly shows no link between abortion (also called induced abortion) and breast cancer.”38 That foundation had referenced a 2004 meta-analysis by Beral, et al.39 to support that statement. That meta-analysis excluded 39 retrospective studies showing the link while including 13 prospective studies that did not show a link. They cited unfounded “recall bias” as the reason for excluding the retrospective studies, while including unpublished data, and using studies that made flawed comparisons.9(pp.38-42)

Authors of prospective studies and meta-analyses usually claim “recall bias” as the reason why case control studies often show the IA-breast cancer association. They claim that women who have an adverse outcome like breast cancer are more likely to claim IA than women who do not have the adverse outcome. It is true that home interviews could result in under-reporting of a stigmatizing or embarrassing event, but there is little evidence that those with cancer would under-report any more or less than those without cancer. In the 1991 study for which a national registry existed to compare retrospective interview data with prospective data, cases had 24 IAs in the registry and did not report 5 (21%) of them, but reported 7 IAs that were not in the registry. Thus, their reporting by interview was more accurate (26 of 31) than the registry (24 of 31). In the same study, controls had 59 IAs in the registry, 16 (27%) of which were not disclosed on the interview, while only 1 unregistered IA was reported.9(pp.93-100) The difference in under-reporting between the two groups is not significant (p=0.78 by 2 Tailed Fisher Exact test), and cannot explain the increased odds of 30% or more breast cancer among women who reported IAs in many case control studies from around the world.

In March 2018, the National Academies of Sciences, Engineering and Medicine (NASEM) released a report, “The Safety and Quality of Abortion Care in the United States”.40 In the consensus study highlights, the report states, “…the committee concludes that having an abortion does not increase a woman’s risk of … breast cancer.”40(p.153) However, as documented at about the same time by the Breast Cancer Prevention Institute, 60 of 76 studies worldwide which differentiated between IAs and SAs, showed an association between IA and an increased risk of breast cancer, with 36 of those studies reaching statistical significance.41 Incredibly, the authors of the NASEM report based their conclusions on only three studies.40(pp.148-149)
The lead authors of the three studies used by the NASEM are Goldacre, Brewster and Newcomb. The Goldacre study was the largest study utilized by the National Academies and involved 28,616 women with breast cancer. Despite its size, the study had serious flaws. The authors acknowledged, “data on abortions are substantially incomplete because they only include women admitted to the hospital, only include those in the care of the National Health Service, and only in the time and area covered by the study.” Since induced abortions in the United Kingdom are usually performed as outpatient procedures, the vast majority of women who had abortions during this time could have been misclassified. In addition, the authors acknowledge that “it was common for abortions to be coded without qualification of whether they were induced or spontaneous.” They also did not examine the timing with respect to maternal age at IAs and live births.

The second study referenced by the NASEM, by Brewster, et al., was flawed in the manner in which women were categorized. The authors compared childless women who aborted and women who had given birth who had also aborted with women who had not experienced an abortion, without regard to their parity. Since women who have not had children are at increased risk for breast cancer, the authors were comparing women who had abortions to a group of women that included those who already had an increased risk of breast cancer, thus muting the increased risk. They had reproductive event data on women before 1981 (start of the study period and linkable databases) yet excluded women who had IAs or SAs prior to that year (using only those whose “number of pregnancies equaled number of births.” In this study population from Scotland, abortion was used primarily as a means to delay childbearing, and 58% of abortions were performed on nulliparous younger women. Yet only 155 (5.6%) of the Brewster, et al. study population had an IA and were nulliparous. In addition, the authors stated, “The important weakness of the study relates to missing data on miscarriage and induced abortion status and potential confounding factors for a substantial proportion of the original study population.” They also excluded in situ breast cancer from their case population.

The third study (Newcomb) was quite small with only 138 cases of breast cancer in the study population. Significantly, in this study the abortion might have occurred at any time before the cancer diagnosis – even just 1 day or 1 week before the diagnosis. Since it takes an average of 8 – 10 years for cancer to be detectable, the study design is inadequate to demonstrate a relationship between abortion and subsequent development of breast cancer. In addition, the authors note that some abortions may not have been recorded in the medical records.

So without evaluating much of the world literature which includes studies that demonstrated a significant link between abortion and breast cancer, and without acknowledging the limitations of the few studies that they used, the authors of the National Academies of Sciences, Engineering and Medicine inappropriately concluded that no link exists.

A 2015 meta-analyses by Guo, et al., based on 15 prospective studies, did not find a positive association or link between IAs or SAs and breast cancer. Prospective studies examining associations between abortions and breast cancer are frequently flawed in design. The Guo meta-analysis used a number of the...
same flawed studies used in the previous meta-analyses by Beral, et al. to reach the same poorly founded conclusion. Flaws in studies used in those meta-analyses include:

1. Exclusion of *in situ* breast cancer (a form of the tested outcome)
2. Not distinguishing between IAs and SAs
3. Not distinguishing timing of IAs with respect to naturally completed pregnancies
4. Comparing women who had IAs without a subsequent live birth to women who were never pregnant, and
5. Using young women and older women in an inadequate prospective study period. Younger women are more likely to have IAs, but they are typically not followed long enough in prospective studies to see if they would develop breast cancer. Older women are less likely to have had IAs, but are more likely to develop breast cancer during a relatively short study period, because of their age.

It is the last flaw that likely plays the largest role in unfounded conclusions of a lack of link from prospective studies. Breast cancer that can result from an IA is most likely to become detectable (1 cm lump) about 8 to 10 years after its origin, yet few prospective studies follow subjects for at least 10 years after their IAs.

Another meta-analysis in 2017 by Deng, et al., that included 25 studies, concluded that “IA might increase the risk of breast cancer in parous women.” Only three of the studies they used had data about the age at which IAs were done. Both the Deng, et al. and the Guo, et al. meta-analyses included highly flawed studies by Erlandsson (2003) and Brewster (2005) that swayed their conclusion of no significant effect of IAs on breast cancer. Selection bias could easily have distorted the findings of both of those studies.

A 2018 meta-analysis by Brind, et al. of 20 studies from South Asia published after 2006 (over half published after 2012) found not only a link between IAs and breast cancer, but an increased risk with more IAs (dose effect). That, together with the meta-analysis by Huang, et al. in 2014 from China support the positive link between IAs and breast cancer. That is in spite of the fact that most of the many IAs in China in recent decades have been encouraged if not enforced by the government to limit family size. Consequently, most of their subjects had probably already had natural births, and breast fed at relatively young ages, reducing their risk of breast cancer. The argument of potential “recall bias” in case control studies is often cited by authors of prospective studies and meta-analyses of such. That “bias” has not held up to scrutiny. At the same time, those prospective studies have significant design flaws that have been largely overlooked by meta-analyses utilizing them. Studies that are properly designed are needed to clarify the conflicts that exist in the literature, but the current assessment is based on the literature to date.

**Conclusion**

Many factors increase the risk of breast cancer, some of which are not modifiable (such as early menarche, late menopause, and unfavorable genes). Induced abortion is a modifiable event, and evidence suggests
that IA prior to a full-term pregnancy contributes to the high rates of breast cancer seen around the world. Studies demonstrating a dose-related association between first or second trimester induced abortions and breast cancer, along with a plausible mechanism by which IA could cause breast cancer, strongly suggest a causal effect. The position by many in mainstream medicine to downplay or even deny a link between IA and breast cancer has been facilitated by relying on studies with flawed designs. Although further study is warranted, this potential risk from IA should be made known to adolescent females, especially those who present with an unwanted pregnancy and those with a family history of breast cancer.

Our cultural shift from family orientation and childbirth at a relatively young age (in 20’s), to one of career orientation and delayed or purposely avoided child bearing may be the greatest influence in the current breast cancer epidemic. Abortion has been a primary tool in achieving the latter aspirations. Using the adoption option and carrying unwanted pregnancies to natural birth, even without breastfeeding, could significantly reduce a woman’s breast cancer risk. The American College of Pediatricians recommends that all medical professionals include this important information in health advice given to all adolescents and their parents. This information should likewise be included in school-based health and sexuality curriculum, especially when abortion is discussed.

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A PDF of this statement is available by clicking here: **Reproductive Choices of Young Women Affect Future Breast Cancer Risk.** A PDF of a short Parent Handout will also be made available on our website.

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