Chapter 7

Biology and epidemiology confirm the abortion-breast cancer link

KEY POINTS

- Once pregnant, if a woman chooses to maintain her pregnancy and achieves a full-term pregnancy, she will *decrease* her risk of breast cancer.
- Never becoming pregnant *increases* breast cancer risk. If a woman undergoes an induced abortion she may remain childless, a condition that also *increases* her breast cancer risk.
- Delaying pregnancy after age twenty increases breast cancer risk. If a woman undergoes an induced abortion and brings a subsequent pregnancy to term, she has effectively delayed that full-term pregnancy, thereby *increasing* her risk of breast cancer.
- Every full-term pregnancy after the first further *decreases* the
 risk of breast cancer. If a woman has already had a full-term
 pregnancy and then chooses to abort a subsequent pregnancy she
 loses the risk reduction that an additional full-term pregnancy
 would have afforded her, thereby *increasing* her risk.
- The use of instruments such as dilators during an abortion increases a woman's risk of having a premature delivery in future births. If that premature delivery occurs before 32 weeks gestation, she will have an *increased* risk of breast cancer.

Introduction

Breast cancer is the leading cause of cancer death of women worldwide. One in ten of all new cancers in the world are female breast cancers. North America has the highest incidence of breast cancer in the world. The age-adjusted rate for non-invasive breast cancer, ductal carcinoma *in-situ* (DCIS), increased 660 per cent between 1973 and 2000, while the rate for invasive breast cancer increased 36 per cent. Ductal carcinoma *in-situ* differs from invasive cancer by the location of all the cancer cells that remain within the milk duct. By definition the *in-situ* cancer cells have not penetrated or invaded the wall of the milk duct (the basement membrane). Should these cells invade the basement membrane, the cancer would be classified as invasive breast cancer, which is able to spread throughout a woman's body. DCIS is the earliest detectable form of breast cancer, which is highly curable (97-99 per cent). Invasive breast cancers commonly arise in *in-situ* cancers. Breast cancer is the leading cause of cancer deaths of women between twenty and 59 years old.

For centuries it was known that remaining childless increased a woman's risk for breast cancer. Conversely, it was also known that pregnancy was protective. In 1743, Ramazzini of Padua observed that there was an increased amount of breast cancer among nuns. In 1842, a hundred years later, Rigioni-Stern noted a threefold increase risk of breast cancer among nuns. Nuns were largely childless whereas the rest of the population had pregnancies early on in their reproductive lives. Yet it was not until the 1980s that the normal physiology of breast development and maturation during pregnancy, which accounts for those reproductive risks, became clear. In the first decade of the 2000s, with advances in technology, scientists learned the genetic changes that occur in breast cells that explain why pregnancy affords protection from cancer.

It is well known that different pregnancy outcomes lead to changes in the rates of breast cancer among women. There are various long-established insights on the relation between pregnancy and breast cancer.

Delayed First Pregnancy

The longer a woman waits to have her first full-term pregnancy (FFTP), the higher her risk of breast cancer as her immature, cancer-vulnerable breast tissue is exposed to carcinogens for a longer duration. A woman who remains childless or has her FFTP when she is more than 30 years of age has a 90 per cent higher

risk of breast cancer than a woman who has her first child before the age of twenty.¹

For each year a woman delays pregnancy after age twenty, she has a five per cent increase in risk for pre-menopausal breast cancer and a three per cent increase in risk for postmenopausal breast cancer.² For example, having an induced abortion at age twenty followed by a full-term pregnancy at age thirty would increase her risk of pre-menopausal breast cancer by 50 per cent. Other studies have shown that breast cancer risk increases 0.7 per cent for each year that subsequent births are delayed after her first birth.³ Yet another study has shown that if a woman has a pregnancy and lactates within five years after an abortion, her risk will be twenty per cent less than if she waits ten or more years to lactate for the first time.4

Increased Number of Pregnancies

For each pregnancy that the woman has subsequent to her first, her risk of breast cancer will decrease another ten per cent.⁵

Abortion and Subsequent Premature Births

Two large meta-analyses show that induced abortion increases a woman's risk of premature delivery.6 Also, the more induced abortions a woman has, the higher her risk of subsequent premature births. In 2006, the Institutes of

Bland K and Copeland E, eds. The Breast: Comprehensive Management of Benign and Malignant Diseases. Philadelphia: Saunders El Sevier, 4th edition, 2 vols, 2009, vol 1, chap. 19: p. 335, Table 19-1.

Clavel-Chapelon F and Gerber M. Reproductive factors and breast cancer risk: do they differ according to age at diagnosis? Breast Cancer Research and Treatment 2002 March; 72(2): 107-15.

Decarli A, La Vecchia C, Negri E and Franceschi S. Age at any birth and breast cancer in Italy. International Journal of Cancer 1996 July; 67(2): 187-9.

Daling JR, Malone KE, Voigt LF, White E and Weiss NS. Risk of breast cancer among young women: relationship to induced abortions. Journal of the National Cancer Institute Cancer Spectrum 1994 November; 86(21): 1584-92.

Lambe M, Hsieh C, Chan H, Ekbom A, Trichopoulos D and Adami H. Parity, age at first and last birth, and risk of breast cancer: a population study in Sweden. Breast Cancer Research and Treatment 1996 January; 38(3): 305-11.

Shah PS and Zao J. Induced termination of pregnancy and low birth weight and preterm birth: a systematic review and meta-analyses. BJOG 2009 October; 116(11): 1425-42; Swingle HM, Colaizy TT, Zimmerman MB and Morriss FH. Abortion and the risk of subsequent preterm birth: a systematic review with meta-analyses. Journal of Reproductive Medicine 2009 February; 54: 95-108.

Rooney B and Calhoun BC. Induced abortion and risk of later preterm births. Journal of the American Physicians and Surgeons 2003 Summer; 8(2): 46-9.

Medicine listed induced abortion as an immutable cause of premature birth.8

Except for those that end in spontaneous abortion, whatever the length of her pregnancy, in the first 32 weeks she will have changes in her breast tissue that will increase her risk of breast cancer. When a woman gives birth naturally, it takes many hours to dilate the cervix for birth. During an abortion the cervix is forcibly dilated and subjected to injury. Owing to the use of instruments such as dilators during an abortion she may deliver a subsequent pregnancy prematurely. If the premature delivery is before 32 weeks, she will have an increased risk of breast cancer. Approximately three per cent of all premature deliveries occur before 32 weeks. Approximately 12.5 per cent of all births are before 37 weeks and are considered premature. 10

Breast Tissue Changes in First Pregnancy

When a woman becomes pregnant for the first time, her immature and cancer-vulnerable breast tissue matures into cancer-resistant tissue. Approximately 85 per cent of her breast will become fully mature Type 4 lobules, which contain the first milk, colostrum. After weaning, theses lobules regress to Type 3 lobules, which are also cancer-resistant. This biological change accounts for the recognized fact that never having a full-term pregnancy (nulliparity) increases a woman's risk of breast cancer (as the experience of nuns demonstrates). After a full-term pregnancy, only fifteen per cent of her breast tissue remains susceptible to forming cancer. It is the genetic changes that occur in the breast lobules during a full term pregnancy that give lifelong protection. Molecular biologists have also determined that progenitor or stem cells in the breast do not become terminally differentiated (reach their full potential growth or maturity) until they have undergone

⁸ Alexander GR. Appendix B: prematurity at birth: determinants, consequences, and geographic variation. In *Preterm Birth: Causes, Consequences and Prevention*. Ed. Behrman RE and Butler AS. Washington, DC: National Academies Press (US), 2007: p. 625.

⁹ Ibid., p. 616.

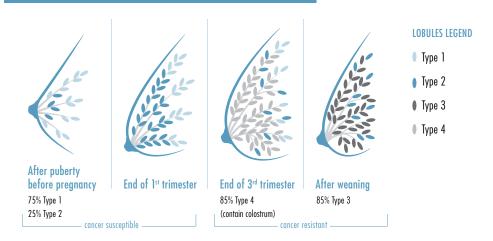
¹⁰ Ibid

Russo J, Rivera R and Russo IH. Influence of age and parity on development of the human breast. Breast Cancer Research and Treatment 1992; 23: 211-8; Russo H, Yang, Russo. Chapter 1: developmental, cellular, and molecular basis of human breast cancer. Journal of the National Cancer Institute Monograph 2000; 27: 17-37.

Russo J, Balogh GA and Russo IH. Full-term pregnancy induces a specific genomic signature in the human breast. Cancer Epidemiology, Biomarkers, and Prevention 2008 January; 17: 51-66; Verlinden I, Gungor N, Wouters K, Janssens J, Raus J and Michiels L. Parity-induced changes in global gene expression in the human mammary gland. European Journal of Cancer Prevention 2005 April; 14(2): 129-37.

the environment of pregnancy and have lactated.¹³ A group of international researchers has found the numbers of these stem cells are lowest in women who have give birth in their early twenties while they are highest in women with high risk for breast cancer, such as those who have inherited a mutated BRCA gene.14

Breast lobule maturation during first pregnancy



Early Termination of Pregnancy (through Prematurity or Abortion) and Increased Risk of Breast Cancer

Several studies have shown that prematurity before 32 weeks gestation increased breast cancer risk.¹⁵ In fact, the biologic mechanism for premature delivery, induced abortion and second-trimester miscarriage as causes for increased risk of breast cancer is the same mechanism for all three: Abortion, premature delivery and second-trimester miscarriage all leave the breast with more places for cancers to start when the pregnancy ends. The woman's breasts have been exposed to the same pregnancy hormones (estrogen, progesterone, human chorionic gonadotropin (hCG)

Boecker W, Weigel S, Heindel W and Stute P. The normal breast. In Preneoplasia of the Breast: A New Conceptual Approach to Proliferative Breast Disease. Ed. Werner Boecker. Munich: Elsevier Saunders, 2006: 1-28.

Choudhury, S et al. Molecular Profiling of Human Mammary Gland Links Breast Cancer Risk to a p27+ Cell Population with Progenitor Characteristics. Cell Stem Cell 2013 July; 13, 117-130.

Melbye M, Wohlfahrt J, Andersen AMN, Westergaard T and Andersen PK. Premature delivery and breast cancer risk. British Journal of Cancer 1999 April; 80: 609-13; Vatten LJ, Romundstad PR, Trichopoulos D and Skjaerven R. Pregnancy related protection against breast cancer depends on length of gestation. British Journal of Cancer 2002 July; 87: 289-90.

and human placental lactogen hPL) all of which lead to the same breast changes. Elevated levels of estrogen and progesterone, stimulated by hCG, cause more cancer-vulnerable breast tissue to form. It is only *after* 32 weeks of gestation that the elevated levels of hPL, in concert with other pregnancy hormones, allow the full maturation to cancer-resistant breast tissue to occur. Therefore, whether the pregnancy ends before 32 weeks with a premature birth, a second-trimester miscarriage (which generally will have normal hormonal levels) or an induced abortion, breast cancer risk is increased.

THE INDEPENDENT LINK BETWEEN BREAST CANCER AND INDUCED ABORTION

There is evidence that induced abortion before 32 weeks gestation in and of itself increases breast cancer risk when other factors such as age at first birth are controlled for in the studies. The various types of evidence supporting an independent link will be addressed in this section.

For a study concerning breast cancer risk to be accurate, all known risks must be controlled for in the case group or cancer group, and the control group or non-cancer group, which is used for comparison. This is the basis for case-controlled studies. For instance, if a study was to look at whether candy increased breast cancer risk or not, the case group and the control group would have to be similar in all other known cancer risks. Thus if the case group had more women in it with a family history of breast cancer than the control group, the study would come under merited criticism if it found that candy increased breast cancer risk. In other words, the case group and control group would have to be comparable in all known risks for the study to be valid.

There have been several recent studies from groups of scientists all over the world that have controlled for induced abortion as a risk factor for breast cancer. For example, an American study looking at oral contraceptives as a risk for subtypes of breast cancer also controlled for induced abortion. ¹⁶ In the discussion section of the study, it reported that as in "previous studies, induced abortion was found to be a risk for breast cancer." The researchers included the chief of the Hormonal and Reproductive Section in the Division of Epidemiology at the National Cancer Institute. Another

Dolle JM, Daling JR, White E, et al. Risk factors for triple-negative breast cancer in women under the age of 45 years. Cancer Epidemiology, Biomarkers and Prevention 2009 April; 18(4): 1157-66.

recent study, this one from Iran, found that induced abortion carried a 62 per cent increased risk of breast cancer. ¹⁷ A paper from China looking into risk factors associated with sub-types of breast cancer found that induced abortion increased breast cancer risk by 26 per cent¹⁸. A recent Turkish study has also found induced abortion to be a risk for breast cancer, with an increased risk of 66 per cent. 19 In the discussion section of this paper the authors noted that their finding was consistent with previous findings in the world's literature concerning induced abortion. Another recent study, this time in Armenia, found an increased risk of breast cancer of 77 per cent for women who had had one to three induced abortions and 95 per cent for women who had had four to ten abortions.²⁰ A study in China found an increased risk of 52 per cent among post-abortive women, even when adjusting for other relevant reproductive factors. It too supports the existence of an independent link. The authors also studied pre- and postmenopausal women separately and found that the increase in risk for postmenopausal women was 82 per cent.21 The study, like many others, also demonstrated a dose-response relationship regarding increase in risk (with an increased risk of 150 per cent for three or more abortions), which, as will be discussed later, is one of the Bradford-Hill criteria for establishing causality.

This year the authors of a Danish cohort study²² reported that they "did

Naieni KH, Ardalan A, Mahmoodi M, Motevalian A, Yahyapoor Y and Yazdizadeh B. Risk factors of breast cancer in north of Iran: a case-control in Mazandaran province. Asian Pacific Journal of Cancer Prevention 2007; 8(3): 395-8. The finding was statistically significant. Xing P, Li J and Jin F. A case-control study of reproductive factors associated with subtypes of breast cancer in Northeast China. Medical Oncology 2010 September; 27(3): 926-31. Another separate Chinese study, that unfortunately did not distinguish between spontaneous and induced abortions, nevertheless showed an increased risk of 120 per cent for one to two abortions and 662 per cent for three or more abortions: Zeng Y, Xu M, Tan S and Yin L. Analysis of the risk factors of breast cancer. Journal of Southern Medical University 2010; 30(3): 622-3.

Ozmen V, Ozcinar B, Karanlik H, et al. Breast cancer risk factors in Turkish women - a university hospital based nested case control study. World Journal of Surgical Oncology 2009 April; $7(3\overline{7})$: 37-44. The finding was statistically significant.

Khachatryan L, Scharpf R and Kaan S. Influence of diabetes mellitus Type 2 and prolonged estrogen exposure among women in Armenia. Health Care for Women International 2011 October; 32(11): 953-71.

Jiang AR, Gao CM, Ding JH, et al. Abortions and breast cancer risk in premenopausal and postmenopausal women in Jiangsu province of China. Asian Pacific Journal of Cancer Prevention 2012; 13: 33-5. http://www.apjcpcontrol.org/page/popup_paper_file_view.php?pno=MzMtMzUgMTIuMiZrY29kZT0yNzAxJmZubz0w&pgubun=i . The finding was statistically significant.

Brauner C, Overvad K, Tjonneland A and Attermann J. Induced abortion and breast cancer risk among parous women: a Danish cohort study. Acta Obstetricia et Gynecologica Scandinavica 2013. http://onlinelibrary.wiley.com/doi/10.1111/aogs.12107/abstract.

not find evidence of an adverse effect of induced abortion on breast cancer risk in parous women overall..." However, their study is seriously flawed, since they only looked at women between 50 and 65 years of age, whom they followed for "approximately 12 years." Given that the average age at abortion in Denmark is 27, and the average age of the women recruited was 57, and that the study design excluded all women with previous cancers, all of the postabortive women who developed cancer within 25 to 30 years of their abortion were excluded. We also know that parous women are less likely to get cancer, especially if they have children soon after an abortion. Another study this year, from India, 23 shows induced abortion as its strongest risk factor. Finally, just as we go to press, a statistically-significant study from Bangladesh reports a more than twenty times increased risk of breast cancer after induced abortion.²⁴

If scientists worldwide did not know and agree that induced abortion is a known risk for breast cancer, they would not refer to it as commonly accepted in their studies and analyses. Induced abortion is specifically acknowledged as a known risk factor in the performance of such studies, as well as in the methodology and discussion sections of the published papers. This is because induced abortion is now a commonly-accepted risk factor for breast cancer except in North America, where it is denied chiefly for political reasons.

The Biological Evidence

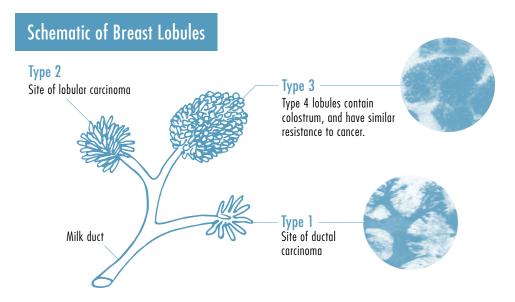
An abortion does not turn back the clock and make a pregnant woman "unpregnant". As soon as conception occurred and before implantation, the embryo released the hormone hCG (human chorionic gonadotropin), which immediately caused the mother's ovaries to produce higher levels of estrogen and progesterone and change her breasts. That earliest sign of pregnancy, sore and tender breasts, is the result of the multiplication of breast cells to produce more breast tissue in preparation for breastfeeding. Abortion cannot remove those newly made cells that will remain cancer-vulnerable for her lifetime or until she completes a pregnancy past 32 weeks. If that same pregnant woman chooses to carry her pregnancy to term, she will have the lifelong benefit of a lower breast cancer risk. These are the undisputed biological facts that cause abortion to be a risk for breast cancer.

There are well-documented, physiological changes that occur in the

Kamath R, Mahajan KS, Ashok L and Sanai TS. A study on risk factors of breast cancer among patients attending the tertiary care hospital, in Udupi district. Indian Journal of Community Medicine, 2013; 38(2): 95-99.
 Jabeen S, Haque M, Islam J, Hossain MZ, Begum A, Kashem MA. Breast cancer and some epidemiological risk factors: A hospital based study, J Dhaka Med Coll 2013; 22(1): 61-6.

mother's breast with a normal pregnancy and result in a lowering of her breast cancer risk if the pregnancy goes past 32 weeks.²⁵ This reduction is due to the maturing hormones produced by the fetus and placenta (after birth) in preparation for breastfeeding.

An overview of breast physiology during pregnancy is necessary to facilitate understanding of the physiological evidence supporting the abortionbreast cancer link. A lobule is a unit of breast tissue consisting of milk glands and ducts that carry the milk toward the nipple. Prior to a first full-term pregnancy, the breast is about 75 per cent Type 1 and 25 per cent Type 2 lobules where ductal and lobular breast cancers form respectively. By the end of the pregnancy, the breast is about 85 per cent fully matured to cancer-resistant Type 4 lobules and only fifteen per cent immature, cancer-vulnerable lobules, thereby reducing the mother's future risk of breast cancer.



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During a pregnancy, the absolute numbers of these lobules also increase as the breast doubles in volume with an increase in number of lobules and

Hsieh CC, Wuu J, Lambe M, Trichopoulous D, Adami HO and Ekbom A. Delivery of premature newborns and maternal breast cancer risk. The Lancet 1999 April; 353(9160): p. 1239.

a decrease in stroma (the surrounding connective tissue).²⁶ A premature delivery before 32 weeks for any reason, whether physician-induced or because of an incompetent cervix (which is commonly due to previous abortions) or any other natural cause, increases breast cancer risk, because the breast has already responded to the hormones estrogen and progesterone, which are produced by the ovaries, fetus or placenta in response to fetal-placental secretion of human chorionic gonadotropin (hCG)²⁷. These hormones cause an increase in breast tissue, Type 1 and 2 lobules, where cancers start. Only after 32 weeks' gestation does the fetal-placental hormone human placental lactogen (hPL) and other hormones enable the breast to fully mature its breast lobules into Type 4, making them cancer-resistant. This cancer resistance is the result of known permanent genetic changes that occur within the breast cells' genome, providing the molecular basis for the protective effect of a fullterm pregnancy.²⁸ An induced abortion before 32 weeks has the same physiological effect on the breast. Its only difference from premature delivery is that the fetus is delivered dead rather than alive.

The breast physiology described above explains the independent breast cancer risk that induced abortions cause in addition to losing the protective effect the mother could have gained by carrying her pregnancy to term. The longer the gestation up to 32 weeks before the induced abortion, the higher the mother's breast cancer risk because she has developed more places for cancers to start.29

Another reason why induced abortion causes an increased risk of breast cancer is its secondary effect of increasing the rate of premature birth in the mother's subsequent pregnancies. Any premature delivery before 32 weeks will increase breast cancer risk through the same biological mechanism that causes induced abortion to increase breast cancer risk. With the stimulation by the pregnancy hormones of estrogen and progesterone, the numbers of cells that are immature and cancer vulnerable are markedly increased in number. In other words, there are more places (cells) for cancers to start. It is only in the hormonal environment that occurs after the first 32 weeks of pregnancy—during which time hPL (human placental lactogen) has been very elevated-that these cells mature through specific genetic changes, which cause them to become cancer resistant. There have been two large

Russo J, Lynch H and Russo IH. Mammary gland architecture as a determining factor in the susceptibility of the human breast to cancer. Breast Journal 2001 September; 7(5): 278-91.

Vatten, Romundstad, Trichopoulos and Skjaerven. See n. 15; Melbye et al. 1999. See n. 15.

Russo, Balogh and Russo. See n. 12. 28

Melbye M, Wohlfahrt J, Olsen JH, et al. Induced abortion and the risk of breast cancer. NEJM 1997 January; 336: 81-5.

meta-analyses confirming that induced abortion increases a woman's risk for premature delivery.³⁰

Even pregnancies ending after 32 weeks but before 40 weeks gestation do not offer the maximum protection afforded by a full-term pregnancy.³¹ Women who remain childless (nulliparous) have an increased risk for breast cancer because they have lifelong, immature, cancer-susceptible lobules, Types 1 and 2.

Without the maturing effects of hPL to form cancer-resistant Type 4 lobules, any mutated or clinically dormant cancer cells present in the mother's breasts before her pregnancy may become cancerous or start to grow under the influence of elevated levels of estrogen and progesterone, causing proliferation and the genotoxic estrogen metabolite 4 hydroxy catechol estrogen quinone. Estrogen levels increase 2000 per cent by the end of the first trimester. This explains why women who have their first child late in life will also have a higher risk of breast cancer. It is because of the additional time that has elapsed for mutations to have formed before pregnancy. This also explains the transient increase in the risk of breast cancer in women who have their first children late in their reproductive life.

The more menstrual cycles a woman has (whether owing to an early age at menarche [first period] or a late menopause), the longer her exposure to estrogen and progesterone during her menstrual cycles and, therefore, the higher her risk of developing breast cancer. Irregular periods during the first five years after menarche lower risk as there are fewer cycles and many are anovulatory (no egg produced), thus exposing a women to less estrogen and progesterone. Breastfeeding lowers a woman's risk for breast cancer because she will often stop menstruating and her cycles can be anovulatory (no ovulation).

Most spontaneous abortions (miscarriages) do not carry the same risk as induced abortions because most spontaneous abortions occur before three months gestation and are therefore associated with low levels of the pregnancy hormones needed for breast development. This in turn is due to an abnormality in the fetal-placental unit or the mother's ovaries, which then results in a spontaneous abortion (miscarriage). 32 Women who miscarry often

³⁰ Swingle, Colaizy, Zimmerman and Morriss. See n. 6; Shah and Zao. See n. 6.

³¹ Vatten, Romundstad, Trichopoulos and Skjaerven. See n. 15; Melbye et al. 1999. See n. 15.

Kunz J and Keller PJ. HCG, HPL, oestradiol, progesterone and AFP in serum in patients with threatened abortion. BJOG 1976 August; 83: 640-4.

report having "not felt pregnant" owing to these low hormonal levels.

Epidemiological Studies

There were seventeen studies that showed a statistically significant increased risk of breast cancer before 1996, when Brind published a quantitative meta-analysis of them in the *British Journal of Epidemiology and Community Health*.³³ The meta-analysis excluded studies that did not differentiate between induced and spontaneous abortion and showed that seventeen of the 23 studies indicated a positive association, ten of which were statistically significant, meaning that there was a 95 per cent certainty that those studies did not show the association by chance. Since that time, many other studies have been published that show a statistically significant risk.³⁴

Brind J, Chinchilli VM, Severs WB and Summy-Long J. Induced abortion as an independent risk factor for breast cancer: a comprehensive review and meta-analysis. Journal of Epidemiology and Community Health 1996 October; 50(5): 481-96.

Community Health 1996 October; 50(5): 481-96.

34 Those studies that show a statistically significant link between abortion and breast cancer are as follows: Segi M, Fukushima I, Fujisaku S, et al. An epidemiological study of cancer in Japan. GANN 1957; 48(Supplement): 1-43; Rosenberg L, Palmer JR, Laufman DW, Strom BL, Schottenfeld D and Shapiro S. Breast cancer in relation to the occurrence and time of induced and spontaneous abortion. American Journal of Epidemiology 1988 May; 127(5): 981-9; Howe HL, Senie RT, Bzduch H and Herzfeld P. Early abortion and breast cancer risk among women under age 40. International Journal of Epidemiology 1989 June; 18(2): 300-4; Laing AE, Demenais FM, Williams R, Kissling G, Chen VW and Bonney GE. Breast cancer risk factors in African-American women: the Howard University Tumor Registry experience. Journal of the National Medical Association 1993 December; 85(2): 931-9; Laing AE, Bonney GE, Adams-Campbell L, et al. Reproductive and lifestyle factors for breast cancer in African-American women. Genetic Epidemiology 1994; 11: 285-310; Daling et al. 1994. See n. 4; Daling JR, Brinton LA, Voigt LF, et al. Risk of breast cancer among white women following induced abortion. American Journal of Epidemiology 1996 August; 144(4): 373-80; Newcomb PA, Storer BE, Longnecker MP, Mittendorf R, Greenberg ER and Willett WC. Pregnancy termination in relation to risk of breast cancer (United States). Cancer Causes and Control 1997 November; 8(6): 841-9; Nishiyama F. The epidemiology of breast cancer in Tokushima prefecture. shikoku Ichi 1982; 38: 333-43 (in Japanese); Le MC, Bachelot A, Doyon F, Kramar A and Hill C. Oral contraceptive use and breast or cervical cancer: preliminary results of a French case-control study in Greece are alougogy. Eds. Wolff JP and Scott JS. Amsterdam: Elsevier, 1984: 139-47; Lipworth L, Katsouyanni K, Ekbom A, Michels KB and Trichopoulos D. Abortion and the risk of breast cancer: a case-control study in Greece. International Journal of Cancer 1995 April; 61(2): 181-

There are now 56 studies that show a positive association between abortion and breast cancer, of which 35 are statistically significant. The first was published in Japan and showed a three-fold increase in the risk of breast cancer in women with a history of induced abortion.³⁵ In 2012, three studies were published, two from China and one from France. All three were statistically significant, and all three supported the ABC (abortion-breast cancer) link.³⁶ In one Chinese study it was found that women who had a previous induced abortion experienced a 58 per cent increased risk of breast cancer even when factors such as number of full-term pregnancies and age at first birth had been controlled. The study also showed a dose-response relationship between abortion and breast cancer risk (with an increased risk of 33 per cent for one abortion, 76 per cent for two abortions and 165 per cent for three or more abortions), and did not show any significant increase in risk with spontaneous abortion. In the other Chinese study,³⁷ one abortion increased breast cancer risk by 150 per cent. The French study showed that an abortion before a full-term pregnancy increased risk by 70 per cent.

Ecological Epidemiological Studies

Ecological epidemiological studies examine trends in large populations based upon comparisons using statistical records kept by governmental agencies.

In 2007 an actuary, Patrick Carroll, published "The breast cancer epidemic: modeling and forecast based on abortion and other risk factors" in the Journal of American Physicians and Surgeons. He found that abortion was the greatest predictor of breast cancer incidence in nine European countries: England, Wales, Scotland, Northern Ireland, the Irish Republic, Sweden, the Czech Republic, Finland and Denmark. Using computerized abortion and breast cancer registries, he found that the greatest predictor of future breast cancer incidence was a nation's abortion rates. Within the United Kingdom, the constituent nations that have the highest abortion

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³⁵ Segi et al. 1957. See n. 34: 1-63.

Jiang et al. See n. 21; Lecarpentier J, et al. Variation in breast cancer risk associated with factors related to pregnancies according to truncating mutation location, in the French National BRCA1 and BRCA2 mutations carrier cohort (GENEPSO). Breast Cancer Research 2012; 14: R99.http://www.apocpcontrol.org/paper_file/issue_abs/Volume13_ No5/2179-84%204.17%20Che%20Yanhua.pdf Yanhua, C, et al. Reproductive variables and risk of breast malignant and benign tumours in Yunnan Province, China. Asian Pacific Journal of Cancer Prevention 2012; 13: 2179-84. http://www.apocpcontrol.org/paper_file/ issue_abs/Volume13_No5/2179-84%204.17%20Che%20Yanhua.pdf Yanhua et al. See n. 34.

rates also have the highest breast cancer rates.³⁸ Thus, England, with the highest abortion rates, has an incidence of breast cancer of 116 per 100,000, while Northern Ireland, where abortion is much less prevalent, has an incidence of 97 per 100,000. Scotland lies between England and Northern Ireland in both breast cancer and abortion rates. There has been a 70 per cent increase in the risk of breast cancer in the United Kingdom between 1971 and 2002.

A 1989 study of breast and cervical cancers in three republics of the USSR (Russia, Estonia and Soviet Georgia) found that their breast cancer rates increased from between 270 and 330 per cent as their abortion rates increased.³⁹ The author commented that in the USSR, the majority of women used abortion as the principal method of birth control, and that in every year since legalization in 1955, the number of abortions has exceeded the number of live births. In the discussion the author declared that, "all four abortion indicators used in the study ... appeared to be the most significant correlates of the incidence of both cancers."⁴⁰ The four independent variables ('indicators') she referred to were: frequency of induced abortion, abortions to live birth ratio, frequency of out-of-hospital abortions and frequency of the termination of the first pregnancy.

Romania had one of Europe's lowest breast cancer rates while abortion was illegal under Ceausescu. Since its legalization in 1989, breast cancer rates appear to have risen dramatically, based on an analysis of the experience in one major county. Breast cancer incidence there increased from 25 per 100,000 women in 1988 to 40 in 1996 and 51 in 2006. The total number of abortions reported in Romania jumped from 193,084 in 1989 to 992, 265 in 1990. In 1960 breast cancer comprised seven per cent of all malignant tumors; by 1996 it had risen to 23 per cent, ranking it first in cancers among women. Given the explosion in the abortion rate, we may expect the incidence of breast cancer to rise even higher in coming years.

Carroll P. The breast cancer epidemic: modeling and forecasts based on abortion and other risk factors. Journal of American Physicians and Surgeons 2007 September; 12(3): 72-8.

³⁹ Remennick LI. Reproductive patterns and cancer incidence in women: a population based correlation study in the USSR. International Journal of Epidemiology 1989 September; 18(3): 498-510.

⁴⁰ Ibid.

⁴¹ Lucaci L and Szucsik IA. Statistical study on the incidence and prevalence of breast cancer, in Arad County, between the years 1999-2009. Arad Medical Journal 2010 November; 13(4): 5-10.

⁴² Council of Europe. Romania, Table 2: births, deaths, and legal abortions. *Recent Demographic Developments in Europe: Demographic Yearbook* 2003, 2004 January. http://www.coe.int/t/e/social_cohesion/population/RTAB2.xls.

In China, the enforcement of a one-child policy, which included compulsory abortion, was followed by a substantial increase in breast cancer rates. Since 1983, there has been an increased incidence of breast cancer in China.⁴³ Over the last ten years, the incidence of breast cancer rose 31 per cent to 55 per 100,000 women per year in Shanghai, and 23 per cent to 45 per 100,000 women per year in Beijing. 44 Even more alarming, a study published in 2008 reported that China was on the "cusp of a breast cancer epidemic,"45 forecasting an incidence of 100 new cases of breast cancer per 100,000 women aged between 55 and 69 by 2021.46

In the United States, subsequent to the legalization of abortion in 1973, there has been a marked increase in the risk of both non-invasive and invasive breast cancers from 1975 to 2000.47 Invasive cancer incidence went from 105 to 136 per 100,000 women. Non-invasive cancer in pre menopausal women went from four to twelve per 100,000 women, a nearly 400 per cent increase in incidence. On a smaller scale, Washington State breast cancer rates in black women rose after abortions began being state-funded (which increased abortion availability to poor black women).48

The Bradford-Hill Criteria

Before any causal statements may be made that a risk factor is a *cause* of a disease, and not just merely associated with it, strict criteria must be met. Just because a study shows a positive association of a factor with a disease, does not necessarily mean that factor is the cause.

⁴³ Ferlay J, Héry C, Autier P and Sankaranarayanan R. "Chapter 1: global burden of breast cancer." In Breast Cancer Epidemiology. Ed. Li C. New York: Springer, 2010: pp. 13-15.

Beijing Centers for Disease Control and Prevention. China says breast cancer on rise in Beijing, Shanghai. Reuters, 30 October 2007. http://in.reuters.com/article/2007/10/30/ us-china-cancer-idINPEK20120020071030.

⁴⁵ Linos E, Spanos D, Rosner BA, et al. Effects of reproductive and demographic changes on breast cancer incidence in China: a modeling analysis. Journal of the National Cancer Institute 2008 October; 100(19): 1352-60, p. 1352.

⁴⁶ Ibid.

⁴⁷ National Cancer Institute. Cancer prevalence. SEER Cancer Statistics Review 1975-2005, 2008. http://seer.cancer.gov/csr/1975_2005/results_merged/topic_prevalence. pdf; White E, Daling JR, Norsted TL and Chu J. Rising incidence of breast cancer among young women in Washington State. Journal of the National Cancer Institute 1987 August; 79: 239-43.

Krieger N. Exposure, susceptibility, and breast cancer risk: a hypothesis regarding exogenous carcinogens, breast tissue development, and social gradients, including black/white differences, in breast cancer incidence. Breast Cancer Research and Treatment 1990 January; 13(3): 205-23.

For example, large, statistically significant and reproducible studies might show that people who carry matches in their pockets have a higher risk of lung cancer. Without the additional criteria of a plausible biological theory of how the matches cause lung cancer, these studies, no matter how many are done, show only a positive association between matches and lung cancer. Knowing that matches were associated with lung cancer might lead scientists to the discovery that the matches were mostly used to light cigarettes. This in turn would uncover the true cause of lung cancer. It was the Bradford Hill criteria, published in 1964, that brought the United States Surgeon General to place warnings on cigarette packages to the effect that they increased the risk of lung cancer.

Epidemiological studies done concerning the abortion-breast cancer link do show that they meet the criteria for classifying abortion as a causal risk for breast cancer. The following nine criteria were established by Sir Austin Bradford Hill in 1964 and were used to show the causal link between cigarettes and lung cancer.

Criterion 1: Strength of Association

Studies should show a large relative risk (RR), greater than 3.0. An RR of 3.0 means there is a 200 per cent increase in risk. (1.0 is null. 0.5 is a 50 per cent decrease in risk.)

If there is only a ten per cent increase in risk, it is difficult to say the risk is causal. There are subsets of women that show a greater than 200 per cent increased risk in breast cancer with abortion. There have been many studies such as the one by Daling (1994) that showed risks in some sub-groups of women to be much more than 200 per cent.⁴⁹ For instance, abortions done on women under the age of 18, at between nine and 24 weeks' gestation, had a relative risk of 9.0, meaning an 800 per cent increase in risk. If a woman aged 30 or older had an abortion at between nine and 24 weeks' gestation, her risk of breast cancer increased by 230 per cent.

Studies must be statistically significant.

Scientists require 95 per cent certainty that the study results were not obtained by chance alone. There are now 35 statistically significant studies that show the abortion-breast cancer link.

Criterion 2: Temporality

The exposure to the risk must occur before the disease is detected, meaning that the abortion must occur before the breast cancers form.

This may seem so obvious that it need not be mentioned. However, a well-known study, that by Melbye and colleagues (1997) violated this rule when it collected breast cancer cases from a registry starting in 1968, but abortions only from 1973.50 The cancer cases between 1968 and 1972 had no place in the study. By including them the authors minimized the link between breast cancer and abortion.

Criterion 3: Consistency

The preponderance of studies must show a positive association.

One or two studies by themselves can never be thought to support a causal link. Out of 73 published world wide studies done to date, 56 show a positive association, 51 of which 35 are statistically significant, while a total of seventeen studies show no link.

There are many reasons why studies might not show a link. If the population studied had abortions followed shortly by a full-term pregnancy in many instances, a very small effect would be expected and might not be observed. If the abortions were used to limit enlarging a family with many children, a small effect would be expected as the mother's breast tissue would have largely matured in previous pregnancies. This circumstance occurs in countries such as Italy, where the effect of induced abortion on breast cancer risk has been found to be less than other European countries. There may be bias in selection of patients so that very few young women, who have the highest risk with abortion, are included. The study may not follow most of the patients long enough for cancers to form; breast cancers take at least eight or more years to be detected, based upon the average doubling time for breast cancer cells. The study may choose not to look at all breast cancers and consider only some, such as invasive breast cancers, thereby eliminating approximately 25 per cent of cancers. For instance, in the US there were 230,480 cases of invasive breast cancers and 57,650 non-

⁵⁰ Melbye et al. 1997. See n. 29.

⁵¹ Breast Cancer Prevention Institute. Epidemiologic studies: induced abortion and breast cancer risk. Fact Sheet. 2012 September. .www.bcpinstitute.org.

invasive breast cancers in 2011.52

Criterion 4: Theoretical Plausibility

The biological mechanism that explains the reason for the risk association must be biologically plausible.

The breast physiology, which explains the risk of breast cancer with induced abortion, has been thoroughly explained in a previous section. Elevated levels of estrogen during pregnancy leave the breast with increased numbers of Type 1 and 2 lobules where breast cancers arise, and without the benefit of a full-term pregnancy maturing the breast into predominantly Type 3 lobules, which are cancer-resistant. This same physiology can account for other well-accepted reproductive risks such as nulliparity (no births) and premature delivery. It has been shown that the longer a woman is pregnant before an abortion (up to 32 weeks), the higher her risk of breast cancer.⁵³

Criterion 5: Coherence

The hypothesis, when proven, does not do violence to related sets of scientific findings but, instead, fits in with them.

The biological hypothesis of the abortion-breast cancer link (abortion causes an increase in the number of cancer-susceptible lobules and cells and, therefore, risk) is consistent with all other reproductive risk factors concerning pregnancy, such as: the protective effect of a full-term pregnancy while nulliparity (no births) increases risk; early age at full-term pregnancy decreasing risk; late age at full-term pregnancy increasing risk; the transient increase in breast cancer risk in older women who have their first pregnancy late in their reproductive life; lower exposure to estrogen and therefore risk after a full-term pregnancy because of an increase in sHBG (serum hormone binding globulin); lower prolactin levels in parous women (who have given birth) decreases risk; lower risk with each full-term pregnancy, and higher risk with premature delivery before 32 weeks.

⁵² American Cancer Society (ACS). *Breast Cancer Facts and Figures* 2011-2012. Atlanta, GA: ACS, Table 1, p. 2. http://www.cancer.org/acs/groups/content/@epidemiologysurveilance/documents/document/acspc-030975.pdf.

⁵³ Melbye et al. 1997. See n. 29. After 32 weeks of course, the risk of breast cancer declines sharply.

Lung cancer does not form the day after you smoke a pack of cigarettes; it takes many packs over many years. Similarly, breast cancer does not develop immediately after an abortion. Yet, the association of breast cancer and abortion is in accord with the known natural history and biology of breast cancer. It takes an average of eight to ten years for one breast cancer cell to keep doubling so that it forms a tumor of clinically detectable size about one centimetre. Most studies show the increase in breast cancers occurring in the time frame appropriate for the development of breast cancer, namely, at least eight to ten years after exposure.

Criterion 6: Specificity of Cause

Factor X leads to outcome Y.

There are ecological epidemiological studies that show induced abortion is the best predictor of breast cancer rates in a country. Induced abortion was found to be the greatest predictor of breast cancer rates in nine European countries.54 Another study done in the USSR also showed such a link.55

Criterion 7: A Dose Effect is Observed

Based on biological mechanisms, the more one is exposed to the risk, the higher the risk of the disease if a factor is causal.

For example, the more cigarettes one smokes, the higher the risk of lung cancer. The longer one is pregnant before an abortion, the more immature breast tissue would be formed up to 32 weeks, and the higher the risk of breast cancer. The Melbye study showed a three per cent increase in the risk of breast cancer with each week of gestation.⁵⁶ However, it lumped together all late-term abortions after eighteen weeks' gestation. There is also evidence that the more abortions a woman has, the greater will be her risk of breast cancer.⁵⁷ Yet it is also true that, just as one exposure to

Carroll. See n. 38. 54

⁵⁵ Remennick. See n. 39.

Melbye et al. 1997. See n. 29; Goldacre MJ, Kurina LM, Seagroatt V and Yeates D. Abortion and breast cancer: a case control record linkage study. Journal of Epidemiology and Community Health 2001 May; 55(5): 336-7; Erlandsson G, Montgomery SM, Cnattingius S and Ekbom A. Abortions and breast cancer: record based case-control study. International Journal of Cancer 2003 February; 103(5): 676-9.

Jiang et al. See n. 21; Yanhua et al. See n. 34; Remennick. See n. 39. 57

asbestos can cause a mesothelioma to form, so only one abortion may be enough to induce breast cancer.

Criterion 8: Experimental Studies

Variables are experimentally controlled for and yield predicted results consistently, first in animal studies, later in human (if ethically permissible, which, of course, is not the case with abortion).

Two pathologists studied the effect of a breast carcinogen (DMBA) given to groups of rats. The rats were either virgin, had a litter of pups, or had been aborted. The aborting rats developed breast cancers at a much higher rate when given DMBA than the virgins. No cancers occurred in one group of rats that had had a full-term pregnancy.⁵⁸

Criterion 9: Analogy

Similar exposures should result in similar effects; for example, cigarette smoking causes bladder, as well as lung, cancer.

By the same token, premature deliveries before 32 weeks also double breast cancer risk because the breasts are left with more lobules where breast cancers can start. An abortion can be thought of as premature delivery by an abortionist.⁵⁹

US racial trends of abortions and breast cancer incidence

In the US, although the incidence of breast cancer is highest among the Caucasian population, African-Americans under the age of 35 have an even higher incidence. The phenomenon has been labeled "inexplicable" by a prominent breast surgeon. However, it becomes both explicable and predictable when abortions and premature births are compared between these racial groups. Although African-Americans make up twelve per cent of the population, they account for 35 per cent of total abortions, even though they constitute barely 12 per cent of the population. Caucasians

Russo J and Russo IH. Susceptibility of the mammary gland to carcinogenesis. American Journal of Pathology 1980 August; 100(2): 497-512.

Melbye et al. 1999. See n. 15.

⁶⁰ Bland K. William Hunter Herridge Lecture: Contemporary management of pre-invasive and early breast cancer. American Journal of Surgery 2011 March; 201(3): 278-89, p. 279.

account for 55 per cent of all abortions. African Americans are thus three times more likely to have an abortion than Caucasians.⁶¹ According to the Institutes of Medicine, in the US the overall premature birth rate before 37 weeks is 12.5 per cent, but 17.8 per cent in African Americans. As we have seen, both abortion and preterm birth are risk factors for breast cancer.⁶²

QUESTIONING THE ABORTION-BREAST CANCER LINK

Recall Bias

"Recall bias" is the most widely and oft-reported argument used against the ABC link. This is the hypothesis that women who have developed breast cancer will be more likely to admit that they have had abortions than women who are well. The theory is based on the assumption that healthy women are more likely to conceal what could be embarrassing behavior but are more likely to tell the truth should they become ill, seeking a reason for their illness. Recall bias thus supposes that many women who do not have cancer will not report their abortions while those who do have cancer will report them. Case control studies in which researchers rely on interviews for their data are thought to be those potentially susceptible to recall bias. This is because researchers assume interviewees will not admit in an interview to "socially unacceptable behavior," such as abortion-unless they are sick. However, recall bias has not posed any such problem in other areas of medical research where case control studies have been used to gather data of other socially unacceptable behavior. For instance, in case control studies testing for a link between alcohol consumption and liver damage, interviewees were assumed to report their alcohol consumption accurately. The same is true for interviews in which interviewees were asked how many sexual partners they had, when inquiring into connections with cervical cancer, and whether they were involved in anal intercourse, when inquiring into HIV. Abortion would not seem to be a more socially unacceptable act than any of these. Why then should recall bias be thought to taint research about abortion but not the others?

Often the "Swedish" study is cited when using the argument of recall

Elam-Evans LD, Strauss LT, Herndon J, Parker WY, Bowens SV, Zane S and Berg CJ. Abortion surveillance—United States, 2000. Centers for Disease Control and Prevention. 28 November 2003. www.cdc.gov/mmwr/preview/mmwrhtml/ss5212al.htm.

Institute of Medicine. Preterm birth: causes, consequences and prevention. 13 July 2006. www.iom.edu/Reports/2006/Preterm-Birth-Causes-Consequences-and-Prevention.aspx.

bias⁶³; yet it was convincingly refuted in a subsequent letter to the editor.

Recall bias is a hypothesis worth testing. Yet, studies that have confirmed the ABC link internally controlled for recall bias in their study populations.⁶⁴ Moreover, a study conducted specifically to test for recall bias in abortion-breast-cancer research reported having found evidence of it; however, methodological problems with the study acknowledged after publication revealed that it actually failed to show that recall bias taints such research. Instead, the study's results supported what is true to clinical experience: almost equal numbers of women with cancer and without cancer concealed their abortions. 65 Researchers in the Lindefors-Harris study had before them both cancer and abortion computer registries in order to verify the responses of the women who were interviewed. Two groups of women were interviewed: those with cancer and those without. The researchers hypothesized that more of those without cancer would deny their abortions while more of those with cancer would admit to them. Such a result would be evidence of recall bias. Instead, they found no statistically significant difference between the responses of the two groups of women. 66 In short, most healthy women and sick women admitted to the abortions officially documented in the abortion registry while some healthy women and some sick women lied. There was a statistically insignificant difference of barely six per cent between the two groups, which even in large studies would not greatly affect the results. On the other hand, researchers did find women—both healthy and sick—who admitted to abortions that were not documented in the abortion computer registry. The researchers labeled this phenomenon "over reporting," claiming that women who told the researchers that they had had abortions that had not been reported in the computer registry were mistaken or lying. The researchers would have been better advised to assume a mistake in the registry or that the women had their abortions in another country. Only with this wrongheaded assumption of over reporting did the authors then conclude that they had significant evidence of recall bias. Over reporting, of course, does not exist. The researchers were forced to acknowledge their error in a subsequent

Lindefors-Harris BM, Eklund G, Adami HO and Meirik O. Response bias in a case-control study: analysis utilizing comparative data concerning legal abortions and two independent Swedish studies. American Journal of Epidemiology 1991 November; 134(9): 1003-8.

⁶⁴ Daling et al. 1994. See n. 4; Lipworth, Katsouyanni, Ekbom, et al. 1995. See n. 34.

⁶⁵ Lindefors-Harris, Eklund, Adami and Meirik. See n. 63.

Women with cancer and women without cancer both underreported their abortion in similar percentages (5 out of 24 women or 21 per cent, and 16 out of 59 or 27 per cent, respectively).

exchange of letters to the editor. 67 Unfortunately, since most doctors read only the abstract of a paper and do not follow letters to the editor, a false impression of the study's results remains.

Studies with Contrary Findings

It is widely reported that the reason that "early" studies showed an association between abortion and breast cancer was that they were small, "case controlled" studies, subject to recall bias.

There is a belief that record linkage studies, in which patients are not interviewed, are more reliable. One such study showed a 90 per cent increased risk of breast cancer in women under 40 who had undergone one or more abortions.⁶⁸ New York State fetal death certificates and breast cancer registries were used, ensuring that there could be no "recall bias", as patients were not interviewed. That study is ignored when web sites of cancer organizations state that there are no record linkage studies demonstrating an abortion-breast cancer link. Rather there are very well-publicized studies, often cited, that purport to show no link. When scrutinized, these studies are found to have major flaws.

Between 1996 and August 2005, ten epidemiological studies were published based on prospective data regarding induced abortion and breast cancer. Brind soon published an analysis of these studies.⁶⁹ In great detail, he uncovered egregious errors, too many to list here. However, we will now analyze five studies often cited to show no link between abortion and breast cancer. In the first, the Scottish Brewster study, we find the transparent use of selection bias. 70 That study selectively used a data base that was not representative of the Scottish population. Although 58 per cent of abortions in Scotland are done on young nulliparous (no

⁶⁷ Meirik O, Adami HO and Eklund G. Letter re: relation between induced abortion and breast cancer. Journal of Epidemiology and Community Health 1998 March; 52(3): p. 209; Brind J, Chinchilli VM, Severs WB and Summy-Long J. Reply to letter re: relation between induced abortion and breast cancer. Journal of Epidemiology and Community Health 1998 March; 52(3): 209-11.

⁶⁸ Howe, Senie, Bzduch and Herzfeld. See n. 34.

Brind J. Induced abortion as an independent risk factor for breast cancer: a critical review of recent studies based on prospective data. Journal of American Physicians and Surgeons; 2005 Winter; 10(4): 105-10.

Brewster DH, Stockton DL, Dobbie R, Bull D and Beral V. Risk of breast cancer after miscarriage or induced abortion: a Scottish record linkage case-control study. Journal of Epidemiology and Community Health 2005 April; 59(4): 283-7.

births) women who would be most affected by abortion, the group that was studied had only 5.6 per cent nulliparous women. Another often cited study, known as the California Teachers Study and which attempts to deny the abortion-breast cancer link, is an extreme example of selection bias.⁷¹ Women were eliminated from this study if they were diagnosed with non-invasive breast cancer (ductal carcinoma in-situ or DCIS) while being followed. DCIS is a precursor of invasive ductal cancer, which may take years to form. So the women most *likely* to develop invasive breast cancer were eliminated from the study before that cancer could develop! Another large British study reportedly showed no association of breast cancer and abortion. 72 However, for the more than 28,000 patients, only 300 abortions were listed over a 30-year period, despite the fact that in the UK the recorded abortion rate was one per cent per year for the study period. This means that 90 per cent of the women listed as having no abortion, almost certainly did have one. In fact, the authors admitted that their "data on abortion are substantially incomplete."73 Their data were incomplete because the authors only considered abortions performed in inpatient hospital facilities, while most abortions are done in outpatient facilities, not in hospitals. Yet this publication is commonly reported as a large study showing no abortion-breast cancer link.

Melbye (1997): 1.5 Million Danish women

When it was published, the Melbye study was hailed as the definitive answer to the question "Does abortion increase breast cancer risk"?⁷⁴ It was the first large study to be published after Dr. Brind's 1996 meta-analysis. In an editorial accompanying the publication, NCI epidemiologist Patricia Hartge proclaimed the 1997 Melbye to be a definitive study so that "In short, a woman need not worry about the risk of breast cancer when facing the difficult decision of whether to terminate a pregnancy."⁷⁵ However, it misclassified 60,000 women who had legal abortions as not having had abortions because the authors used abortion registries starting in 1973 instead of 1940. It also violated the Bradford Hill criterion of temporality

Henderson KD, Sullivan-Halley J, Reynolds P, et al. Incomplete pregnancy is not associated with breast cancer risk: the California Teachers Study. Contraception 2008 June; 77(6): 391-6.

⁷² Goldacre 2001. See n. 56.

⁷³ Ibid., p. 337.

⁷⁴ Melbye et al. 1997. See n. 29.

⁷⁵ Hartge P. Editorial: abortion, breast cancer, and epidemiology. NEJM 1997; 336(2): 127-8.

by collecting breast cancer cases starting in 1968 while collecting data on abortions using records that started in 1973. Another factor that contributed to the study's methodological flaws relates to the biology of breast cancer. It takes an average of eight to ten years for a cancer cell to grow into a clinically detectable cancer of one cm. diameter, based on average doubling times for cancer cells (the time needed to undergo one mitosis or cell replication). If an abortion in an eighteen-year-old causes a breast cancer cell to form, it is not likely to be detectable until she is at least 26. Fully one-quarter of the patients in the Melbye study were under 25 when the study ended, accounting for only eight cases of breast cancer. Because of what is known about the time needed for the development of breast cancer, none of these young women should have been included in the study.⁷⁶

Yet even with this and other major flaws, the study showed a statistically significant risk in women who have had abortions performed over eighteen weeks' gestation. This fact was not mentioned in the conclusion of the paper, which merely stated that there was no link at all between abortion and breast cancer. This paper is still cited as large and conclusive, as if a single study could be conclusive. It is often used in major textbooks to show there is no link between abortion and breast cancer (see below, pp. 136-7).

Beral Re-Analysis

In 2004, The Lancet published a study⁷⁷ hailed by its authors, Valerie Beral and colleagues, as the definitive analysis that puts to rest the claim that abortion increases breast cancer risk. Beral was quoted as saying, "Scientifically, this is really a full analysis of the current data."78 However, a review of the study reveals that it is not a "full analysis". Indeed, serious methodological flaws—especially in the selection of studies to be included—render the Beral study unreliable. The authors were guilty of several errors in selecting the studies to include in their analysis. First, they eliminated eleven studies for unscientific reasons (e.g., "principal investigators could not be found," or "researchers declined to take part in collaboration"), and four other studies'

⁷⁶ Gersho-Cohen J, et al. Roentgenography of Breast Cancer Moderating Concept of "Biologic Predeterminism". Cancer 1963 August; 16: 961-4.

Collaborative Group on Hormonal Factors in Breast Cancer. Breast cancer and abortion: collaborative reanalysis of data from 53 epidemiological studies, including 83,000 women with breast cancer from 16 countries. The Lancet 2004 March; 363(9414):

Wahlberg D. Study: breast cancer not tied to abortion. Atlanta Journal Constitution, 26 March 2004.

worth of data were simply not mentioned at all.⁷⁹ Thus, in the end they included only 24 of the 41 studies in existence at the time of the re-analysis that contained data on induced abortion and breast cancer. To supplement these 24 studies, the researchers added a further 28 unpublished studies. This means that the majority of the studies included in their analysis had not themselves stood the test of peer review, nor could they be consulted by other researchers.

A closer look reveals that many of the statistically significant studies that demonstrate a link between abortion and breast cancer have been excluded. To be precise, of the 41 studies published up to 1994, 29 actually showed increased risk of breast cancer among women who chose abortion (epidemiologists call this a "positive association"). Seventeen of these 29 studies were statistically significant. Yet ten of the seventeen significantly positive studies in the literature were excluded by Beral and her colleagues. If the results of the fifteen studies supposedly excluded for being unscientific are averaged, they show an increase in breast cancer risk of 80 per cent among women who had abortions.

Beral and colleagues also divided the studies into two separate categories: those that used retrospective methods of data collection (i.e., interviews of breast cancer patients versus control subjects), and those that used prospective methods (i.e., medical records taken long before the breast cancer diagnosis). The 39 studies that used retrospective methods showed a significant overall eleven per cent increase in risk with abortion. The thirteen studies that used prospective methodology showed a significant seven per cent decrease in risk with abortion. Instead of reporting the results of her findings accurately (that is, that the retrospective studies showed that abortion increases the risk of breast cancer), they simply declared that these studies were unreliable—because of "recall bias". Despite the theoretical possibility that recall bias exists, we have seen that tests for such bias have proven negative.

Finally, at least three of the prospective data-based studies merit

The four studies not mentioned in the Beral et al. analysis were: Laing, Demenais, Williams, Kissling, Chen and Bonney. See n. 34: A300; Bu, Voigt, Yu, Malone and Daling. See n. 34; Bu, et al. Risk of breast cancer associated with induced abortion in SER Abstracts S85 (abstract 337); Luporsi. See n. 34; Zaridze DG 1988, in Andrieu, Duffy, Rohan, et al. Familial risk, abortion and their interactive effect on the risk of breast cancer—a combined analysis of six case-control studies. British Journal of Cancer 1995; 72(3): 744-751.

exclusion from the Beral study because of vast gaps in their databases, and consequent misclassification of subjects.80 The Melbye study, discussed above, is one such study that should have been excluded. Another major flaw was that an inappropriate comparison group was chosen. Beral compared apples and oranges when the effects of having had a pregnancy that ended in abortion were compared with the effect of "not having had that pregnancy." Once a woman has had a healthy pregnancy, however long, her breasts are different than before that pregnancy started. Pregnancy forever alters the breast and physiologically these women are as different as pre- and post-menopausal women. Just as the effect of hormone replacement for post-menopausal women is studied in relation to other post-menopausal women who have no exposure to hormones, pregnant women who undergo abortion need to be compared to pregnant women who do not undergo induced abortion.

The National Cancer Institute

In addition to denying the abortion-breast cancer link, flatly incorrect information regarding breast cancer risk is given to patients on the website of the National Cancer Institute (NCI) in Washington D.C. For example, under the section on protective factors and decreased exposure to estrogen, it is stated that the exposure to estrogen "is reduced in the following ways: Pregnancy: estrogen levels are lower during pregnancy."81 In fact, estrogen levels rise 2000 per cent by the end of the first trimester. Either the scientists at the NCI are unaware of this, or they are avoiding the biological explanation of why an early first full-term pregnancy reduces breast cancer risk.

In 2003, the NCI conducted a "Workshop on Early Reproductive Events and Breast Cancer Risks". The opinion and conclusion arrived at by the 100 scientists who attended the workshop are often cited to justify disregarding the evidence for the abortion-breast cancer link. They asserted that induced abortion was not a risk factor for breast cancer and need not be studied any further. However, they did note that premature birth was a risk for breast cancer. Interestingly, they also declared premature delivery to be an "epidemiological gap" that should be studied further. The obvious analogy between induced abortion and premature delivery causing the same physiological changes in a woman's breasts, and thereby similar

⁸⁰ Melbye et al. 1997. See n. 29; Goldacre 2001. See n. 56; Erlandsson, Montgomery, Cnattingius and Ekbom. See n. 56.

National Cancer Institute. Breast cancer prevention. 20 September 2011. http:// www.cancer.gov/cancertopics/pdq/prevention/breast/Patient/page3.

breast cancer risks, was ignored. Researchers who had conducted studies that supported the abortion-breast cancer link were not asked to present on that topic. Scientists present were not permitted to see the data presented before they were published. More telling was an interview given the day the workshop ended by an epidemiologist and workshop leader, Leslie Bernstein. She stated that having a child was the surest way to reduce breast cancer risk, but added "I would never be a proponent of going around and telling them that having babies is the way to reduce your risk." More tellingly, she went on, "I don't want the issue relating induced abortion to breast cancer risk to be a part of the mix of the discussion of induced abortion, its legality, its continued availability."82 A detailed report by a workshop participant who disagreed with the official conclusion and noted the irregularities in the conduct of the meeting was submitted as a "Minority Report."83 In 2009, a more damning situation arose with the publication of a paper regarding oral contraceptives and breast cancer risk, which put the veracity of the "consensus" in doubt.84 The fourth author, affiliated with the NCI, was Louise Brinton, who had also chaired their February 2003 Workshop. This was significant because the paper concluded that induced abortion was a 40 per cent statistically significant risk factor for breast cancer. When a journalist for the Toronto Globe and Mail, Gloria Galloway, tried to question Louise Brinton in January 2010 about her apparent change of view on induced abortion and breast cancer risk, Brinton refused to be interviewed.85 It should be borne in mind that scientific truth is determined by the examination of evidence derived through study and experiment, not by voting or consensus, which are subject to political and personal pressures. After all, it was once the scientific consensus that the universe was composed of only four elements, and that the sun revolved around the earth. The NCI's directors are political appointees of the US President. As appointees, they are influenced by political forces and agendas.

⁸² Lanfranchi A. The federal government and academic texts as barriers to informed consent. Journal of American Physicians and Surgeons 2008 Spring; 13(1): 12-15.

⁸³ Brind J. Early reproductive events and breast cancer: a minority report. 2003 March. Paper given at the NCI workshop "Early Reproductive Events and Breast Cancer," February 24—26, 2003, Bethesda, MD.

⁸⁴ Dolle et al. See n. 16.

Galloway G. Was Maurice Vellacott right about abortion? The Globe and Mail, 8 January 2010. http://www.theglobeandmail.com/news/politics/ottawa-notebook/was-maurice-vellacott-right-about-abortion/article4351341/.

Political and Social Pressures

In 1860, Dr. Oliver Wendell Holmes, a physician, essayist, and father of the celebrated US jurist, in an address to the Massachusetts Medical Society, stated, "Theoretically, [medicine] ought to go on its own straightforward inductive path without regard to changes of government or to fluctuations of public opinion [...] The truth is that medicine, professionally founded on observation, is as sensitive to outside influences, societal, religious, philosophical, imaginative, as the barometer is to the changes of atmospheric pressure."86 That powerful statement also reflects what has continued to be a part of the fabric of medicine today. Physicians are human and susceptible to the same pressures as other people. Although ideally physicians are trained to be inured to those pressures, sadly we are not. There is documented evidence of widespread fraud in connection with National Institute of Health (NIH) funded research. (The NCI is a part of the NIH.) A paper in the British journal, *Nature*, using anonymous questionnaires, revealed that a statistically significant 15.5 per cent of scientists admitted to "changing the design, methodology or results of a study in response to pressure from a funding source." That funding source was the NIH.87

There is extensive evidence for the existence of the bias, which continues to make the independent abortion-breast cancer link ignored or unknown.

Pressure to include public advocacy in epidemiology

It is recognized that there are two competing schools of thought regarding the field of epidemiology. Epidemiology is viewed as an objective science in one school and as a science that must include public advocacy in the other. Raj Bhopal, an eminent epidemiologist, has stated that the fundamental question is "...whether epidemiology is primarily an applied public health discipline...or primarily science in which methods and theory dominate over practice and application." In 1999, a commentary in the American Journal of Public Health succinctly put the question as whether epidemiology was a science or mission. Clearly a strong advocacy position can lead to bias in reporting the data that science has collected. Recent revelations in

Holmes OW. Address to annual meeting, Massachusetts Medical Society, 30 May 86 1860. Para. 7. In Currents and Counter-currents in Medical with Other Addresses and Essays. Boston: Ticknor and Fields, 1861.

Martinson BC, Anderson MS and deVries R. Scientists behaving badly. Nature 2005 June; 435: 737-8.

the media concerning the global warming controversy have shown that eminent scientists on either side are capable of suppressing or ignoring data that do not support their position. Imagine if an epidemiologist wants to advocate for zero population growth as a method to help reduce pollution and disease. Is it not doubtful that he or she would also favour widespread dissemination of data that support the abortion-breast cancer link when abortion is still used as the primary method of birth control in China and the countries that once constituted the USSR? For example, an epidemiologist financially supported by the NCI, Lynn Rosenberg, is on the record as a staunch abortion advocate who has also testified as an expert before governmental bodies making laws on abortion regulation.⁸⁸ Rosenberg also wrote an editorial in the Journal of the NCI dismissive of Janet Daling's 1994 landmark study, which appeared in the same issue and showed an overall 50 per cent increase in breast cancer risk with abortion.⁸⁹

ii. Misleading academic texts

Bias is seen in academic breast cancer texts concerning prevention and risks. The preventive effects of child-bearing and the risk-increasing effects of induced abortion are misstated in major textbooks. In the 2000 edition of *Diseases of the Breast* by Jay Harris and colleagues, early full-term pregnancy is not listed in its table of methods of prevention because, according to the accompanying text, "unplanned early pregnancy and an average of more than two completed pregnancies per woman have undesirable social and ecologic consequences."⁹⁰ The fact that it takes a fertility of at least 2.1 children per woman just to maintain a given population is disregarded. The book's recommendations appear to be influenced by the notion that human beings are bad for the "ecology." Busy practising clinicians may rely on tables for a quick answer, rather than reading the whole text.

Although the 1991 and 1998 editions of *The Breast: Comprehensive Management of Benign and Malignant Disease* clearly stated that induced abortion was a risk factor for breast cancer in the chapter concerning

North Florida Women's Health and Counselling Services, Inc., et al., v. State of Florida, et al. Circuit Court, Second Judicial District, Leon County, FL, No. 99-3203, 1999.

⁸⁹ Rosenburg L. Induced abortion and breast cancer: more scientific data are needed. Journal of the National Cancer Institute 1994 November 2; 86(21): 1569-70.

⁹⁰ Harris J, Lippman ME, Morrow M and Osborne CK. *Diseases of the breast*, 2nd ed. Baltimore, MD.: Lippincott, Williams & Wilkins, 2000: 211-2. Editions are as follows: 1st Ed. (1996 Lippincott Raven); 2nd Ed. (2000 Lippincott Williams & Wilkins); 3nd Ed. (2004 Lippincott Williams & Wilkins); 4th Ed. (2010 Lippincott Williams & Wilkins).

molecular biology, the 2004 edition removed that information.⁹¹ In its place was a misleading table of breast cancer risks. Induced abortion is listed in the table as having "no effect" on breast cancer risk. This statement is contradicted by the accompanying text, which states that abortion after 12 weeks carries a relative risk of 1.38, a 38 per cent increase. 92 By the 2009 edition, induced abortion was not listed in any of the tables that tabulated risk factors; yet, it had a similar paragraph of text as in the 1998 edition.

iii. Sociological pressures

It is very difficult for the public to believe that physicians who are thought to put their patients' health first, or scientists looking for scientific truth, could be involved in misinformation. It is difficult for breast surgeons to tell their patients that abortion increases breast cancer risk when the referring physician performs abortions. It may also be seen by their patient that they are being told that their own behavior has caused their cancer. Even patients who have had no abortions yet developed breast cancer may feel tainted by being perceived to have abortion as part of their medical history.

Knowing that approximately 40 per cent of women in the US will have had an abortion by the age of 40, there is the risk of telling a cancer patient that she may have contributed to the development of her own disease. It is much more comfortable not to risk offending professional colleagues who perform abortions, refer for abortions, or have had abortions as part of their personal history. Maintaining a cordial professional relationship ensures continued referrals and a pleasant practice environment.

iv. Political pressures

There are many historical examples of political pressures on governmental

Dickson RB and Lippman ME. Chapter 27: growth and regulation of normal and malignant breast tissue. In The Breast: Comprehensive Management of Benign and Malignant Diseases. Eds. Bland KI and Copeland EM. 2nd ed. Philadelphia: Saunders, 1998, p. 523; Editions of this book are as follows: Bland KI and Copeland EM. The Breast: Comprehensive Management of Benign and Malignant Disorders. Philadelphia: Saunders, 1991; Bland and Copeland. The Breast: Comprehensive Management of Benign and Malignant Disorders. 2nd ed. 2 vols. Philadelphia: W.B. Saunders, 1998; Bland and Copeland. The Breast: Comprehensive Management of Benign and Malignant Disorders. 3rd ed. 2 vols. St. Louis, MO: Saunders, 2004; Bland and Copeland. The Breast: Comprehensive Management of Benign and Malignant Disorders. 4th ed. 2 vols. Philadelphia: Saunders Elsevier, 2009.

Vogel V. "Chapter 16: epidemiology of breast cancer." In The Breast: Comprehensive Management of Benign and Malignant Diseases. Eds. Bland KI and Copeland EM. 2004. See n. 90, Table 16-1, pp. 343-4.

institutions that led to public health care information and policies that were damaging to public health. One especially egregious and welldocumented case was the political pressure upon the NCI, which allowed a lung cancer epidemic to evolve into a major health care liability. For decades the epidemiological evidence of the cigarette-lung cancer link was suppressed. 93 The first study linking cigarettes to lung cancer was published in 1928. Even though thoracic surgeons were reporting huge increases in lung cancers after World War II, the NCI was largely silent and minimized smoking's risk in the development of lung cancer. This was shown to be due to pressure from southern senators. They complained that if it became widely known that cigarettes caused lung cancer, their states' economies, which were based on the production of tobacco and tobacco products, would collapse, causing financial ruin. In fact it was not the NCI that brought the cigarette-lung cancer link to public attention. Rather, it was the US Surgeon General, when in 1964 he made his first report to protect the public health, and required warnings on all cigarette packs.

Political pressure can also be brought to bear upon professional groups by governments to advance their political agendas. For example, during the presidency of Bill Clinton, a case was argued before the Supreme Court over whether the state of Nebraska was constitutionally entitled to ban partial birth abortion. The American College of Obstetrics and Gynecology (ACOG) had prepared a statement concerning partial-birth abortion, which was thought by legal counsel at the White House to be too problematic and a "disaster" for them, the term used by the future Supreme Court Justice, Elena Kagan, while working for Clinton. According to the initial ACOG statement, experts "could identify no circumstances under which the [partial-birth] procedure...would be the only option to save the life or preserve the health of the woman." This made it impossible for the White House to claim that there were medical reasons to support partial-birth abortions. ACOG's final statement added the phrase that Kagan wanted for purely political reasons, namely that the procedure may be "the best or most appropriate procedure in a particular circumstance to save the life or preserve the health of a woman." This information came to light several years later, when Kagan was interrogated in the confirmation hearings before being ratified as a Supreme Court Justice. She explained to the Senate judiciary committee that her meetings with ACOG were for the purpose of ensuring that ACOG had the opportunity to paint the whole picture.

⁹³ Kessler DA. *A Question of Intent: A Great American Battle with a Deadly Industry.* New York, NY: Public Affairs, 2001.

This answer was characterized by Senator Hatch as a "real politicization of science."94 Former Surgeon General C. Everett Koop wrote an open letter to all the senators urging that Kagan should be rejected for her disgraceful and unethical action in seeking the replacement of a medical statement with a political one.95

Political pressures are brought to bear by breast cancer advocacy groups with links to the abortion industry to deny the abortion breast cancer link. The Susan G. Komen Foundation is a breast cancer advocacy group in the United States, which has raised millions of dollars since its inception over twenty years ago. Its founder, Nancy Brinker, was also a board member of Planned Parenthood, a leading abortion provider. Planned Parenthood was also the recipient of grants from Komen. Komen denies the abortionbreast cancer link, as does the National Breast Cancer Coalition, an advocacy group that heavily lobbies the US Congress, influencing research funded by the Department of Defense. 6 The coalition maintains that there is no way to prevent breast cancer, despite the clear evidence that when fifteen million women stopped their hormone replacement therapy, breast cancer incidence began decreasing. Abortion is a large industry with trade organizations that lobby politicians to maintain a favorable environment in which to function.

Conclusion

There can be no doubt that a woman who is pregnant will increase her risk of breast cancer if she aborts that pregnancy. She will either remain childless, which in itself increases breast cancer risk, or she will delay her first full-term pregnancy, another known risk for breast cancer. She is also

⁹⁴ AUL Action: The Legislative Action Arm of Americans United for Life. Investigating the Confirmation Testimony of Elena Kagan Before the US Senate Judiciary Committee and the Negative Impact of Her Amendment of the 1997 Policy Statement of the American College of Obstetricians and Gynecologists (ACOG) on the Federal Administration of Justice and the US Supreme Court. Washington, DC: AUL Action, July 15, 2010. http://www.aul.org/featuredimages/Kagan-Ethics-Report.pdf.

Kiely KC. Everett Koop urges senators to block Kagan. USA Today, 19 July 2010. http://content.usatoday.com/communities/onpolitics/post/2010/07/everett-koop-urgessenators-to-block-kagan/1; Bravin J. Dr. Koop: keep Kagan off high court. Wall Street Journal, 19 July 2010. http://blogs.wsj.com/washwire/2010/07/19/dr-koop-keep-kagan-offthe-supreme-court/.

National Breast Cancer Coalition. Truth #30: I can influence what happens in Washington D.C. about breast cancer. 2011. http://www.breastcancerdeadline2020.org/ know/31-myths-and-truths/truth-30-i-can-influence-capitol-hill.html.

deprived of breastfeeding her baby, which would further reduce her breast cancer risk.

By the end of a full-term pregnancy, a woman will cause 85 per cent of the Type 1 and 2 breast lobules she developed at puberty (where ductal and lobular cancers start respectively) to mature to Type 4 lobules, which are cancer-resistant. There are documented changes in the breast cells' genomes, which have been studied and provide the known molecular basis for the protective effect of a full-term pregnancy. In addition to the loss of the benefit of a full-term pregnancy, abortion increases her risk for breast cancer by increasing the number of breast cells where cancers can start. This fact is not only supported by the known biology of breast maturation, but through the world-wide epidemiological studies that have been done since 1957, which show that induced abortion increases breast cancer risk. After an abortion, a woman is more likely to have a premature delivery, which again increases her breast cancer risk. Scientific honesty makes it impossible to disregard over half a century of world medical literature confirming the abortion-breast cancer link. To dismiss those studies as flawed is scientifically untenable. On the other hand, the few studies recently acclaimed as disproving the abortion-breast cancer link have been guilty of major methodological flaws. They also fly in the face of the most recent studies from around the world, which continue to confirm the link between abortion and breast cancer. The fact that induced abortion significantly increases the risk of breast cancer deserves to become widely known to the public and to the medical profession. Women must be told of these risks so they can be fully informed before consenting to abortion.