

Abortion and the Link to Breast Cancer

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“It is only reasonable to conclude, from all extant evidence, that induced abortion is indeed a risk factor for breast cancer, despite the strong and pervasive bias in the recent literature in the direction of viewing abortion as safe for women.” So states Joel Brind, in a landmark review of the evidence for the abortion - breast cancer (ABC) link (2005, p. 110). Over the last forty years, this issue has been an intriguing topic of research, for both scientific and ideological reasons. But what can we conclude from the published studies currently available? According to a comprehensive meta-analysis by Beral and associates, there is no scientifically verifiable link between induced abortion and breast cancer (2004). However many researchers remain convinced that there is valid scientific evidence for this link. This paper will examine the evidence for the ABC link and show that it is verified by both the physiology of the breast as well as the published, scientifically valid data.

The biological basis for the ABC link is the cause for ninety percent of all breast cancers (Lanfranchi, 2003). This rests on two principles. The first is that the more estrogen that a woman is exposed to in her lifetime, the greater her risk of developing breast cancer. The second principle is that as a woman’s breast tissue matures it develops from type 1 and type 2 lobules (susceptible to cancer) to type 3 and type 4 (cancer resistant).

The first principle comes into play based on the total number of menstrual cycles (and therefore estrogen surges) in a woman’s lifetime. Estrogen working in the presence of progesterone causes breast cells to increase their mitotic rate. As breast cells divide more rapidly, there are more chances for copying errors and translocations which may result in cancer producing cells.

Overall, estrogen has many positive effects in the body. However it also acts as a carcinogen by indirectly damaging DNA. This DNA damage is caused by the catechol estrogen quinone which is produced by the body as a metabolite of estrogen (Lanfranchi, 2005).

The second principle is especially important as it applies to pregnancy. Any woman's breasts that have gone through puberty are composed of type 1 and type 2 lobules. Such lobules are immature and incapable of producing milk. They are also the site of ductal breast cancers, the most common type. Type 3 and 4 lobules have a different microscopic structure from types 1 and 2. They replicate their DNA more slowly, allowing more time for DNA repair. Once a woman has undergone a full-term pregnancy, seventy percent of her lobules convert to cancer resistant type 3 & 4 lobules. Hence full term pregnancy offers the woman natural protection against breast cancer (Lanfranchi, 2003).

Both of these principles make breast cancer more likely in women who have induced abortions. Pregnancy hormones have different effects at certain times during pregnancy. During the first two trimesters surges in estrogen cause the breast to enlarge by forming a greater number of type 1 & 2 lobules. Therefore pregnancies that terminate before thirty two weeks result in an increased breast cancer risk. It is important to note that while some pregnancies end in spontaneous abortion (miscarriage), such are commonly pregnancies with low hormonal levels. This situation differs from what is found in induced abortions, since most induced abortions occur in normal pregnancies. After an induced abortion, a woman is left with more type 1 & 2 lobules, providing a greater number of places for breast cancer to begin. This is the primary reason for an increased risk of breast cancer (Lanfranchi, 2005). Experimental evidence for these mechanisms has been demonstrated in rat models (Brind, 2005).

Lanfranchi in her article entitled “The Abortion Breast Cancer Link” lays out six epidemiological qualifications to draw a relationship between a causal factor and a disease. These qualifications include: the patient must be exposed to the casual factor before developing the disease, there must be similar findings in many studies, the studies must have statistical significance, studies must indicate a dose-related effect, the causal factor must have a large effect on the disease, and the causal factor must work through a biologically plausible mechanism (2003).

The body of data for the ABC link fulfills all of these criteria. Twenty eight of the thirty five worldwide studies show support for this. Seventeen of these studies have indicated the ABC link with 95% certainty that the results did not occur from chance alone. One example was the Howe study of 1989 which looked at 1,451 patients, was case controlled, and found a statistically significant association between the induced abortion rate and breast cancer rate, specifically in patients under forty (Brind, 2005). A dose effect can be seen in this research as well. A dose effect is defined as: “the more one is exposed to the risk, the higher the risk of the disease if the factor is causal” (Lanfranchi, 2005, p. 12). In regard to the ABC link, the longer a woman is pregnant, the higher her risk for developing breast cancer. This relationship was also demonstrated by Darling in a 1994 report. Other studies have also shown that a woman has a 30% increased risk of breast cancer as a result of an induced abortion. This qualifies as a large enough effect to draw a significant causal relationship, when considered along with the other evidence for this link (Lanfranchi, 2005).

With all of the biological and epidemiological evidence for the abortion breast cancer link, one wonders why there is still an intense debate. Some articles have disputed this claim. One of these in the *Lancet* in 2004, and was a meta-analysis that sought to look at all of the

studies available, and come up with a more definitive answer. The authors concluded that no such link exists (Beral et al, 2004).

However, according to several authors, the methodology of this study was flawed, with three major errors. First of all, selection bias occurred in that 14 of 41 previously published studies that were excluded for non-scientific reasons. Secondly, the authors made an assumption of recall bias, which assumes that women who have breast cancer are more likely to admit having had an abortion than women without breast cancer. This recall bias has been shown insignificant even when explicitly tested. Finally, this study used an inappropriate comparison group in comparing women who had an abortion with women who had never been pregnant, instead of properly comparing them with women who had full term pregnancies (Lanfranchi, 2003).

The *Lancet* study of 2004 is only one paper written in opposition to the ABC link. In these cases, it seems likely that ideology has overshadowed the quest for scientific accuracy. Any controversy surrounding the ABC link is rooted not in the facts themselves, but in the desire to keep the ideology of a safe abortion alive. Furton offers this summary: “Would it be cynical to think that the issue that causes this unwillingness to confront the facts squarely is abortion? There is no topic that is more divisive or that carries with it more ideological freight” (2004, p. 36).

This paper has examined the evidence for the abortion-breast cancer link and has shown that it is verifiable by both the physiology of the breast as well as by published, scientifically valid data. It is only when scientists put aside their personal biases that they can look honestly at the data and take steps towards improving women’s health.

References:

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