



Scientific Developments Relating to the Effect of Abortion on Risk of Future Breast Cancer

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Executive Summary

- Higher risk of future breast cancer in women with abortion versus pregnancy and delivery has been consistently reported in medical literature since 1970 multinational study (including the UK) by the World Health Organization (WHO).
- Oxford University epidemiologists have led the effort to give public false assurance of safety since 1982, with 3 studies on UK women, and a recent (2004) “reanalysis” by Valerie Beral et al., of worldwide data, which reported the false conclusion of no risk increase with abortion.
- The Oxford “reanalysis” was biased in selecting studies for review, including at least 4 large, scientifically invalid studies, and excluding or omitting 15 valid studies for non-scientific reasons.
- The Oxford “reanalysis” used the clinically impossible standard “of never having had that pregnancy” to which women who chose abortion are compared.
- The majority of world-wide published evidence shows abortion raises breast cancer risk beyond “never having had that pregnancy”, as we reported in our 1996 meta-analysis, published by the British Medical Association.
- Established facts of breast physiology support independent effect of abortion in raising breast cancer risk.
- The flawed methodology used for abortion, in Oxford “reanalysis” and in general, is compared with correct methodology used to identify HRT as a significant risk factor, even by the same Oxford researchers (Beral et al.) in their 2003 “Million Women Study”.
- The same inappropriate standard of comparison used for abortion would also make HRT appear not to increase risk, as demonstrated by Million Women Study results.
- Recent US experience with hormone replacement therapy (HRT) shows honest reportage of risks results in women avoiding risk, by stopping HRT use.
- Recent US drop in breast cancer shows striking results of women stopping HRT use, thus avoiding risk.
- RCOG Clinical Guideline No. 7 acknowledges lower breast cancer risk with pregnancy and delivery, yet it contradicts its own evidence with the claim of no risk increase, and violates its own Clinical Governance Advice No. 6 re: obtaining informed consent.
- RCOG Clinical Guideline No. 7 misrepresents evidence *against* their recommendation as “Evidence supporting recommendation”.
- Open disclosure of abortion’s effect in raising breast cancer risk will reduce future medical costs and demographic decline in the UK.

Focus of this report

1. This report will focus primarily on recent developments in research as published in the peer-reviewed medical literature, mostly in the UK, concerning “the relative risks of early abortion versus pregnancy and delivery” (Select Committee instruction 2(a)), specifically in relation to “evidence of (the) long-term ... adverse health” outcome(s) from abortion” (instruction 3) of breast cancer.

Background

2. Compared to pregnancy and delivery, that the risk of future breast cancer is increased in women who choose abortion has been documented in the peer-reviewed medical literature since at least 1970, with the publication of a 7-nation (including the UK) international epidemiological study in the *Bulletin of the World Health Organization*¹. That study established that early full-term—but not aborted—pregnancy confers substantial protection against future breast cancer. The authors described the “striking relation ... that women having their first child when aged under 18 have only about one-third the breast cancer risk of those whose first birth is delayed until the age of 35 years or more.” Importantly, the authors further observed that, where differences were observed regarding the frequency of abortion they “were in the direction which suggested increased risk associated with abortion—contrary to the reduction in risk associated with full-term births.” Hence, a pregnant women choosing to abort her first pregnancy was found to be putting herself at substantially higher risk of future breast cancer, compared to her choosing to complete the pregnancy.

3. Despite multiple confirmations of higher risk with abortion compared to full-term pregnancy, many epidemiological researchers have continued to “reassure” the public about the safety of induced abortion vis-à-vis breast cancer risk. This effort has been most consistently made over the years by researchers at Oxford University²⁻⁵, most recently by a group headed by Valerie Beral. The quantitative evidence to support this false claim of safety was inappropriately compiled in several important ways.

2004 Oxford “reanalysis” of worldwide data

4. While purporting to be comprehensive in having “brought together worldwide epidemiological evidence” on abortion and breast cancer, the Oxford group’s 2004 “collaborative reanalysis” in *the Lancet* comprising 52 studies⁴, was highly selective. Although 41 studies with abortion-breast cancer data had been published by that time, the total of 52 was arrived at by the exclusion or omission of 17 published studies and the inclusion of 28 studies’ worth of previously unpublished data.

5. Of the 13 studies excluded in the 2004 Beral “reanalysis,” only two were excluded for valid scientific reasons, i.e., specific information on “induced abortions had not been recorded systematically for women with breast cancer and a comparison group.” However, an additional three large published studies—should have been excluded under the same criterion, to wit: a 1997 study in Denmark⁶ in which *all* the data on legal abortions before 1973 were missing (80,000 abortions on 60,000 women), a 2001 study of Oxford women³, in which over 90% of the abortions in the study population were unrecorded, and a 2003 Swedish study⁷, in which data on all abortions after the most recent childbirth (i.e., a majority of abortions) were missing. All of these inappropriately included studies reported no increased breast cancer risk associated with induced abortion, and substantially biased the “reanalysis” in the negative direction.

6. An additional large but unpublished (subsequently published in 2005) study on women in Scotland⁵ included in the “reanalysis, should also have been excluded on the same grounds. In fact, the Scotland study by Brewster *et al.*, which was co-authored by Beral herself, distorted an otherwise excellent database and arrived at an entirely invalid result. Specifically, the database of NHS reproductive histories of women in Scotland, had been computerized in 1981. Since pre-1981 events were also incorporated into the database in 1981, such events could also be included in the Brewster study. But inexplicably, Brewster *et al.* included only “those with some reproductive events occurring before 1981, and (for whom) number of pregnancies equalled number of births—that is, no miscarriages or induced abortions before 1981.” This egregious application of selection bias eliminated over 90% of the women for whom abortion preceded the first live birth; a majority of abortions in Scotland. The resulting analysis consequently embodied such a thorough distortion of the database as to render the study’s negative result entirely invalid. I have published a detailed deconstruction of the Brewster study elsewhere⁸.

7. A bias in the Beral *et al.* “reanalysis” in the same direction (of finding no risk increase with induced abortion) is evidenced by the pattern of exclusion of valid studies. Specifically, eleven studies were excluded from the analysis for non-scientific reasons, i.e.: “Principal investigators ... could not be traced, original data could not be retrieved”, or “researchers declined to take part in the collaboration” or “judged their own information on induced abortion to be unreliable” (this last justification being particularly remarkable, as the data in question had been published in a prominent peer-reviewed journal⁹ and never retracted). Four additional studies simply never appeared in the analysis, with no justification given, even though they had been previously published as abstracts or included in other reviews.

8. That the Beral *et al.*⁴ “reanalysis” was severely biased by these 15 inappropriate and unscientific exclusions and omissions is clear from the fact that this excluded group includes all the large studies which had reported overall relative risks in excess of 2 (i.e., double the risk). The “reanalysis” therefore gives the false impression that no single, substantial study in the published record reported an overall relative risk in excess of 1.41, when in fact, four of the studies had reported overall relative risks greater than 2; one as high as 3.1¹⁰.

9. Despite the conclusion of Beral *et al.*⁴ in their 2004 reanalysis that induced abortions “do not increase a woman’s risk of developing breast cancer”, and notwithstanding the substantial selection bias inherent in the “reanalysis”, Beral *et al.* still reported a summary, statistically significant relative risk of 1.11 for all studies based on retrospective data, in contrast to a slightly negative association (RR = 0.93) for studies based on prospective data. Finding the difference between the prospective and retrospective study results “highly significant” (despite the egregious flaws in 4 of the prospective studies, described in paragraph 5 and 6 above), Beral *et al.* reported that the difference was likely attributable to “the systematic difference in reporting induced abortion between cases and controls indicated by the Swedish retrospective study”. The Swedish study¹¹ to which Beral *et al.* refer, however, was based on the presumption that women who had reported abortions which did not appear in the computerized registry had “overreported” them, i.e., imagined them to have taken place. This preposterous assumption of “overreporting” was retracted in 1998¹², a fact not mentioned in the “reanalysis”.

10. Last but not least in the list of methodological flaws of the Oxford “reanalysis” is the manner in which the relative risk estimates were inappropriately calculated, i.e., the assignment of an artificial

and clinically irrelevant comparison between choosing abortion v. the literally impossible situation of “never having had that pregnancy”⁴.

World-wide published epidemiological and biological evidence of ABC link

11. The accumulated epidemiological evidence of the independent effect of induced abortion in raising breast cancer risk was compiled by my own research team and published in the British Medical Association’s *Journal of Epidemiology and Community Health* in 1996¹³. In that report, we compiled all available published data, which dated as far back as 1957¹⁴, and documented, in our meta-analysis, an average increased risk (beyond “never having had that pregnancy”) of approximately 30 percent.

12. Denials of the independent link—indeed any link—between induced abortion and breast cancer notwithstanding, even the largest, most often cited study⁶ to claim “induced abortions have no overall effect on the risk of breast cancer” shows a clear elevation in risk (beyond no pregnancy at all) among women with abortions beyond the first trimester. Thus, Melbye *et al.*, in their nationwide prospective study on Danish women¹² reported a clear, significant trend of increasing risk with gestational age at abortion, the risk increase reaching 89% beyond 18 weeks’ gestation. The same Danish research group confirmed this trend for premature livebirths, reporting more than a twofold risk increase for livebirths delivered before 32 weeks’ gestation¹⁵. There is no difference between a premature livebirth and an induced abortion, in terms of hormonal effects on the breasts and the future risk of breast cancer. This effect is well understood in terms of the lack of breast maturation into cancer-resistant lobules before 32 weeks, and argues for a careful reevaluation of the safety of late term abortions (before as well as after 24 weeks) and the regulations regarding such abortions.

13. Since induced abortion has also been established as a risk factor for subsequent premature and very premature births (i.e., before 32 weeks)¹⁶, it secondarily increases the risk of breast cancer in such women.

14. In addition to the effect of abortion in raising future breast cancer risk by abrogating the protective effect of full-term pregnancy, is the well-documented independent effect of abortion in raising risk even beyond that of “never having had that pregnancy” (see paragraph 11, above). This effect is attributable to the growth-stimulating effects of sharply elevated levels of oestrogen and progesterone during pregnancy; these hormones causing the formation of greater numbers of immature type 1 and 2 breast lobules, where almost all breast cancers start¹⁷. (During pregnancy, milk-producing lobules only mature to cancer-resistant type 3 and 4 lobules after 32 weeks gestation, resulting in full-term pregnancy’s long-term protective effect.)

Comparison of abortion with another avoidable risk: Hormone-replacement therapy (HRT)

15. The absurdity of the claim of abortion’s safety vis-à-vis “never having had that pregnancy” is underscored by comparison of this flawed methodology with the correct methodology used to quantify the breast cancer risk increase attributable to combination (i.e., oestrogen-progestogen) hormone replacement therapy (HRT). This therapy is used by many postmenopausal women, and its breast cancer danger was documented in another large study (among others)—“the Million Women Study”—published by Beral’s group¹⁸ in *The Lancet* in 2003.

16. In striking contrast to the comparison made in the 2004 “reanalysis” on induced abortion and breast cancer, the 2003 HRT study appropriately compared postmenopausal women taking HRT to women of

the same age not taking HRT. As other groups have amply confirmed, the risk of future breast cancer was significantly increased—in fact, doubled—among combination HRT users.

17. Had Beral and co-workers instead applied an inappropriate standard for HRT risk as they had applied in the case of induced abortion, little if any risk increased would have been observed. Specifically, the analogous comparison would have been between HRT users and premenopausal women of comparable age; women in the position of “never having had that” menopause. The analogy between the two types of methodology is strictly correct, as both full-term pregnancy and menopause have similar effects on a woman’s future breast cancer risk: They lower risk in inverse proportion to the woman’s age at which the event (full-term pregnancy or menopause) occurs.

18. The proof that inappropriate comparison of postmenopausal women who took HRT to comparably aged premenopausal women, would have resulted in little or no increased risk, actually appears in the Million Women Study¹⁸. Specifically, “among never users of HRT the relative risk of invasive breast cancer was ... 0.63 (95% CI 0.58-0.68) (i.e., significantly reduced by an average 37%) for postmenopausal, compared with premenopausal women.” Hence, the (higher) risk of premenopausal women was similar to the risk of postmenopausal women who used HRT. That late menopause increases the risk of breast cancer is well established, and attributable to the risk-increasing effect of ovarian sex hormones, whether made by a woman’s own ovaries (before the menopause), or taken exogenously (as HRT).

19. The HRT-breast cancer link is of particular relevance to the abortion-breast cancer link in that both are avoidable risks, and recent events in the US have illustrated how women take these risks seriously when accurate information is given. A large, 5-year randomized trial—the Women’s Health Initiative study¹⁹—began in 1999 to test HRT’s putative effect in lowering heart attack risk—was terminated in 2002 after only 3 years because heart attack risk was found to be significantly elevated, rather than reduced. News of the study’s termination included confirmation of HRT’s carcinogenic effect on the breasts, which most women on HRT found out for the first time. Over the next 2-3 years, use of HRT declined by 70%.²⁰

20. Strikingly, within a year of the WHI study’s termination, the breast cancer incidence rate for women in the US over 50 years old began to decline in parallel with the decline in HRT use, the extent of the decline reaching almost 12% within three years. That the breast cancer incidence decline was due to the massive cessation of HRT use is widely accepted as the only likely explanation²⁰.

RCOG gives inaccurate and contradictory clinical advice to abortion practitioners

21. Importantly, The RCOG Clinical Guideline No. 7²¹ on “the care of women requesting induced abortion” acknowledges the Beral reanalysis⁴ as “a major systematic review ... which lent further support to these conclusions (of no association between induced abortion and breast cancer risk)”, despite the fact that the very first line of text in the Beral reanalysis states: “Pregnancies that result in a birth are known to reduce a woman’s long-term risk of developing breast cancer”.

22. Hence, false reassurance of the safety of abortion vis-à-vis future breast cancer risk is embodied in the RCOG’s Clinical Guideline No. 7²¹ (Recommendation 16.7): “Induced abortion is not associated with an increase in breast cancer risk.” This advice, if taken by abortion practitioners when advising patients and obtaining their consent, results in the flagrant violation of the RCOG’s own Clinical Governance Advice No. 6²² which describes the requirements for “obtaining valid consent.” This

Advice clearly advises the practitioner: “You should already have: ... discussed with the patient the risks and benefits of having no treatment. These points should be reinforced at the time of signing of the consent form.” As it is unequivocal that “having no treatment”, i.e., carrying the pregnancy to term, results in a lower long-term risk of breast cancer, the practitioner clearly has a duty to inform the patient of this fact not once, *but twice*, in order to obtain valid consent for abortion.

23. Yet another stark contradiction in the RCOG Clinical Guideline on induced abortion practice²¹ is the recommendation (No. 16.7) of no association between induced abortion and breast cancer risk in the face of the same Guideline’s acknowledgement, under “Evidence supporting recommendation 16.7” of our 1996 meta-analysis¹³. Specifically, the Guideline cites our summary conclusion “that induced abortion was a significant, independent risk factor for breast cancer, with an odds ratio of 1.3 (95% CI 1.2-1.4)”, and further acknowledges that our review had been “carefully conducted”, “and that the Brind *et al.* study had no major methodological shortcomings and could not be disregarded.” Clearly, our review constitutes credible evidence *against* rather than “supporting” the Guideline’s unequivocal recommendation of induced abortion’s being “not associated with an increase in breast cancer risk.”

Conclusions.

24. It is therefore likely that providing full and accurate information on the effect of abortion in raising future breast cancer risk substantially, compared to pregnancy and delivery, is likely to reduce the abortion rate substantially. Such a reduction in numbers of a procedure which is not safe for women as originally thought in 1967, will provide benefits in terms of improved future health for British women, lower costs for cancer treatment and treatment of other sequelae, as well as amelioration of the alarming demographic decline evident in the UK in recent years.

Respectfully submitted this 30th day of August, 2007 in evidence presented to the Select Committee on Science and Technology of the United Kingdom Parliament, by:

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