Surgical Associates of Central New Jersey

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Specializing in Breast Surgery

Feb. 8, 2012 **Proponent, HB 2598**

Report in support of informed consent provisions of Kansas HB 2598 "Pro-Life Protections Act"
Submitted by Angela Lanfranchi, MD FACS
to the House Federal - State Affairs Committee; Rep. Steve Brunk, chair

Medical authorities and textbooks now accept:

1) that a full-term pregnancy lowers a woman's risk of breast cancer;
2) that each additional pregnancy further lowers her risk by 10%; and
3) that for each year a woman delays a full-term pregnancy her risk of premenopausal breast cancer increases by 5% and postmenopausal breast cancer by 3%.[i]

These three facts alone necessarily cause a pregnant woman who chooses to end her pregnancy by abortion to increase her risk of breast cancer. This is because:

1) abortion causes her to lose the benefit of a full-term pregnancy;
2) she will have fewer or no full-term pregnancies; and
3) she necessarily delays a full-term pregnancy.

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 breast feeding. **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.

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 which 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 meta-analyses confirming that induced abortion increases a woman's risk for premature delivery.[ii], [iii]

There is acknowledgement that abortion is a risk for breast cancer made by scientists worldwide in recently published studies concerning all breast cancer risks.

In order for a study concerning breast cancer risk to be accurate, all known risks must be controlled for in the study (or case) group which has the risk to be studied and the control 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 study group who ate candy and the control group who did not eat candy 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 in for merited criticism if it found that candy increased breast cancer risk. In other words, the study group and control group of women have to be equal 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 instance, an American study[iv] looking at oral contraceptives as a risk for subtypes of breast cancer also controlled for induced abortion. 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 on this study included Louise Brinton, the chief of the Hormonal and Reproductive Section in the Division of Epidemiology at the National Cancer Institute. A paper from China looking into risk factors associated with sub-types of breast cancer found that induced abortion increased breast cancer risk[v]. Another, separate Chinese study also showed an increased risk of breast cancer with induced abortion.[vi] This study also showed an increase risk with increase in numbers of abortions. A recent Turkish study[vii] has also found induced abortion to be a risk for breast cancer. In the discussion section of this paper the authors reported that their finding was consistent with previous findings in the world's literature concerning induced abortion.

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

There is a known normal breast development during pregnancy which is consistent with all known reproductive risks of breast cancer, including induced abortion.

There are well documented, physiological changes which occur in the mother's breast with a normal pregnancy and result in a lowering of her breast cancer risk if the pregnancy goes past 32 weeks[viii]. This reduction is due to the maturing hormones produced by the fetus and placenta (afterbirth) in preparation for breast feeding.

A lobule is a unit of breast tissue consisting of milk glands and ducts which carry the milk toward the nipple. Prior to a first full-term pregnancy, the breast is about 75% Type 1 and 25% Type 2 lobules where ductal and lobular breast cancers form respectively. By the end of the pregnancy, the breast is about 85% fully matured to cancer-resistant Type 4 lobules and only 15% immature, cancer-vulnerable lobules, thereby reducing the mother's future risk of breast cancer. 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 a decrease in stroma (the surrounding connective tissue)[ix].

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, **doubles 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)[x],[xi]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) 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 to protective effect of a full-term pregnancy.[xii] **An induced abortion before 32 weeks has the same physiological effect on the breast, and differs from premature delivery only in that the fetus is delivered dead and not alive.**

The above breast physiology 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.[xiii]

Even pregnancies ending after 32 weeks but before 40 weeks gestation do not offer the maximum protection afforded by a full-term pregnancy.[xiv] 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 the genotoxic and proliferative effects of estrogen and progesterone. Estrogen levels increase 2000% 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 children late in their reproductive life.

The more menstrual cycles a woman has, (whether owing to an early age at menarche [first period] or to a late menopause), the longer her exposure to estrogen and progesterone during her menstrual cycle and the higher her risk. 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.

Breast feeding lowers a woman's risk for breast cancer because she will often stop menstruating and her cycles can be anovulatory (no release of an egg).

Spontaneous abortions (miscarriages) do not carry the same risk as induced abortions because spontaneous abortions are 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)[xv]. Women who miscarry often report having "not felt pregnant" owing to these low hormonal levels.

For these and many other reasons, I support telling a woman that an induced abortion will increase her risk of breast cancer.

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[i] Breast Cancer Epidemiology Li CI, Ed. Chapter 6 Reproductive Factors pg 122 Springer Science 2010

[ii] Swingle H et al. Abortion and the Risk of subsequent preterm birth. J Reprod Med 2009 Feb; 54(2):95-108

[iii] Shah PS et al. Induced termination of pregnancy and low birth weight: a systematic review and meta-analyses BJOG 2009 116: 1425-1442

[iv] Dolle J et al. Risk Factors for Triple-Negative Breast Cancer in Women under the age of 45 Years. Cancer Epidemiology, Biomarkers and Prevention 2009; 18(4). April 2009

[v] Xing P et al A case-control study of reproductive factors associated with subtypes of breast cancer in Northeast China,. Medical Oncology 23 Sept 2009

[vi] Yan,XU et al. Analysis of Risk Factors of Breast Cancer. J South Med Univ 2010; 30(3)

[vii] Ozmen V, et al. Breast cancer risk factors in Turkish women-a University Hospital based nested case control study. World Journal of Surgical Oncology 2009, 7:37

[viii] Hsieh C, et al. Delivery of premature newborns and maternal breast cancer risk. *The Lancet* 1999;353:1239

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[xii] J. Russo et al., "Full-Term Pregnancy Induces a Specific Genomic Signature in the Human Breast," *Cancer Epidemiology Biomarkers and Prevention* 17 (2008): 51–66.

[xiii] . Melbye M, Wohlfahrt J, Olsen JH, Frisch M, Westergaard T, Helweg-Larsen K, Andersen PK. Induced abortion and the risk of breast cancer. *N Engl J Med* 1997;336:81-5

[xiv] Hsieh C, et al. Delivery of premature newborns and maternal breast cancer risk. *Lancet* 1999;353:1239

[xv] Kunz J, Keller PJ, "HCG, HPL, Oestradiol,, Progesterone and AFP in serum in patients with threatened abortion, Br J Obstet Gynaecol 83 (1976): 640-44

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FACT SHEET Factors Which Increase and Decrease Breast Cancer Risk

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Factors Which INCREASE Breast Cancer Risk

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

Factors Which DECREASE Breast Cancer Risk

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

Factors Which HAVE NO EFFECT on Breast Cancer Risk

Factor	Reason
Saturated fat	Saturated fat intake not related to obesity
Spontaneous abortions (miscarriages) in the first trimester	No increased levels of estrogen as found in healthy pregnancies

This information is taken from Breast Cancer Risks and Prevention, 4th Edition, (Chapter 15), by Angela Lanfranchi, MD.FACS & Joel Brind, PhD. (©2007 Breast Cancer Prevention Institute)

The Breast Cancer Prevention, Risks and Prevention 4th Edition booklet and this Fact Sheet are available to view online: www.bcpinstitute.org

For booklet ordering information, please visit the Breast Cancer Prevention Institute website or call toll-free 1-866-622-6237 (1-86 NO CANCER).

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Epidemiologic Studies: Induced Abortion and Breast Cancer Risk

Updated November 2011

No.	Year	Reference	OR (95% CI)	Statistically Significant	Pos/Neg Correlation	Country/ Population
1	1957	Segi M, et al. An epidemiological study on cancer in Japan. *GANN.* 48 1957;1–63.	2.63 (1.85-3.75)	Yes	Positive	Japan
2	1968	Watanabe H, et al. Epidemiology and clinical aspects of breast cancer. [in Japanese], *Nippon Rinsho* 26, no. 8. 1968;1843–1849.	1.51 (0.91-2.53)		Positive	Japan
3	1978	Dvoyrin VV, et al. Role of women's reproductive status in the development of breast cancer. *Methods and Progress in Breast cancer Epidemiology Research* Tallin 1978;53-63.	1.71 (0.80-3.64)		Positive	USSR/ Estonia
4	1979	Burany B. Gestational characteristics in women with breast cancer. *Jugosil Ginekol Opstet* 1979;19:237-47 (in Serbo-Croatian).	0.50 (0.33-0.74)		Negative	Yugoslavia
5	1981	Pike MC, et al. Oral contraceptive use and early abortion as risk factors for breast cancer in young women. *Br J Cancer* 43, no. 1. 1981;72-6.	2.37 (0.85-6.93)		Positive	United States
6	1982	Nishhiyama, F. The epidemiology of breast cancer in Tokushima prefecture. *Shikoku Ichi* 1982; 38:333-43 (in Japanese).	2.52 (1.99-3.20)	Yes	Positive	Japan
7	1983	Brinton LA, et al. Reproductive factors in the etiology of breast cancer. *Br J Cancer* 47, no. 6. 1983:757-762.	1.2 (0.6-2.3)		Positive	United States
8	1984	Le M-G, Bachelot A, et al. Oral contraceptive use and breast or cervical cancer: Preliminary results of a case-control study In: Wolff J-P, Scott JS, eds. Hormones and sexual factors in human cancer aetiology. Amsterdam: *Elsevier* 1984:139-47.	1.3 (0.97-1.77)		Positive	France
9	1985	Hirohata T, et al. Occurrence of breast cancer in relation to diet and reproductive history: a case-control study in Fukuoka, Japan. *Natl Cancer Inst Monographs* 69 1985:187-90.	1.52 (0.93-2.48)		Positive	Japan
10	1987	LaVecchia C, et al. General epidemiology of breast cancer in northern Italy. *Intl J of Epidemiol.* 1987;16 3:347-355.	1.19 (0.82-1.71)		Positive	Italy
11	1988	Ewertz M, et al. Risk of breast cancer in relation to reproductive factors in Denmark. *Br J Cancer* 58, no. 1 1988:99-104.	3.85 (1.08-13.6)	Yes	Positive	Denmark

No.	Year	Reference	OR (95% CI)	Statistically Significant	Pos/Neg Correlation	Country/ Population
24	1994	Andrieu N, Clavel F, Gairard B, Piana L, Bremond A, Lansac J, Flamant R., Renaud R. Familial risk of breast cancer and abortion. *Cancer Detect Prevent* 1994;18(1):51-55.	1.2 (0.8-1.8)		Positive	France
25	1994	Daling JR, et al. Risk of breast cancer among young women: relationship to induced abortion. *J Natl Cancer Inst* 86, no. 21 1994;1584-92.	1.5 (1.2-1.9)	Yes	Positive	United States
26	1994	Laing AE, et al. Reproductive and lifestyle factors for breast cancer in African-American women. *Gent Epidemiol* 1994;11:A300.	2.4 (1.0-6.0)	Yes	Positive	United States
27	1995	Andrieu N, Duffy SW, Rohan TE, Le MG, Luporsi E, Gerber M, Renaud R, Zaridze DG, Lifanova Y, Day NE. Familial risk, abortion and their interactive effect on the risk of breast cancer—a combined analysis of six case-control studies. *Br J Cancer* 1995;72:744-751.	1.5 (1.1-1.9)	Yes	Positive	Multi-National
28	1994	White E, et al. Breast cancer among young US women in relation to oral contraceptive use. *J Natl Cancer Inst* 1994;86:505-14.	1.36 (1.11-1.67) [if IA before FFTP and nulliparous 1.7 (1.11-2.6)]	Yes	Positive	United States
29	1995	Brinton LA, et al. Oral contraceptives and breast cancer risk among younger women. *J Natl Cancer Inst* 1995;87:827-35.	[0.98 (0.8-1.2) if 1 IA] [1.02 (0.8-1.4) if ≥ 2 IA]		Negative	United States
30	1995	Bu L, et al. Risk of breast cancer associated with induced abortion in a population at low risk of breast cancer. *Amer J Epidemiol* 141 1995;S85.	2.9 (1.9-4.4) [if BrCa ≤ 35 yo 4.5 (1.9-10.7)] [if ≤ 2 IA 3.6 (2.2-6.0)]	Yes	Positive	China
31	1995	Lipworth L, et al. Abortion and the risk of breast cancer: a case-control study in Greece. *Intl J Cancer* 61, no. 2 1995;181-4.	1.51 (1.24-1.84) [if IA before FFTP 2.06 (1.45-2.9)]	Yes	Positive	Greece
32	1995	Rookus MA, et al. Breast Cancer risk after an induced abortion, a Dutch case-control study. *Amer J Epidemiol* 1995;141:S54 (abstract 214).	1.9 (1.2-3.1)	Yes	Positive	Netherlands
33	1996	Daling JR, Brinton LA, Voigt LF, et al. Risk of breast cancer among white women following induced abortion. *Amer J Epidemiol* 1996;144:373-380.	1.3 (1.0-1.6)	Yes	Positive	United States
34	1996	Newcomb PA, et al. Pregnancy termination in relation to risk of breast cancer. *J Amer Med Assoc* 275, no. 4 1996:283-287.	1.23 (1.0-1.51)	Yes	Positive	United States

No.	Year	Reference	OR (95% CI)	Statistically Significant	Pos/Neg Correlation	Country/ Population
47	2001	Robertson C, et al. The association between induced and spontaneous abortion and risk of breast cancer in Slovenian women aged 25-54. *Breast* 2001;10:291-298.	2.71 (0.72-10.26) if IA nulliparous		Positive	Slovenia
48	2001	Sanderson M, et al. Abortion history and breast cancer risk: Results from the Shangai Breast Cancer Study. *Intl J Cancer* 96, no. 6 2001:899-905.	1.3 (0.8-2.3) if IA ≥ 3 and post-menopausal BC		Positive	China
49	2002	Ye Z, et al. Breast cancer in relation to induced abortions in a cohort of Chinese women. *Br J Cancer* 87, no. 9. 2002:976.	1.06 (0.9-1.25) [if IA ≥ 13 wks 1.95 (0.83-4.56)] [if IA before FFTP 2.16 (0.79-5.91)]		Positive	China
50	2003	Becher H, Schmidt S, Chang-Claude J. Reproductive factors and familial predisposition for breast cancer by age 50 years. A Case control family study for assessing main effects and possible gene-environment interaction. *Intl J Epidemiol* 2003;32:38-50.	1.35 (1.03-1.78)	Yes	Positive	Germany
51	2003	Erlandsson G, et al. Abortions and breast cancer: record-based case-control study. *Intl J Cancer* 103, no. 5. 2003:676-9.	0.8 (0.63-1.02)		Negative	Sweden
52	2003	Mahue-Giangreco M, Ursin G, Sullivan-Halley J, Bernstein L. Induced abortion, miscarriage, and breast cancer risk of young women. *Cancer Epidemiol Biomarkers & Prev* 2003;12:209-214.	1.05 (0.75-1.48)		Positive	United States
53	2003	Paoletti X, Clavel-Chapelon F. Induced and spontaneous abortion and breast cancer risk: results from the E3N cohort study. *Intl J Cancer* 106, no. 2 2003:270-6.	0.91 (0.82-0.99)		Negative	France
54	2004	Meeske K, et al. Impact of reproductive factors and lactation on breast carcinomas in situ. *Intl J Cancer* 2004 110:103-109.	1.04 (0.56-1.94)		Positive	United States
55	2004	Palmer JR, et al. A prospective study of induced abortion and breast cancer in African-American women. *Cancer Causes & Control* 15, no. 2 2004:105-11.	1.1 (0.8-1.4) parous women [0.9 (0.5-1.4) nulliparous women]		Positive	United States
56	2005	Brewster DH. Risk of breast cancer after miscarriage or induced abortion: a Scottish record linkage case-control study. *J Epidemiol & Community Health* 59, no. 4 2005:283-287.	0.8 (0.72-0.89)		Negative	Scotland
57	2006	Reeves GK. Breast cancer risk in relation to abortion: Results from the EPIC study. *Intl J Cancer* 119, no. 7 2006:1741-5.	0.95 (0.87-1.03) (8 countries: 4 with positive association)		Negative	Europe

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The Cost of "Choice" Women Evaluate the Impact of Abortion Chapter 8 The Abortion Breast Cancer Link: The Science and the Studies, Editor Bachiochi, E , Encounter Books, SanFrancisco 2004

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The Abortion Breast Cancer Link, What today's evidence shows. *Ethics and Medics, The National Catholic Bioethics Center* 2003; Vol 28; No 1

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A Patient's Right to Know. *The Lancet Oncology* 4/1/2002; Vol 3 No 4:206.

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Induced abortion as a predictor for HER2 overexpression in malignant breast tumors, Breast Cancer Research & Treatment, 10/2001; Vol 69 No 3.

Lectures:Medical

11/13/10 St.Joseph's Health System, Hehman Hall, Lexington, KY
Induced Abortion as a Risk for Breast Cancer

9/13/10 Ob-Gyn Grand Rounds, Somerset Medical Center Somerville, NJ
Pregnancy Associated Breast Cancer

3/9/10 United Nations, NY 54th Commission on the Status of Women, Reproductive Breast Cancer Risks

3/3/10 United Nations, NY 54th Commission on the Status of Women Reproductive Health and Breast Cancer Risk

10/24/09 University of Dayton, Ohio, Sears Recital Hall, Reproductive Breast Cancer Risks and Prevention

1/24/09 Catholic University, Washington DC Women's Reproductive Health and Breast Cancer Risk

11/3/08 St John's Medical School, Bangalore, India, Grand Rounds, Reproductive Breast Cancer Risks

11/18/08 Vanderbilt Medical School, Nashville, TN, Lecture Reproductive Breast Cancer Risks

3/3/08 United Nations, Church Centre, Hardin Room, Commission on the Status of Women Conference, Reproductive Breast Cancer Risks

1/24/08 Geneisis Medical Center, Davenport, IA Tumor Board, Pregnancy Outcomes as Reproductive Breast Cancer Risks

1/17/08 Medical Grand Rounds at Somerset Medical Center, Somerville, NJ Reproductive Breast Cancer Risks and Breast Maturation

4/11/07 SUNY at Stony Brook Medical School, Health Science Center, Stony Brook, NY, Breast Cancer Reproductive Risks, including oral contraceptives and the Abortion Breast Cancer Link

3/30/07 Fox Chase Cancer Center, Philadelphia, PA, Reproductive Breast Cancer Risks and Breast Lobule Maturation

3/6/07United Nations, New York, NY Church Centre, 10th Floor, Commission on the Status of Women, Reproductive Risks and Breast Development

11/3/06 University of Illinois, School of Medicine, Chicago, IL Breast Cancer Risk and Lobular Development

2/7/2003. St. Vincent's Hospital, Dept. of Oncology, Sydney, Australia. **Induced abortion as a predictor of HER2 (erbB-2/neu) overexpression in malignant breast tumors.**

2/1/2003. AAPLOG (American Association for Pro Life Obstetricians and Gynecologists) Midwinter Conference, Holy Cross Hospital, Ft. Lauderdale, Florida. **Breast Cancer Risks and Prevention**

10/2001. Somerset Medical Center, Somerville, NJ. Dept. of Medicine & Family Practice Grand Rounds. **Understanding Breast Cancer Risk**

2/3/2000. Somerset Medical Center, Somerville, NJ. Dept. of Medicine Grand Rounds. **How to Handle Breast Problems in the Office or How to Survive Breast Cancer Awareness Month.**

10/15/1996. Somerset Medical Center, Somerville, NJ. Dept. of Family Practice Grand Rounds. **How to Handle Breasts in the Office: A Potpourri of Tips and Practical Information.**

Lecture Presentations: Lay Community

5/10/06 Wellington Bldg., Parliament Hill, Ottawa, Canada. **The Impact of Women's Choices of ReproductionFertility Control and Subsequent Breast Cancer Risks**

2/18/2003. National Library Auditorium, Wellington, New Zealand. **Breast Cancer Risks and Prevention.**

2/18/2003. Parliament House, Beehive Theatrette, Wellington, New Zealand. Host: Judy Turner, MP for MPs, Senators & Staffs. **Breast Cancer Risks and Prevention.**

2/14/2003 & 2/15/2003. Cancer Support Association of Western Australia, Cottesloe, W.A., Australia. **Breast Cancer Risks and Prevention.**

2/12/2003 & 2/13/2003. Bethlehem House, Adelaide, Australia. **Breast Cancer Risks and Prevention.**

2/11/2003. Parliament House, Canberra ACT, Australia. Host: Senator Brian Harradine, Parliament of Australia. Reception for MPs, Senators and Staffs. **A Call to Reason, the Abortion Breast Cancer Link.**

2/9/2003. Thomas More Center, North Melbourne, Australia. Summer School Conference. **A Call to Reason, the Abortion Breast Cancer Link.**

10/30/2002. Somerset Medical Center, Somerville, NJ, Community Lecture Series. **Total Breast Health, More Than Mammography.**

6/2001. Relay for Life, American Cancer Society of Somerset County, Hillsborough High School, Hillsborough, NJ, **Announcer.**

6/2000. Relay for Life, American Cancer Society of Somerset County, Hillsborough High School, Hillsborough, NJ, **Sponsor.**

3-10

10/17/1999. Somerset County Representative, American Cancer Society, Somerset County Chapter/Eastern Division, Making Strides Against Breast **Cancer Campaign.**

10/17/1999. American Cancer Society, Somerset County Chapter/Eastern Division, Lecture for **Reach to Recover Volunteer.**

3/20/1999. Holiday Inn, Somerset, NJ. A Woman's Day – Just for the Health of It, sponsored by Somerset Medical Center. **What Every Woman Should Know About Her Breasts.**

10/17/1998. Committee member, American Cancer Society, Somerset County Chapter/Eastern Division, Committee member of Making Strides Against **Breast Cancer Campaign.**

3/28/1998. Holiday Inn, Somerset, NJ. A Woman's Day – Just for the Health of sponsored by Somerset Medical Center. **Breakthroughs in Breast Cancer.**

4/8/95. Holiday Inn, Somerset, NJ. A Woman's Day – Just for the Health of It, sponsored by Somerset Medical Center. **Breast Health Awareness.**

10/15/1994. Somerset Medical Center, Somerville, NJ. **Life Goes On: Breast Cancer Update.**

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Education	Bronx High School of Science New York, NY	1967
	State University of New York at Stony Brook B.S. Magna cum laude	1971
	Georgetown School of Medicine M.D. Washington, D.C.	1975
Postdoctoral Training	Family Practice Residency Somerset Medical Center Somerville, NJ	1975-1978
	General Surgery Residency State University of New York at Stony Brook & Affiliated Hospitals New York	1978-1982
	Fellowship in Vascular Surgery Winthrop Univ. Hosp. (formerly Nassau Hosp.) Mineola, NY	1982-1983
Work History	Surgical Associates of Central New Jersey, PC Bound Brook, NJ, President 2003	1985-present
	Private Practice, General and Vascular Surgery Bound Brook, NJ	2/84-7/85
	Central General Hospital Plainview, NY ER Physician	7/83-2/84
Board Certification	American Board of Surgery Re-certification Re-certification American Board of Family Practice National Board of Medical Examiners	1983 1993 2003 1978 1976
Licensure	State of New Jersey #MA32005 State of New York #150231 DEA #AL2389662 New Jersey CDS #3D36460	

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