THE FIT BETWEEN THE ELEMENTS FOR AN INFORMED CONSENT CAUSE OF ACTION AND THE SCIENTIFIC EVIDENCE LINKING INDUCED ABORTION WITH INCREASED BREAST CANCER RISK

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I. INTRODUCTION

In 1994, an epidemiological study on the relationship between induced abortion and breast cancer risk, published in the *Journal of the National Cancer Institute,* made national headlines. Dr. Janet Daling and a team of researchers at Seattle’s Fred Hutchinson Cancer Research Center reported that “[a]mong women who had been pregnant at least once, the risk of breast cancer in those who had experienced an induced abortion was 50% higher than among other women.” When women underwent abortions before the age of eighteen or at age thirty or older, the study found more than a twofold (150%, or 110% higher, respectively) increase in risk. Since an average American woman’s lifetime risk of developing breast cancer is already about twelve percent, a twofold increase would imply an “absolute effect” from a single

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1. 588 F. Supp. 247, 404 (D. Utah 1984), rev’d on other grounds, 816 F.2d 1417 (10th Cir. 1987).

Epidemiology has been defined as “the study of the distribution and determinants of disease frequency in man.” KENNETH J. ROTHMAN & SANDER GREENLAND, MODERN EPIDEMIOLOGY 29 (2d ed. 1998). The two primary types of epidemiological studies are the “cohort” study and the “case-control” study. See id. at 73. A cohort study of a possible link between an exposure and a disease starts with a group who has been exposed and a group who has not, none of whom have the disease. See id. Researchers then observe how many in each cohort develop the disease over time. See MARCIA ANGELL, SCIENCE ON TRIAL: THE CLASH OF MEDICAL EVIDENCE AND THE LAW IN THE BREAST IMPLANT CASE 99 (1997). If more in the exposed group develop the disease, this difference would support the hypothesis that the exposure is a risk factor for the disease. See id. A case-control study, on the other hand, starts with a group who already has the disease (“cases”) and a group who does not (“controls”). See id. The researchers then find out how many in each group had been previously exposed. See id. If more cases than controls had been exposed, this difference would support the hypothesis that the exposure is a risk factor for the disease. See id.

4. Daling et al., *supra* note 2, at 1584.
5. See id. at 1591.
7. The difference in “average risks,” or “incidence proportions,” is called the “absolute effect,” or “excess risk” due to the exposure. See ROTHMAN & GREENLAND, *supra* note 2, at 37, 48.
induced abortion that is comparable to the risk of lung cancer from long-
term, heavy smoking.\footnote{8}

The Daling study is just one of many published since 1957 showing
a statistical link between induced abortion and the occurrence of breast
cancer.\footnote{9} In October 1996, the British Medical Association’s
Journal of Epidemiology and Community Health published a “comprehensive review
and meta-analysis” of all previously published studies including data
specifically on induced abortion and breast cancer incidence.\footnote{10} Lead
author Dr. Joel Brind, Professor of Endocrinology at Baruch College in
New York, and three co-authors from the Hershey Medical Center at
Penn State University found twenty-three studies in the worldwide
literature, nineteen of which indicate increased risk.\footnote{11} The meta-

\footnote{8}[M]en who are heavy cigarette smokers are said to have approximately a
10% lifetime risk of developing lung cancer.” \textit{Id.} at 8.
\textit{See generally} ANGELL, \textit{supra} note 2, at 165:
[O]ne of the recent studies of postmenopausal estrogen and breast cancer
showed . . . a 30 percent increase in the risk of breast cancer . . . [S]ince
we already know that 3 or 4 of every 100 post-menopausal women will get
breast cancer in the next 10 years, we could say that this study shows that
estrogen increases that risk to 5 in 100.

\footnote{9} See, e.g., M. Segi et al., \textit{An Epidemiological Study on Cancer in Japan}, 48
\textit{GANN} 1 (1957). The authors of this study, which was published in an English-language
medical journal, reported that “[t]he rate of artificial interruption of pregnancy is
significantly larger in all the subgroups among the cancer cases than the control cases.”
\textit{Id.} at 42.

\footnote{10} Joel Brind et al., \textit{Induced Abortion as an Independent Risk Factor for Breast
Cancer: A Comprehensive Review and Meta-Analysis}, 50 \textit{J. EPIDEMIOLOGY \\
& COMMUNITY HEALTH} 481 (1996).

\footnote{11} The review actually listed five of the 23 studies as negative, \textit{see id.} at 483,
but noted that one of these five studies, Louise A. Brinton et al., \textit{Oral Contraceptives and
Breast Cancer Risk Among Younger Women}, 87 \textit{J. NAT’L CANCER INST.} 827 (1995), had
in focusing on the effect of oral contraceptives adjusted its calculations for race. \textit{See
Brind et al., supra} note 10, at 486-87. Brinton et al. noted “breast cancer incidence is
higher in premenopausal African-American women than in white American women,” but
that adjusting for this difference in calculating the odds ratio for induced abortion was
questionable, “since the reason for the racial difference is unknown, and since African-
American women are vastly over represented among induced abortion patients. Thus it
is possible that adjustment for race rather than eliminating the effect of a confounding
variable, actually nullifies the effect of the variable under study.” \textit{Id.} at 487. Brinton et al.
subsequently published another analysis of their study which focused on induced
abortion. \textit{See Janet R. Daling et al., Risk of Breast Cancer Among White Women
Following Induced Abortion}, 144 \textit{AM. J. EPIDEMIOLOGY} 373 (1996). They confined this
analysis to white women, “[s]ince the reproductive histories of the black women and other
nonwhite women who participated in the study were very different from those of the white
women,” \textit{id.} at 374, and reported a statistically significant breast cancer risk increase
associated with induced abortion. \textit{See id.} at 373.
analysis of these studies showed an overall “odds ratio” or “relative risk” of 1.3, meaning that the risk of breast cancer among those who had experienced an induced abortion was thirty percent higher than among women who had not.

If this risk increase reflects a causal link, then its impact on world health is substantial. In 1986, four prominent epidemiologists criticized in The Lancet a study's suggestion that the increasing incidence of breast cancer among Swedish women was due to oral contraceptive use: “This reasoning overlooks the more likely role of other factors, especially induced abortion. Induced abortion before first term pregnancy increases the risk of breast cancer.” Among American women, forty-three percent will have an abortion at some point in their lives, if current rates

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Brind et al. also listed an additional 33 studies published to date which did not distinguish between induced and spontaneous abortions. See Brind et al., supra note 10, at 482. Since induced abortions and miscarriages affect a woman's body differently, the results of these studies are predictably inconsistent. See id. at 483.

12. “Meta-analysis” is a regularly used epidemiological technique in which raw data from different independent studies are combined and re-analyzed to “enhance the precision of the estimate of the effect size and reduce the plausibility that the association found is due to random sampling error.” Federal Judicial Ctr., Reference Manual on Scientific Evidence 174 (1994) (hereinafter Reference Manual).

13. See Brind et al., supra note 10, at 481. This figure represents the effect of induced abortion on breast cancer incidence independently of the “known increased risk attributable to a delay in the first full term pregnancy by any means.” Id. at 491. Dalig et al., on the other hand, chose not to discount this latter effect in arriving at their calculation of a 50% risk increase, thus focusing their analysis directly on the “difference in the subsequent risk of breast cancer between pregnant women who did and did not choose to terminate that pregnancy.” Dalig et al., supra note 2, at 1585.

Two other reviews of the epidemiological literature on induced or spontaneous abortion and breast cancer risk did not calculate overall estimates of risk. See Karin B. Michels & Walter C. Willett, Does Induced or Spontaneous Abortion Affect the Risk of Breast Cancer?, 7 Epidemiology 521, 521 (1996) (“Studies to date are inadequate to infer with confidence the relation between induced or spontaneous abortion and breast cancer risk, but it appears that any such relation is likely to be small or nonexistent.”); Phyllis A. Wingo et al., The Risk of Breast Cancer Following Spontaneous or Induced Abortion, 8 Cancer Causes & Control 93, 93 (1997) (“Definitive conclusions about an association between breast cancer risk and spontaneous or induced abortion are not possible at present because of inconsistent findings across studies.”).

Dr. Wingo's conservative language is in stark contrast to a more positive statement she made specifically about induced abortion over a decade earlier. See infra note 15 and accompanying text.

14. See generally Angell, supra note 2, at 164-65. The ratio between the incidence of the disease in the exposed group and the incidence in the unexposed group is called the “relative risk” in cohort studies and the “odds ratio” in case-control studies. See id. at 164.

are sustained. Bernadine Healy, M.D., former director of the National Institutes of Health, warns: "Some 1.5 million women undergo abortion in this country each year; if the breast cancer connection is valid, we will be seeing a continuous rise in breast cancer in this country for many years into the future." The incidence of breast cancer among American women has in fact been rising for the last several decades.

Dr. Stuart Donnan, editor-in-chief of the Journal of Epidemiology and Community Health, expressed his convictions concerning the abortion-breast cancer (ABC) link in an editorial about the Brind meta-analysis:

Some readers may consider that the calculation made by Brind and colleagues of possible numbers of breast cancers following—conceivably caused by—induced abortion is alarmist. It is certainly true that a relative risk of only 1.3 adds up to a large absolute increase in risk with a very high prevalence of the underlying factor. However, in the light of recent unease about appropriate but open communication of risks associated with oral contraceptive pills, it will surely be agreed that open discussion of risks is vital and must include the people—in this case the women—concerned. I believe that if you take a view (as I do), which is often called 'pro-choice,' you need at the same time to have a view which might be called 'pro-information' without excessive paternalistic censorship (or interpretation) of the data.

Nevertheless, these views contrast sharply with the disclosure practice of most abortion providers, as exemplified by the following statement by Planned Parenthood Federation of America (PPFA):

The possible link between induced abortion and breast cancer is a theory whose principal promoters oppose abortion regardless of its safety. The theory awaits conclusive confirmation by medical researchers. While Planned Parenthood believes that

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18. "Between 1940 and 1982, the age-standardized incidence rose by an average of 1.2 percent per year . . . . Between 1982 and 1986, the incidence in the United States rose more sharply, at 4 percent per year." Harris et al., supra note 6, at 319.
women should have access to information about all factors that influence the risk of disease, PPFA also believes that women deserve information that is medically substantiated and untainted by a political agenda. Because the research community has not reached a consensus on breast cancer and abortion, Planned Parenthood advises women who are considering terminating a pregnancy that there is no currently demonstrated health risk from abortion that would warrant basing a decision on that factor alone.  

Planned Parenthood justifies this position—while acknowledging the biological plausibility of a causal link—by pointing to the National Cancer Institute and the American Cancer Society, which have called


The quotation of Planned Parenthood Federation of America at several points in this Comment reflects the fact that PPFA is the only abortion provider (of which the author is aware) that has engaged in the public debate and that has made quotable statements on this issue, and is not meant to single out this organization for criticism. On the other hand, this Comment’s use of the categorical term “abortion providers” is not meant to imply a claim that no abortion providers disclose information about the ABC link to their patients.

21. [R]eproductive factors have been associated with risk for the disease since the 17th century . . . . It is known that having a full-term pregnancy early in a woman's childbearing years is protective against breast cancer . . . . The theory behind a possible link between pregnancy termination and breast cancer is based on the hormonal disruption that occurs when a woman's pregnancy is interrupted. Pregnancy initiates a surge of sex hormones (estrogen, progesterone, and prolactin), which leads to differentiation of the cells in the breast glands in preparation for lactation. The changing concentrations of hormones during the second and third trimesters of pregnancy lead to increased differentiation. In a first pregnancy, the results of these hormonal changes permanently alter the structure of the breast. Interruption during the first trimester of a first pregnancy causes a cessation of cell differentiation, which may result in a subsequent increase in the risk of cancerous growth in these tissues.

Id. at 1.

22. Physicians, as well as lay jurors, are capable of intelligently weighing the likelihood that a body of research indicates a significant potential risk:

[It is a] false belief that medical research is somehow too complex to be understood by nonscientists. This may be true of the details of any given study, but it is not true of the broad outlines. The general approach is easy to understand, because it is largely a matter of common sense. If
the body of research on the ABC link “inconclusive.” According to
the National Cancer Institute, “available studies do not permit definite
conclusions about the relationship between breast cancer and either
spontaneous or induced abortions,” while the American Cancer Society
has described induced abortion as a factor that “may be associated with
increased breast cancer risk.”

The purpose of this Comment is to show that the current level of
scientific evidence linking induced abortion with increased breast cancer
risk is sufficient to support an ethical and legal duty to disclose fully the
risk to women who are considering pregnancy termination. The
Comment approaches this goal by examining the relationship between this
evidence and the elements for a medical malpractice claim alleging failure
to obtain “informed consent.”

At its core, the common law doctrine of informed consent imposes
a legal duty on the physician to inform her patient of significant facts
about a proposed procedure so the patient can choose intelligently whether
to consent. Recognizing that the patient’s lack of vital information
about a procedure renders her apparent consent meaningless, the duty
to inform is grounded in respect for the patient’s right to self-

non-scientists had a better feeling for the approach, they could gauge the
probable strength of many scientific claims while knowing very little of the
technical details on which they are based.

Angell, supra note 2, at 91.
23. See Planned Parenthood Fact Sheet, supra note 20, at 1.
24. National Cancer Institute, Abortion and Breast Cancer (last modified Oct.
The biological hypothesis which explains why induced abortion would be expected
to increase breast cancer risk does not apply to spontaneous abortion:
The inconclusive study findings about the relationship between breast cancer
and abortion and the biologic and sociodemographic differences between
spontaneous and induced abortions argue for separate epidemiologic analyses
of these pregnancy outcomes . . . . In a viable pregnancy, human chronic
gonadotropin, serum estrogen, and serum progesterone levels rise to
predictable levels early in pregnancy . . . . Spontaneous abortion occurs when
the embryo is nonliving or the pregnancy is abnormal . . . and serum
hormone levels are below normal or do not increase as in a viable pregnancy.

Wingo et al., supra note 13, at 103-04 (footnotes omitted).
27. See, e.g., Gouse v. Cassel, 615 A.2d 331, 334 (Pa. 1992) (The Supreme
Court of Pennsylvania has “defined the scope of ‘consent’ as necessarily requiring
‘informed consent.’ Lack of informed consent is the legal equivalent to no consent.”
(citation omitted).
determination. The doctrine's historical genesis in the law of assault and battery highlights the essential role of this dignitary interest in the law of informed consent. In the often quoted words of Justice Cardozo, "[e]very human being of adult years and sound mind has a right to determine what shall be done with his own body; and a surgeon who performs an operation without his patient's consent commits an assault, for which he is liable in damages."

In addition to facilitating compensation for injured parties, establishing the duty to disclose the ABC link by demonstrating the viability of a civil claim for damages based on breach of that duty has at least three corollary advantages.

First, civil law articulates a standard of persuasion (in Wisconsin, "to a reasonable certainty by the greater weight of the credible evidence") that is more in tune with the informational needs of patients than is the much higher standard apparently now applied by abortion providers, who await "conclusive confirmation" of the link by a "consensus" of "medical researchers."

Second, as Judge Weinstein observed in In re "Agent Orange" Product Liability Litigation, the standard of persuasion ("greater than 50%") required for compensation of injuries is "far higher" than that required for prospective regulatory purposes. Therefore,

28. See, e.g., 2 LOUSELL & WILLIAMS, supra note 26, ¶ 22.01. The duty to inform is also based on the "fiduciary relationship" between physician and patient, which "exists because patients and physicians are unequal in possession of information and power to control the circumstances under which they meet. One party is fit and medically knowledgeable, the other sick and medically ignorant." RUTH R. FADEN & TOM L. BEAUCHAMP, A HISTORICAL AND THEORY OF INFORMED CONSENT 26 (1986) (footnote omitted).

29. See generally FADEN & BEAUCHAMP, supra note 28, at 121-25.


32. Planned Parenthood Fact Sheet, supra note 20 and accompanying text; cf. DeLuca v. Merrell Dow Pharm., Inc., 911 F.2d 941, 957 (3d Cir. 1990) ("The fact that a scientific community may require a particular level of assurance for its own purposes does not necessarily mean that expert opinion with somewhat less assurance is not sufficiently reliable to be helpful in the context of civil litigation.").


34. Id. at 781.

The distinction between avoidance of risk through regulation and compensation for injuries after the fact is a fundamental one. In the former, risk assessments may lead to control of a toxic substance even though the probability of harm to any individual is small and the studies necessary to assess the risk are incomplete.
demonstrating that many plaintiffs should be able to meet the higher burden required for compensation highlights the compelling need for legislative and administrative action mandating disclosure of the ABC link.

Third, the politically sensitive nature of abortion and breast cancer suggests the judiciary, through the adversarial system, may be the governmental branch initially best equipped to bring to light the true weight of the scientific evidence. Federal and state legislatures and administrative agencies have thus far failed to ensure that women are

[On the other hand, in both the regulatory and tort models, the techniques for assessment of the probabilities of risk can be similar—courts need not deny themselves the same sophisticated methods used by regulatory agencies.

Id.

35. According to an article in *Newsweek*, abortion politics have “pummeled” the scientific research on the abortion-breast cancer link. *See Sharon Begley, The Science Wars, Newsweek, Apr. 21, 1997, at 54* (examining several scientific issues that call into question the objectivity of science and which suggest that research is influenced by political and social fashions): In 1994 Daling published, in the Journal of the National Cancer Institute a study indicating that abortion increases the risk of breast cancer. *See Daling et al., supra note 2.* JNCI was evidently uncomfortable with this conclusion: it ran an editorial pointing out several ways the study might have erred. *See Lynn Rosenberg, Induced Abortion and Breast Cancer: More Scientific Data Are Needed, 86 J. Nat’l Cancer Inst. 1569-70 (1994).* In contrast, a study published this January in *The New England Journal of Medicine* found no additional risk of breast cancer after abortion. *See Mads Melbye et al., Induced Abortion and the Risk of Breast Cancer, 336 New Eng. J. Med. 81 (1997).* The editorial about it implied that the question had been resolved forever: “A woman need not worry about the risk of breast cancer” rising after an abortion. *Patricia Hartge, Abortion, Breast Cancer, and Epidemiology, 336 New Eng. J. Med. 127-28 (1997).* It did not point out that this study, no less than Daling’s, might have flaws.

Begley *supra*, at 54.

Drs. Joel Brind and Vernon Chinchilli wrote a criticism of the Melbye study, to which the authors wrote a reply, arguing that serious errors of misclassification and data adjustment had likely masked a statistically significant risk increase. *See Joel Brind & Vernon Chinchilli, Letter, Induced Abortion and the Risk of Breast Cancer, 336 New Eng. J. Med. 1834-35 (1997).*

Dr. Daling, who is strongly pro-choice, stated: If politics gets involved in science... it will really hold back the progress that we make. I have three sisters with breast cancer, and I resent people messing with the scientific data to further their own agenda, be they pro-choice or pro-life. I would have loved to have found no association between breast cancer and abortion, but our research is rock solid, and our data is accurate. It’s not a matter of believing, it’s a matter of what is.

informed of the ABC link. The 1995 Wisconsin Act regulating informed consent for abortions, for example, requires abortion providers to give patients state-printed materials describing the medical risks commonly associated with induced abortion. The Act also requires the state to be "diligent" in providing informational materials that are "objective," "accurate," and "current." Nevertheless, the pamphlet published by the Wisconsin Department of Health and Family Services, and distributed to abortion providers in April 1998, fails to mention breast cancer as a medical risk associated with induced abortion.

This Comment focuses on the elements for a cause of action predicated on negligence, which has replaced battery as the theory of liability most commonly applied to informed consent cases. Negligence traditionally requires four elements: duty, breach, causation, and injury. As applied to an informed consent claim, these four elements require the plaintiff to establish: (1) the physician had a duty to disclose information; (2) which she failed to disclose; and that (3) this failure to disclose was a legal cause of (4) the plaintiff’s injury. The causation element of an informed consent claim includes two sub-elements: the physician’s failure to disclose was a legal cause of the plaintiff’s decision to undergo the procedure (decision causation), and the procedure was a legal cause of the plaintiff’s injury (injury causation).

Part II provides an exposition and comparison of the two prevalent standards for determining which risks a physician has a duty to disclose. This Part concludes that both the “reasonable physician” and “reasonable patient” standards come down to the issue of “materiality,” and that whether a risk is material or not is ultimately a question for the trier of fact.

37. Id. § 253.10(3)(c)(2). The Act also requires the physician to orally inform the woman of “[t]he medical risks associated with the abortion procedure. Id. § 253.10(3)(c)(1).f.
38. Id. § 253.10(3)(d).
40. See, e.g., Trogun v. Fruchtman, 58 Wis. 2d 569, 597-600, 207 N.W.2d 297, 312-13 (1973). For a discussion of the differences between negligence and battery theories of informed consent, see Faden & Beauchamp, supra note 28, at 26-30. (“Because the essential purpose of the battery theory of liability is the protection of a so-called dignitary interest—the individual’s bodily integrity—no injury need result from violation of this interest. Treatment without consent is itself an actionable wrong in battery law.”).
Part III reviews the scientific evidence of the ABC link and explains why it survives both the Frye and the Daubert test for admissibility of expert testimony. Since admissibility under either standard focuses solely on principles and methods, not on generated conclusions, this Part concludes that the scientific evidence of the ABC link is clearly admissible as a basis for expert testimony, despite the lack of “general acceptance” for the conclusion that abortion increases breast cancer risk.

Part IV assesses the “materiality” of the risk posed by the ABC link by separating the risk into three interrelated components: (1) the probable consequences of developing breast cancer; (2) the increased probability that breast cancer will develop, assuming that the statistical association reflects a causal link; and (3) the probability that the statistical association does reflect a real, biological link. Emphasizing that “potential” risks are actual risks for patients facing a decision, this Part concludes that the ABC link is likely to be significant in the decision-making process of a reasonable patient, and therefore must be disclosed.

Part V discusses how a plaintiff may establish legal causation by convincing the trier of fact, to a reasonable certainty by the greater weight of the credible evidence, both that the defendant’s failure to inform was a “deciding factor” in her choice to undergo the abortion, and that the abortion was a “but for” component of the causal mechanism that produced her breast cancer.

Part VI discusses legal theories that recognize that the failure to inform is a dignitary harm, which may support a punitive damage award independently of establishing decision causation and injury causation.

Part VII concludes that all elements for an informed consent civil action can be satisfied for many plaintiffs whose breast cancer risk was increased by an induced abortion without their knowledge or consent.

II. THE BASICS OF INFORMED CONSENT

Two opinions by the Kansas Supreme Court in Natanson v. Kline were the first explicitly to apply negligence theory, rather than battery theory, to an informed consent case, largely because the plaintiff had only alleged negligence. In doing so, the court imported from the traditional cause of action for negligent medical treatment two elements that have since become controversial in the informed consent context: the requirement of proving causation, discussed below in Part V, and a duty

44. See Natanson, 354 P.2d at 672; Faden & Beauchamp, supra note 28, at 129-30.
standard determined largely by professional practice. Under this "reasonable physician" standard:

The duty of the physician to disclose . . . is limited to those disclosures which a reasonable medical practitioner would make under the same or similar circumstances . . . [T]he physician's choice of plausible courses should not be called into question if it appears, all circumstances considered, that the physician was motivated only by the patient's best therapeutic interests and he proceeded as competent medical men would have done in a similar situation.

Like the ordinary medical malpractice case, the plaintiff was required to produce "expert testimony of a medical witness" to establish that the defendant's disclosures failed to meet the professional standard.

Although adopting this seemingly natural extension of negligence principles, Natanson strongly reiterated, in language echoing the early battery cases, that the physician's duty to disclose is grounded in the patient's right to "thorough-going self-determination." In the landmark 1972 case of Canterbury v. Spence, the District of Columbia Circuit Court determined that the logic of this right required a rejection of the "reasonable physician" standard in favor of a more patient-centered approach.

The court thus formulated the "reasonable patient" standard, under which the physician must inform her patient of all "material" risks, defined as those to which "a reasonable person, in what the physician knows or should know to be the patient's position, would be likely to attach significance . . . in deciding whether or not to forego the proposed therapy." The court described risk disclosure as a "non-medical judgment," making the application of the special standard based on professional custom unnecessary and inappropriate.

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45. See FADEN & BEAUCHAMP, supra note 28, at 131.
46. Natanson, 350 P.2d at 1106.
47. Natanson, 354 P.2d at 673.
50. See id. at 783-84.
51. Id. at 787 (quoting Jon R. Waltz & Thomas W. Scheuneman, Informed Consent to Therapy, 64 NW. U. L. REV. 628, 640 (1970)).
52. Id. at 785:

There is . . . no basis for operation of the special medical standard where the physician's activity does not bring his medical knowledge and skills peculiarly into play . . . . The decision to unveil the patient's condition and the chances as to remediation . . . is oftentimes a non-medical judgment and, if so, is a decision outside the ambit of the special standard. Where that is the situation,
important corollary for a plaintiff of this change in standards was the replacement of expert witnesses with the lay jury as "certifiers" of what information ought to be disclosed.\footnote{53}

In \textit{Scaria v. St. Paul Fire & Marine Insurance Co.},\footnote{54} the Wisconsin Supreme Court followed \textit{Canterbury} in adopting the reasonable patient standard and explicitly rejected the notion that the physician's duty to disclose is necessarily limited to a "self-created custom of the profession."\footnote{55} Extrapolating from this language and citing Learned Hand's observation that "a whole calling may have unduly lagged" in the adoption of practices appropriate to new information,\footnote{56} Professor Theodore Schneyer argued that \textit{Canterbury}, \textit{Scaria}, and related cases should be read principally as a "precaution against the danger that medical disclosure practices are being influenced by personal or professional interests other than those of immediate patients and therefore are biased indicators of the information reasonable patients would find valuable in deciding whether to consent to proposed treatment."\footnote{57} Professor Schneyer discussed, as an example of judicial response to such bias, the case of \textit{Reyes v. Wyeth Laboratories},\footnote{58} in which the Fifth Circuit held the manufacturer of a polio vaccine liable for its failure to warn a child's parents of the less than one in a million chance that the vaccine would cause polio in the child. The court rejected a policy judgment by the state department of health that disclosure of the remote risk might be significant to some patients, and for that very reason, should be withheld to ensure the success of public health efforts to immunize the population against polio.\footnote{59} This policy judgment by state public health officials had formed the prevailing practice among physicians of not informing their patients of the risk of polio.\footnote{60}
The principal way in which the reasonable patient standard protects against such bias is by lifting the plaintiff's burden of producing expert testimony concerning disclosure practices. On the other hand, which standard is applied in a particular jurisdiction does not generally determine the substance of what must be disclosed. Under the reasonable patient standard, evidence of professional practice is still relevant and material in determining what the reasonable patient would want to know. Conversely, the reasonable physician standard does not deny the patient's fundamental right to self-determination, but assumes that the interaction of doctors and patients will ordinarily result in customary disclosure practices that adequately correspond to the needs of those served. Even where a whole group of physicians has "unduly lagged" in the adoption of disclosures appropriate to new information, application of the reasonable physician standard does not necessarily mean that the defective custom of that group determines the scope of disclosure required. Although under this standard the plaintiff must produce expert testimony concerning what a reasonable physician would have disclosed under similar circumstances, a physician-witness generally need not be a specialist in the field in which she is giving her opinion.

In sum, whether the duty to warn is initially viewed from the perspective of a reasonable physician or of a reasonable patient, the question should essentially come down to whether a reasonable patient would consider the information material. In other words, a reasonable physician presumably informs her patient of all risks a reasonable person in the patient’s position would likely consider significant in making her decision. Unfortunately, "[t]here is no bright line separating the significant from the insignificant." Both the reasonable physician and

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61. See supra note 53.
62. See 2 LOUSELL & WILLIAMS, supra note 26, ¶ 22.15:
   Despite the courts' often sophisticated analyses justifying the professional standard rule or the prudent patient rule, the major difference between these rules focuses not on the substance of a physician's disclosure, but on the evidentiary requirements, i.e. whether a patient must present expert testimony in establishing the standard of care.
   These general rules are merely statements of principle which emphasize that a physician must make a reasonable disclosure of significant facts that would enable a patient to intelligently decide whether to consent to the proposed treatment.
   (footnotes omitted).
63. See 2 id. ¶ 22.10.
64. See Schmeyer, supra note 53, at 157; supra note 48 and accompanying text.
66. See, e.g., REFERENCE MANUAL, supra note 12, at 58-61.
reasonable patient standards are of "limited value . . . in determining the proper scope of disclosure in a specific situation." Materiality must therefore be determined on a case-by-case basis by the trier of fact. According to the Wisconsin Supreme Court, "[w]henever the determination of what a reasonable person in the patient's position would want to know is open to debate by reasonable people, the issue of informed consent is a question for the jury."

III. THE SCIENTIFIC EVIDENCE

The scientific evidence of the ABC link is relevant in proving two elements of the contemplated informed consent cause of action. First, it establishes the existence and magnitude of the risk posed by the ABC link, and therefore the physician's duty to disclose the risk. Second, it tends to make it more probable that the failure to inform and subsequent abortion were legal causes of the plaintiff's breast cancer.

The scientific evidence of the link includes numerous epidemiological studies, a meta-analysis of those studies, and an experimental study on laboratory animals. A plausible biological hypothesis, based on known facts about pregnancy and breast cancer, explains why induced abortion would be expected to increase breast cancer risk.

This Part first describes the biological explanation for the ABC link. It then discusses the epidemiological and experimental evidence supporting the link, and demonstrates that this evidence would be an admissible basis for expert testimony.

A. A Plausible Biological Hypothesis

Biologic plausibility is an important factor in judging the likelihood that a statistical association reflects a causal link. The biologic plausibility of the ABC link rests on its consistency with existing knowledge about the "hormonal disruption that occurs when a woman's

68. 2 LOUISELL & WILLIAMS, supra note 26, ¶ 22.17.
69. See id. ¶ 22.10.
70. Johnson v. Kokomo, 199 Wis. 2d 615, 634 n.25, 545 N.W.2d 495, 503 n.25 (1996).
71. See Brind et al., supra note 10.
72. See infra note 88.
73. See supra note 168 and accompanying text. For example, the Environmental Protection Agency argued, albeit without success, that certain observations establishing the biologic plausibility that environmental tobacco smoke (ETS) is a human lung carcinogen were alone sufficient to classify ETS as a Known Human (Group A) Carcinogen. See Flue-Cured Tobacco Coop. Stabilization Corp. v. United States Envtl. Protection Agency, 4 F. Supp. 2d 435, 450-51 (M.D.N.C. 1998).
pregnancy is interrupted.”

Two independent hormonal effects are implicated: the cancer-promoting effect of early pregnancy and the protective effect of late pregnancy.

Within a few days after a woman conceives, her body begins to secrete large quantities of estrogen, a hormone that causes “immature” breast cell tissue to grow. These proliferating cells are in a transitional state and thus especially susceptible to carcinogenic stimuli and cancerous mutations. “The more the cells proliferate, the greater the chance that a replication will go awry, producing a cancerous cell.”

Most other acknowledged risk factors for breast cancer are likewise associated with estrogen exposure, including early first menstruation and late menopause. On the other hand, miscarriage, which has generally not been statistically associated with an increased risk of breast cancer, typically occurs when estrogen levels fail to rise above normal, non-pregnant levels.

In late pregnancy, other hormones induce the breast tissue to differentiate into mature, milk-producing cells that are no longer susceptible to potentially cancerous growth. “It is known that having a full-term pregnancy early in a woman’s childbearing years is protective against breast cancer.” Delaying this protective, differentiating effect of a completed pregnancy presumably increases the time period during

74. Planned Parenthood Fact Sheet, supra note 20.
75. “A full-term pregnancy may have opposing influences on risk of breast cancer: a short-term increase in risk due to the growth-enhancing properties of pregnancy estrogens, and a long-term decrease in risk from terminal differentiation of mammary tissue. Abortion, as an incomplete pregnancy, might similarly affect breast cancer risk.” Michels & Willett, supra note 13, at 421 (footnotes omitted).
76. See, e.g., CHARLES B. SIMONE, BREAST HEALTH: WHAT YOU NEED TO KNOW ABOUT DISEASE, PREVENTION, DIAGNOSIS, TREATMENT, AND GUIDELINES FOR HEALTHY BREAST CARE 147 (1995).
77. See id.
78. Healy, supra note 17, at 237.
79. See id.
80. See, e.g., Daling et al., supra note 2, at 1584.
81. See, e.g., Wingo et al., supra note 13, at 103-04.

In a viable pregnancy, human chronic gonadotropin, serum estrogen, and serum progesterone levels rise to predictable levels early in pregnancy. Thus, induced abortion usually interrupts the increasing hormone levels present in a viable pregnancy. Spontaneous abortion occurs when the embryo is nonliving or the pregnancy is abnormal... and serum hormone levels are below normal or do not increase as in a viable pregnancy.

(footnotes omitted).

82. See, e.g., SIMONE, supra note 76, at 147.
83. Planned Parenthood Fact Sheet, supra note 20.
which undifferentiated breast tissue can accumulate potentially [cancerous]
mutations.”

Induced abortion, therefore, increases a woman’s risk of breast
cancer in two independent ways: first, by subjecting the breasts to a
prolonged high dosage of estrogen, which may promote tumor growth as
well as the proliferation of immature, vulnerable cells; and second, by
abrogating a completed pregnancy’s protective effect. Strictly
speaking, the pregnancy itself, not its termination, increases the estrogen
exposure. However, the induced abortion not only forgoes the protective
effect, but does so at a time when estrogen overexposure has left the
breasts with an abnormally high number of cells vulnerable to cancerous
growth.

The plausibility of this biological explanation for the statistical
association between induced abortion and breast cancer is admitted even
by those reluctant to draw a causal inference. According to Dr. Clark
Heath, Vice President of the American Cancer Society, if the division and
maturation of the breast cells are “interrupted at a stage early on, perhaps
that will lead to an increased risk of cancer, of carcinogenesis, which is
more frequent when cells are rapidly dividing and are young.” Planned
Parenthood acknowledges that “[i]nterruption during the first
trimester of a first pregnancy causes a cessation of cell differentiation,
which may result in a subsequent increase in the risk of cancerous growth
in these tissues.”

**B. Studies Supporting the Hypothesis**

A 1980 landmark study on laboratory animals by Jose and Irma
Russo is regularly cited for the biologic hypothesis that an abortion
foretells the protective effect of breast cell differentiation in late
pregnancy, thereby increasing the risk of cancer. The authors reported

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84. Brind et al., supra note 10, at 491.
85. See id.
86. Testimony of Dr. Clark Heath at 165, Christ’s Bride Ministries, Inc. v.
(3d Cir. 1998) (No. 96-3631).
87. Planned Parenthood Fact Sheet, supra note 20.
88. Jose Russo & Irma H. Russo, Susceptibility of the Mammary Gland to
Carcinogenesis, 100 AM. J. PATHOLOGY 497 (1980).
89. See, e.g., Brind et al., supra note 10, at 481; Daling et al., supra note 2, at
1591; Holly L. Howe et al., Early Abortion and Breast Cancer Risk among Women under
Age 40, 18 INT’L J. EPIDEMIOLOGY 300, 303 (1989); Melbye et al., supra note 35, at 81
(footnote omitted) (“Russo and Russo have proposed that . . . an abortion forsestalls the
late protective effect of differentiation, thereby increasing the risk of breast cancer.”);
Michels & Willett, supra note 13, at 521; Wingo et al., supra note 13, at 94.
that seventy-seven percent of rats whose first pregnancy was artificially aborted developed breast cancer after subsequent exposure to a chemical carcinogen, compared to zero percent of similarly exposed rats in the control group whose pregnancy was carried to term.\textsuperscript{90} Seventy-one percent of rats that were never allowed to get pregnant developed breast cancer after the same carcinogen exposure.\textsuperscript{91} Russo and Russo also noted in 1980 that “epidemiologic observations indicate that there are factors that exert either a protective or a stimulating influence on the development of breast cancer in women. Among the protective factors are a first full-term pregnancy before 24 years of age . . . . Among the risk factors are . . . abortion.”\textsuperscript{92}

Since the publication of the 1996 Brind meta-analysis,\textsuperscript{93} six out of the eight epidemiological studies that have become available indicate a positive statistical association,\textsuperscript{94} bringing the total to twenty-five out of thirty-one. Seventeen of the twenty-five positive studies are “statistically significant,” a technical term meaning that the data provide at least ninety-five percent certainty that the association is not simply due to random error.\textsuperscript{95} Nevertheless, Dr. Mads Melbye, the lead author of a 1997 cohort study reporting no overall positive association,\textsuperscript{96} told the \textit{Wall Street Journal}, “I think this settles it. Definitely—there is no overall increased risk of breast cancer for the average woman who has had an abortion.”\textsuperscript{97} This extraordinary claim,\textsuperscript{98} echoed in the \textit{New England

\textsuperscript{90} See Russo & Russo, supra note 88, at 502.

\textsuperscript{91} See id.

\textsuperscript{92} Id. at 497.

\textsuperscript{93} Brind et al., supra note 10.

\textsuperscript{94} The eight studies are the following: N. Andrieu et al., \textit{Familial Risk, Abortion and Their Interactive Effect on the Risk of Breast Cancer—A Combined Analysis of Six Case-Control Studies}, 72 BRIT. J. CANCER 744 (1995) (contains data from studies of Luporsi et al., Rohan et al., and Zaridze et al.); L. Bu et al., \textit{Risk of Breast Cancer with Induced Abortion in a Population at Low Risk of Breast Cancer}, 141 AM. J. EPIDEMIOLOGY, at S85 (1995); Melbye et al., supra note 35; J.R. Palmer et al., \textit{A Case-Control Study of Induced and Spontaneous Abortion in Relation to Breast Cancer}, 143 AM. J. EPIDEMIOLOGY, at S32 (1996); Alessandra Tavani et al., \textit{Abortion and Breast Cancer Risk}, 65 INT'L J. CANCER 401 (1996); and A.H. Wu et al., \textit{Menstrual and Reproductive Factors and Risk of Breast Cancer in Asian-Americans}, 73 BRIT. J. CANCER 680 (1996) (literature search conducted by Dr. Joel Brind). The two studies not reporting a positive association are Zaridze et al. and Melbye et al.

\textsuperscript{95} See REFERENCE MANUAL, supra note 12, at 152-53.

\textsuperscript{96} Melbye et al., supra note 35, at 81.


\textsuperscript{98} According to Dr. Karin Michels of Harvard Medical School, “[y]ou should never end a debate with one study and say this is the definitive study.” \textit{Id.; cf.} Michels & Willett, supra note 13.
Journal of Medicine editorial accompanying the study was based on the study’s large sample size and on its exclusion of possible recall bias. However, Drs. Joel Brind and Vernon Chinchilli argued in a letter to the New England Journal of Medicine that serious errors of misclassification and data adjustment in the Melbye study had likely masked a statistically significant risk increase. Moreover, the Melbye study itself reported that "[w]ith each one-week increase in the gestational age of the fetus...there was a 3 percent increase in the risk of breast cancer," from a 0.81 relative risk associated with induced abortion at less than seven weeks of gestation, up to a statistically significant 1.89 relative risk associated with induced abortions after eighteen weeks of gestation. Melbye et al. reported that this trend was itself statistically significant, and concluded:

Induced abortion had no overall effect on the risk of breast cancer, but we found a statistically significant increase in risk among women with a history of second-trimester abortion. The fact that such an increase did not affect the overall result clearly indicates that it is based on small numbers and therefore requires cautious interpretation. The increased risk among women who had had second-trimester abortions finds biologic support in experiments in rats and is in line with the hypothesis of Russo and Russo.

...We cannot explain why a very early induced abortion was associated with a slight, although insignificant, decrease in risk.

99. See Hartge, supra note 35.
100. See Lagnado, supra note 97. "Recall bias" is discussed infra Part IV.C.1.
101. See Brind & Chinchilli, supra note 35.
102. Melbye et al., supra note 35, at 83.
103. Id.
104. Id. at 84; cf. Daling et al., supra note 11, at 379:

If induced abortion does increase a woman’s risk of breast cancer, it may be a consequence of the massive growth that the breast undergoes during the first trimester not being followed (because of pregnancy interruption) by cell maturation and differentiation during the second and third trimesters. This hypothesis would predict that a woman’s risk associated with induced abortion might be particularly high for pregnancies that extend through much of the first trimester before the abortion occurs.
C. The Admissibility of Expert Testimony Based on Scientific Evidence

Prior to the United States Supreme Court's decision in Daubert v. Merrell Dow Pharmaceuticals, Inc.\textsuperscript{105} in 1993, the dominant approach to determining the admissibility of expert testimony was the so-called "general acceptance," or Frye, test. In the course of deciding whether to admit expert testimony based on the results of a polygraph test, the court in Frye v. United States\textsuperscript{106} stated: "While courts will go a long way in admitting expert testimony deduced from a well-recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs."\textsuperscript{107}

The Daubert Court affirmed that the focus of the admissibility inquiry must remain "solely on principles and methodology, not on the conclusions that they generate."\textsuperscript{108} In addition, Daubert theoretically extended even further the lengths to which the courts should go in admitting expert testimony deduced from a "scientific principle," by holding that under the Federal Rules of Evidence, "general acceptance" of the relied-upon principle was no longer a necessary precondition to admissibility.\textsuperscript{109} Daubert is therefore widely viewed as representing a move beyond a rigid adherence to scientific orthodoxy in favor of a rule that more accurately reflects the dynamic nature of science.\textsuperscript{110}

Daubert thus requires the judge to determine "nothing less" than whether the methods relied on by the expert for his testimony are "scientific."\textsuperscript{111} "General acceptance" can still be an important factor in making this determination.\textsuperscript{112} The Court also mentioned three other factors that judges ruling on admissibility might take into account: whether the method employed by the expert has been subjected to peer review and publication; whether it can be and has been tested; and whether the known or potential rate of error is acceptable.\textsuperscript{113} However,

\textsuperscript{105} 509 U.S. 579 (1993).
\textsuperscript{106} 293 F. 1013 (D.C. Cir. 1923).
\textsuperscript{107} Id. at 1014.
\textsuperscript{108} Daubert, 509 U.S. at 595.
\textsuperscript{109} See id. at 597.
\textsuperscript{110} See, e.g., Heidi Li Feldman, Science and Uncertainty in Mass Exposure Litigation, 74 Tex. L. Rev. 1, 17-18 (1995) ("A dynamic enterprise like science... produces a range of opinions, some more widely shared than others. A rigid general acceptance standard... gives the legal factfinder an edited version of science. Strip away the editing, as Daubert has done, and the factfinder will more often encounter scientific uncertainty.").
\textsuperscript{111} Daubert v. Merrell Dow Pharms., Inc., 43 F.3d 1311, 1315 (9th Cir. 1995).
\textsuperscript{112} See Daubert, 509 U.S. at 594.
\textsuperscript{113} See id. at 593-94.
these factors do not constitute a “definitive checklist or test,” nor is each factor necessarily applicable in every case.114

“Peer review” refers to the process by which scientific journals normally screen submissions for methodological error.112 Publication in a peer-reviewed journal is a “significant indication” that a study is “taken seriously by other scientists,”116 and that its methods are “generally accepted” as sound by the scientific community.117 The thirty-one epidemiological studies of the ABC link, as well as the Brind meta-analysis and the Russo and Russo animal study, were all published in recognized scientific journals and based on generally accepted scientific methods.

As noted by the Third Circuit, “[t]he reliability of expert testimony founded on reasoning from epidemiological data is generally a fit subject for judicial notice; epidemiology is a well-established branch of science and medicine, and epidemiological evidence has been accepted in numerous cases.”118 Although individual epidemiological studies are subject to criticism, such claims of methodological error or bias go to the weight rather than the admissibility of testimony based on those studies.119

Some courts have regarded statistical significance as critical to a study's admissibility.120 However, statistical significance should not be confused with the practical significance of a research finding for public health.121 The Utah federal district court noted in Allen v. United States that “[t]he cold statement that a given relationship is not ‘statistically significant’ cannot be read to mean ‘there is no probability of a relationship.’”122 An epidemiologist whose sample size was too small to reveal an association with ninety-five percent certainty might still be ninety or eighty percent confident that the association measured is not due to random error. Thus, “[p]eremptorily rejecting all studies that are not statistically significant would be a cursory and foolish judgment,

114.       Id. at 593.
115.       See Daubert, 43 F.3d at 1318.
116.       Id.
117.       See generally ANGELL, supra note 2, at 129.
120.       See, e.g., Brock v. Merrell Dow Pharm., Inc., 874 F.2d 307, 312 (5th Cir. 1989), amended by 884 F.2d 167, 167 (5th Cir. 1989).
121.       See REFERENCE MANUAL, supra note 12, at 177.
particularly if there are multiple studies tending to show a consistent effect." 123

The epidemiologic technique of meta-analysis,124 although not without critics in the epidemiological community,125 is regularly employed in peer-reviewed scientific journal articles.126 Dr. Brind’s meta-analysis was admitted into evidence as the basis for his expert testimony in a 1996 lawsuit.127 Dr. Clark Heath, Vice President of the American Cancer Society, appeared as an expert witness for the opposing party.128 While testifying that “statistical associations, however statistically significant they may seem to be, don’t guarantee a biologic association,” Dr. Heath admitted that Dr. Brind’s report is “quite extensive and the statistical coverage of the papers and the literature that bear on the induced abortion question is quite thorough.”129 Dr. Janet Daling of the Fred Hutchinson Cancer Research Center has likewise described Dr. Brind’s meta-analysis as “very objective and statistically beyond reproach.”130

Although experiments on animals are generally viewed as less probative than epidemiological studies of human populations,131 the Russo and Russo study should also be admissible as a basis for expert testimony. A federal district court in Longmore v. Merrell Dow Pharmaceuticals, Inc.132 admitted animal studies as evidence of causation, despite an opposing epidemiological record of more than thirty-five studies finding no statistically significant causal link.133 The court reasoned that an epidemiological study might label an observed association as statistically insignificant, even though the “certainty that the observed increase is related to its hypothetical cause rather than mere

124. See REFERENCE MANUAL, supra note 12.
126. See In re Paoli R.R. Yard PCB Litig., 916 F.2d 829, 857 (3d Cir. 1990) ("[H]undreds of meta-analyses are done each year.").
128. Dr. Heath’s background as an expert in “leukemia clusters” is described in JONATHAN HARR, A CIVIL ACTION 41-43 (1995).
131. See REFERENCE MANUAL, supra note 12, at 130-31 n.23.
133. See id. at 1118.
chance is still far more likely than not."  

The court concluded that the defendant's epidemiological evidence did not "overwhelm" the admissibility of the animal studies because such studies are also generally relied upon by experts in determining causal links.  

IV. EXISTENCE OF A DUTY/MATERIALITY OF THE EVIDENCE

The scope of the physician's duty to warn extends to all material risks. A risk is material if a reasonable patient, in what the physician knows or should know to be the patient's position, would be likely to attach significance to the risk in deciding whether or not to forego the proposed procedure. This standard is distinct from the issue of causation, triggering the physician's duty to warn even when disclosure of the risk would not in fact have caused the particular patient to forego the procedure.

Although the standards are different, both inquiries imply a weighing of the risks posed by the procedure against the seriousness of the condition being treated. Consequently, the fact that induced abortion is ordinarily an elective procedure, which is rarely necessary to preserve the life or health of the mother, is important in framing both the questions of materiality and of causation. Furthermore, while the social and economic pressures of an unplanned pregnancy may often result in a decision to abort despite information about the increased risk of breast cancer, such pressures do not preclude a woman from attaching significance to the risk in deciding whether to undergo the procedure.

Having established the evident but important point that induced abortion is a choice, this Part analyzes the nature of the risk posed by the ABC link. It suggests that a trier of fact is likely to find that the ABC link is a material risk in light of other risk factors that are acknowledged and disclosed by government agencies and in light of similar fact patterns in past informed consent actions. To facilitate the analysis, this Part separates the risk posed by the ABC link into three interrelated

134. Id. at 1119 (quoting Allen v. United States, 588 F. Supp. 247 (D. Utah 1984), rev'd on other grounds, 816 F.2d 1417 (10th Cir. 1987)).
135. See id. at 1120.
136. See, e.g., 2 LOUISELL & WILLIAMS, supra note 26, ¶ 22.10.
138. A survey published by the Alan Guttmacher Institute indicated that the two most important reasons for obtaining abortions are "can't afford baby now" (21%) and "is unready for responsibility" (21%). Aida Torres & Jacqueline Darroch Forrest, Why Do Women Have Abortions?, 20 FAMILY PLANNING PERSPECTIVES 169, 170 (1988). Three percent of the women surveyed cited a personal health problem as the most important reason for having the abortion. See id. at 170.
components: (1) the probable consequences of developing breast cancer; (2) the increased probability that breast cancer will develop, assuming that the statistical association reflects a causal link; and (3) the probability that the association does reflect a real, biological link. This Part concludes by addressing the likely significance of “potential” risks to the decisions of reasonable patients.

A. The Probable Consequences of Developing Breast Cancer

The first component is well-defined and uncontroversial: breast cancer is a potentially debilitating and deadly disease. According to the American Cancer Society, twenty percent of women die within five years after a diagnosis of breast cancer, while the ten-year mortality rate is thirty-seven percent. Many women who develop breast cancer must undergo a breast amputation, or mastectomy, in order to prevent the cancer from spreading. These potential consequences of developing breast cancer are ingrained in the national consciousness. The prevention of breast cancer is a high priority for the federal government and many women’s groups. The fear of this disease is therefore very reasonable, and for most women quite real.

Whenever the possible consequences of a procedure are extremely serious, the courts will generally find a duty to disclose the risk. The question of foreseeability and duty, including the duty to warn, is not one of mathematical probability alone:

The odds may be a thousand to one that no train will arrive at the very moment that an automobile is crossing a railway track, but the risk of death is nevertheless sufficiently serious to require the driver to look for the train . . . . As the gravity of the possible harm increases, the apparent likelihood of its occurrence need be correspondingly less.

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141. See 2 Louisell & Williams, supra note 26, ¶ 22.16.
B. The Increased Probability of Developing Breast Cancer

The second component of the risk posed by the abortion-breast cancer link is roughly defined by the magnitude of "relative risk" measured in the epidemiological literature. Relative risk is the ratio of the incidence of disease in exposed individuals compared to the incidence in unexposed individuals. A relative risk of 1.5 would mean that the risk of breast cancer is increased by fifty percent in women who have undergone an induced abortion, while a 2.0 relative risk would indicate that exposed individuals are twice as likely to develop breast cancer as unexposed individuals.

The Brind meta-analysis of the worldwide epidemiological literature on the ABC link reported an overall 1.3 relative risk. A 1989 study on New York women by Howe et al. found a 1.9 relative risk. The risks are higher in certain subgroups of women. Two studies by Laing et al. in 1993 and 1994 on African-American women indicated relative risks of 3.1 and 2.44 respectively. The 1994 Daling study, while finding an overall relative risk of 1.5, reported a relative risk of 2.5 when the abortion was done before age eighteen and a relative risk of 2.1 when procured over age thirty. The Daling study also reported that a woman with a family history of breast cancer who had had an abortion was 1.8 times more likely to develop breast cancer than a woman with a family history who had not. When a woman with a family history underwent the abortion at age thirty or older, her relative risk was 3.7. If the abortion occurred prior to age eighteen, her relative risk was immeasurably high, since all twelve such women in the study had developed breast cancer.

The above figures represent the risk elevation associated with induced abortion independently of, and in addition to, the known increased breast cancer risk of delaying the protective effect of a first full-term pregnancy, except for those from the Daling study, which included both effects. Although these studies separated out as a confounding factor the effect of delaying a first full-term pregnancy in order to isolate the independent

144. See id.
145. See Brind et al., supra note 10, at 481.
146. See Howe et al., supra note 89, at 300.
147. See Brind et al., supra note 10, at 483, 486.
148. See Daling et al., supra note 2, at 1585.
149. See id. at 1588.
150. Id.
151. Id.
152. See Brind et al., supra note 10, at 481, 483; supra note 13.
effect of an induced abortion, both effects are relevant in determining the actual risk elevation associated with aborting a pregnancy. A woman contemplating abortion is already pregnant. If the pregnancy is not artificially interrupted, she will benefit from the natural protective effect of a completed pregnancy. Therefore, the total risk increase associated with an induced abortion should be measured relative to the reduced average lifetime risk of a woman who has completed a pregnancy, rather than to that of a woman who has never been pregnant. The bottom line is that a woman considering abortion has two alternatives, and that her lifetime risk of breast cancer is significantly affected by which alternative she chooses. Consequently, if the plaintiff was under age thirty and had not yet had a full-term pregnancy at the time of the abortion, the risk increase associated with delaying a first full-term pregnancy should be factored in. A summary of the results of twenty-three previous studies of this effect estimated that a woman who completes her first pregnancy at age thirty or later has a ninety percent greater risk of breast cancer than a woman who completes her first pregnancy before the age of twenty.153

The real significance of a relative risk increase depends upon the background risk which is increased. For example, although smoking increases the risk of lung cancer by a factor of 10.0, the background risk of lung cancer for nonsmokers is very low.154 By contrast, an average American woman's lifetime risk of breast cancer is about twelve percent.155 A 1.3 relative risk increase from an induced abortion would therefore indicate about a four percent increase in absolute terms. Estimating a twenty-five percent mortality rate,156 this figure would suggest that about 1 out of 100 women who have had an induced abortion die from breast cancer attributable to the abortion.

According to the American Civil Liberties Union, "even a 1 in 10,000 risk of death must always be disclosed."157 The courts have

155. See supra note 6.
156. See AMERICAN CANCER SOC'y, supra note 139 and accompanying text.
157. GEORGE ANNAS, THE RIGHTS OF PATIENTS: THE BASIC ACLU GUIDE TO PATIENT RIGHTS 86 (2d ed. 1992). In the regulatory context:

The gravity of the cancer risk is judged by how far the risk is above the "acceptable" or de minimis, risk level ... In the case of large populations at risk of cancer, a review of one hundred thirty-two federal regulatory decisions disclosed that a de minimis level of one lifetime death per million population at risk is generally used by federal agencies ... In 1990, Perrier Corporation recalled its entire worldwide stock of bottled water because of benzene contamination, at a cost of over seventy million dollars in the United
likewise generally found that a physician must inform her patient of risks of such magnitudes. In *Hartke v. McKelway*, for example, the District of Columbia Circuit Court affirmed that the jury could conclude that a three in 1000 chance that a laparoscopic cauterization performed to prevent pregnancy would fail was a material risk, given the plaintiff's history of pregnancy-related health problems and the availability of other alternatives.

C. The Probability of a Causal Link Between Induced Abortion and Breast Cancer

The probability that the statistical association between induced abortion and breast cancer reflects a biologic relationship is the third and most critical risk component, on which the previous two depend for relevance. The causality of the increased risk associated with induced abortion's delay of a full-term pregnancy's protective effect is not controversial, and alone supports a duty to inform. On the other hand, the scientific community has not yet reached a consensus on whether induced abortion has a stimulating biologic effect on the development of breast cancer in women.

"[E]pidemiology cannot prove causation," nor can it quantify statistically the likelihood of a causal link. Rather, "causation is a judgment issue," and making such judgments is arguably not part of science at all. Deciding how much data is needed to justify a public

States alone. Perrier’s recall was prompted by an FDA risk assessment which showed that the lifetime risk of cancer from drinking two bottles of Perrier per day was one death per million exposed.


158. See 2 LOUISELL & WILLIAMS, supra note 26, ¶ 22.16.
159. 707 F.2d 1544 (D.C. Cir. 1983).
160. See id. at 1548-49.
161. *REFERENCE MANUAL*, supra note 12, at 157. On the other hand:

Perhaps the most important common thread that emerges from the debated [modern scientific] philosophies is Hume's legacy that proof is impossible in empiric science. This simple fact is especially important to epidemiologists, who often face the criticism that proof is impossible in epidemiology, with the implication that it is possible in other scientific disciplines.

ROTHMAN & GREENLAND, supra note 2, at 22.
162. "Statistical methodology does not permit assessments of those probabilities."

*REFERENCE MANUAL*, supra note 12, at 153 n.80.
163. *Id.* at 157.
164. See *id.* at 157 n.93; *cf.* supra note 22.
health effort is "strongly influenced by economic and political considerations as well as by societal values." 165

Furthermore, "[n]ot all researchers are conservative when it comes to assessing causal relationships, often calling for stronger evidence and more research before a conclusion of causation is drawn." 166 In the context of the ABC link, one might expect this conservative tendency to be strengthened by researchers’ apprehension that declaring a causal relationship between abortion and breast cancer could have serious societal and political consequences. Moreover, an individual epidemiologist’s natural reluctance to draw a conclusion not held by most peers, especially when epidemiology cannot prove that inference, is readily understandable. These extra motivations to avoid premature judgments and to wait for greater certainty potentially widen the gap between the standard of proof implicitly applied by the epidemiologist and the standard of proof required in civil litigation. 167 Therefore, the fact that many epidemiologists decline to infer a causal link between induced abortion and breast cancer should not dissuade a lay juror from drawing such a conclusion if she finds herself convinced by a preponderance of the available evidence.

The Federal Judicial Center’s Reference Manual on Scientific Evidence lists the following five factors as important guidelines in judging the likelihood that a statistical association reflects a causal link: (1) consideration of alternative explanations; (2) strength of the association; (3) consistency of the association; (4) biologic plausibility; and (5) temporal relationship. 168 Since the biologic plausibility169 and

166. REFERENCE MANUAL, supra note 12, at 157.
167. See DeLuca v. Merrell Dow Pharm., Inc., 911 F. 2d 941, 957 (3d Cir. 1990) ("The fact that a scientific community may require a particular level of assurance for its own purposes ... does not necessarily mean that expert opinion with somewhat less assurance is not sufficiently reliable to be helpful in the context of civil litigation.").
168. See REFERENCE MANUAL, supra note 12, at 161. The Reference Manual also lists two other factors—"specificity of the association" and "dose-response relationship," but notes that these factors "differ in significant ways from the five factors mentioned above. Although the presence of specificity and dose-response strengthens the inference of causation, the absence of either does not weaken the inference. Epidemiologists have begun to question the use of these two factors as guidelines for causation in non-infectious diseases." Id. at 163.
169. See supra notes 86-87.
temporality of the link are generally not disputed, only the first three factors are applied to the ABC link in the following discussion.

1. CONSIDERATION OF ALTERNATIVE EXPLANATIONS

Two types of "alternative explanations" for the statistical association must be considered and ruled out: possible confounding factors and methodological bias. A confounding factor in the ABC link context would be something other than abortion that is both a risk factor for breast cancer and associated with abortion. Epidemiological studies control for confounding variables through what is called a "multivariate analysis." The following potential confounding variables "were generally included in the multivariate analyses" of the abortion-breast cancer studies: age at first full-term pregnancy, number of children borne, age at first menstruation, oral contraceptive use, and "some measure of socio-economic status (usually education level)." The Brind meta-analysis also took into account these variables by recalculating the relative risk using only those studies that had accounted for the respective variable and confirming that the recalculation made no significant difference to the relative risk.

The most common criticism of the body of scientific evidence supporting the ABC link concerns the potential role of "recall bias" in studies that rely on retrospective patient interviews. This theory, singled out by the National Cancer Institute as "[p]erhaps the most serious potential weakness" of the ABC link, hypothesizes that women with breast cancer are significantly more likely to accurately report having had an abortion than women without breast cancer, leading to a false association between the two. However, the authors of the study cited by the National Cancer Institute, which is the only study ever claiming to provide statistically significant direct evidence of this hypothesis, have

170. The temporality criterion refers to the requirement that a cause must precede its effect in order to support a causal inference. See Reference Manual, supra note 12, at 162. A study must ensure that exposure to induced abortion preceded the occurrence of breast cancer in study participants in order to satisfy the temporality requirement.

171. See id. at 156.
172. "A confounding factor is both a risk factor for the disease and associated with the exposure of interest." Id. at 173. For example, in studying a potential causal relationship between gray hair and mortality, old age would be a confounding factor.
173. See id. at 160. Multivariate analysis is a "set of techniques used when the variation in several variables has to be studied simultaneously." Id. at 175.
174. Brind et al., supra note 10, at 492.
175. See id.
176. National Cancer Institute, supra note 24.
since retracted the key assumption on which their finding was based.\textsuperscript{177} Several other studies have explicitly tested for recall bias and found none.\textsuperscript{178}

It is never possible to rule out every alternative explanation for a statistical association, and there is always a chance that an unknown confounding factor is responsible.\textsuperscript{179} However, controlled experiments on laboratory animals are not susceptible to confounding or recall bias.\textsuperscript{180} The results of the 1980 Russo and Russo study therefore lessen the plausibility that the ABC link is explained by these factors.\textsuperscript{181}

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Dr. Brind described the method and conclusion of the study as follows: "[T]heir computer records showed that seven of the women who had reported having an induced abortion had no record of it on the computer. And, they concluded that the computer was accurate and they relied upon this statistic to say that there was this statistically significant difference." Testimony of Dr. Joel Brind at 63, Christ's Bride Ministries, Inc. v. Southeastern Pa. Transp. Auth., 937 F. Supp. 425 (E.D. Pa. 1996), rev'd, 148 F.3d 242 (3d Cir. 1998) (No. 96-3631).

Dr. Daling and her co-authors, whose own study explicitly tested for recall bias and found none, said of the Swedish study: "[W]e believe it is reasonable to assume that virtually no women who truly did not have an abortion would claim to have had one." Daling et al., \textit{supra} note 2, at 1590. Daling recalculated the data from the Lindefors-Harris study with the "overreporting" of the seven women eliminated, which reduced the supposed risk increase due to recall bias from a statistically significant 50% to a non-significant 16%. See id.

The authors of the Swedish study have since admitted that "[s]ome women . . . had induced abortions abroad or unrecorded terminations of pregnancy." Olav Meirik et al., Letter, \textit{Relation Between Induced Abortion and Breast Cancer}, 52 J. Epidemiology Community Health 209 (1998) (footnote omitted).

178. See Daling et al., \textit{supra} note 2, at 1590; Howe et al., \textit{supra} note 89, at 303; Loren Lipworth et al., \textit{Abortion and the Risk of Breast Cancer: A Case-Control Study in Greece}, 61 Int'l J. Cancer 181 (1995).

The 1989 Howe cancer registry study, which precluded by its methodology even the possibility of recall bias (and which found a 1.9 relative risk), observed that "underreporting and inconsistent reporting" of abortion histories on fetal death certificates "occurred similarly among the cases and the controls." Howe et al., \textit{supra} note 89, at 303.

179. See REFERENCE MANUAL, \textit{supra} note 12, at 159, 163.

180. See \textit{id.} at 130.

2. CONSISTENCY OF THE ASSOCIATION

"Consistency" refers to the replication of an observed association in different populations under different circumstances and is an important factor in judging the likelihood of causation. The charge is sometimes made that the epidemiological literature on the ABC link is "inconsistent." For example, in 1996 the National Cancer Institute described the epidemiological literature as follows:

Three recent reviews published in scientific journals have assessed more than 30 studies and concluded that the available data on the relationship between induced abortions or spontaneous abortions (miscarriages) and breast cancer are inconsistent and inconclusive. Some studies indicate small elevations in risk, while others show no risk associated with either induced or spontaneous abortions.

Some inconsistencies in the findings of the Daling study were puzzling, as the study [did not] show an increase in risk associated with spontaneous abortions.

This assessment of the literature as inconsistent is accurate if the numerous studies that did not distinguish between induced abortions and miscarriages are included in the record. Because induced abortions involve estrogen overexposure and spontaneous abortions do not, such inconsistency is not surprising. As Planned Parenthood points out, "miscarriages and induced abortions affect a woman's body differently, but many studies have not distinguished between them." The 1996 Brind meta-analysis, on the other hand, confined itself to the twenty-three studies then available that reported data specifically on induced abortion and reached the following conclusion: "We believe that the present review and meta-analysis summarises a literature that documents a remarkably

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183. National Cancer Institute, supra note 24.
184. See supra note 11.
185. "Spontaneous and induced abortions should be studied separately. Induced abortion limits a full-term pregnancy to its early phases, but the hormonal changes should be the same as in a normal pregnancy up to the time of abortion. Miscarriage might reflect an insufficient rise of pregnancy hormones." Michels & Willett, supra note 13, at 522.
186. Planned Parenthood Fact Sheet, supra note 20, at 1.
consistent, significant positive association between induced abortion and breast cancer incidence."\textsuperscript{187}

3. STRENGTH OF THE ASSOCIATION

The strength of an association is measured by the relative risk.\textsuperscript{188} The greater the relative risk, the more likely it is that the relationship is causal.\textsuperscript{189} The reason for this is simply that a weak association, such as a 1.5 or 2.0 relative risk, could plausibly be the result of mere bias or chance, while a relative risk of 9.0 or 10.0 could not.\textsuperscript{190} Thus, "[a] strong association serves only to rule out hypotheses that the association is entirely due to one weak unmeasured confounder or other source of modest bias."\textsuperscript{191} Weaker effects are not believed to be rarer phenomena than stronger effects.\textsuperscript{192} In fact, they are more likely to occur when the background risk, which is increased, is already high.\textsuperscript{193} It would be impossible, for example, for a twelve percent risk of breast cancer to be increased by a factor of 9.0.

Nevertheless, weaker statistical associations must be scrutinized more closely for methodological bias.\textsuperscript{194} Although the relative risk associated with the ABC link is classified as "weak," the evidence described above supports neither chance nor bias as an explanation for the link.\textsuperscript{195} A comparison of the ABC link with other risk factors that are recognized and disclosed may be useful in convincing the trier of fact that a reasonable patient would want to know about the ABC link before choosing an abortion.\textsuperscript{196} The relative risk of breast cancer typically associated with early first menstruation is about 1.3, while that associated with late menopause is about 1.5.\textsuperscript{197} According to the Surgeon General, the relative risk of spontaneous abortion, fetal death, and neonatal death

\textsuperscript{187} Brind et al., supra note 10, at 494.
\textsuperscript{188} See Reference Manual, supra note 12, at 161.
\textsuperscript{189} See id.
\textsuperscript{190} See id. at 161 n.109.
\textsuperscript{191} ROTHMAN & GREENLAND, supra note 2, at 25.
\textsuperscript{193} One explanation for the weak relation between cigarette smoking and cardiovascular disease is that "cardiovascular disease is common, making any ratio measure of effect comparatively small compared with ratio measures for diseases that are less common." ROTHMAN & GREENLAND, supra note 2, at 24 (citation omitted).
\textsuperscript{194} See Reference Manual, supra note 12, at 161.
\textsuperscript{195} See supra Part IV.C.1.
\textsuperscript{196} See cf. supra notes 34, 63 and accompanying text.
\textsuperscript{197} See Harris et al, supra note 6, at 321.
from maternal smoking during pregnancy is about 1.25, but that of premature birth is approximately 1.4. The Environmental Protection Agency's classification of secondhand smoke as a Known Human (Group A) Carcinogen, "used only when there is sufficient evidence from epidemiologic studies to support a causal association," was based on a 1.19 relative risk of lung cancer. Moreover, a federal district court found that the EPA had to lower its meta-analysis confidence level from the standard ninety-five percent down to ninety percent in order to make this association statistically significant.

D. The Materiality of “Potential” Risks

While the degree of probability that induced abortion has a stimulating biologic effect on the development of breast cancer is still open to debate by reasonable people, such debate has often ignored an essential point: the probability that a causal link exists does not necessarily have to be extremely high before it can be significant in the decision-making process. A risk is simply a "probability that an event will occur," and a measure of uncertainty as to causality fits naturally within the matrix. In mathematical terms, even a fifty percent chance that a one in 100 risk of death is not real still amounts to a one in 200 risk of death.

The implication is that potential risk factors are actual risks for patients faced with a decision and can be far more material to that decision than well-established risk factors. Therefore, a scientist’s judgment that the evidence of a causal link between induced abortion and breast cancer is inconclusive is almost meaningless for real-life decision-


199. Four studies reported relative risks ranging from 1.36 to 1.47. See U.S. DEP’T OF HEALTH & HUMAN SERVS., THE HEALTH CONSEQUENCES OF SMOKING FOR WOMEN 219 (1980).

200. U.S. ENVTL. PROTECTION AGENCY, THE RISK ASSESSMENT GUIDELINES OF 1986 1-12 (1986). An agent would be classified as a Probable Human (Group B) Carcinogen if the epidemiologic evidence is "limited," meaning that "a causal interpretation is credible, but that alternative explanations, such as chance, bias, or confounding, could not adequately be excluded." Id. at 1-11, 1-12.


202. See id. at 462.

203. REFERENCE MANUAL, supra note 12, at 176.
making. Conclusive certainty is not required. 204 "[A]rguably, there are no certainties in science," 205 or anywhere else, for that matter. Most practical decisions in life are based not on certainties, but on probabilities.

This truth is implicitly recognized in other contexts. Scientists still do not know what specific component in cigarettes causes lung cancer, yet "the early epidemiological studies provided grounds for assuming for all practical purposes that the observed relationship was causal." 206 For decades the Food and Drug Administration has required manufacturers of oral contraceptives to discuss the evidence of a possible link with breast cancer in the warnings section of package inserts. Yet, "[a]t best it would seem that the possible association . . . remains unresolved . . . . Even if an association was established, it may not be causative." 207 Likewise, the Occupational Safety and Health Administration considers just one statistically significant epidemiological study as "sufficient to establish a hazardous effect" which must be disclosed. 208 In 1994, breast implant manufacturers agreed to a $4.25 billion class-action settlement before any epidemiological studies on a possible link with connective tissue disease had yet been published. 209

In *Harbeson v. Parke Davis, Inc.*, 210 the Ninth Circuit affirmed the liability of three physicians for failing to discover and disclose the potential risks of birth defects from taking Dilantin during pregnancy. 211 The district court found that the following warning in the Physicians' Desk Reference was sufficient to put the physicians on notice of these risks: "Although evidence of a teratogenic [that which causes fetal malformations] effect in the human has not been established, the use of

204. "Scientific proof of a link between a chemical or biological agent and a disease is not, never has been, and never should be required for a government to take action against a substance suspected of causing epidemics." PETER PRINGLE, CORNERED: BIG TOBACCO AT THE BAR OF JUSTICE 117-18 (1998).


206. PRINGLE, supra note 204, at 118.


Nevertheless . . . perhaps patients should, at best, be allowed to make informed choices—and bearing in mind that alternative methods of contraception are available . . . and that the risk under discussion is a very grave and not uncommon one—it seems reasonable to conclude that patients should be told of this unresolved problem before . . . they embark on the use of oral contraceptives.

Id. at 31.


209. See ANGELL, supra note 2, at 22-23.

210. 746 F.2d 517 (9th Cir. 1984).

211. See id. at 518-19.
this drug in pregnancy requires that its potential benefits be weighed against possible hazards to the fetus." 212 The physicians had a duty to conduct a reasonable "literature search," which would have revealed "several articles" on the risks of birth defects from taking Dilantin. 213

In upholding the district court's finding that these risks were "material," the Ninth Circuit stated:

[W]e believe a risk must be disclosed even if it is but a potential risk rather than a conclusively determined risk . . . . It may be that those risks had not yet been documented or accepted as a fact in the medical profession. Nonetheless, under the doctrine of informed consent, those risks should have been disclosed. Medical knowledge should not be limited to what is generally accepted as a fact by the profession. To hold otherwise would defeat the purpose of the doctrine, give little weight to exploratory medical research, and invite impossible line drawing. 214

Compared to the several articles at issue in Harbeson, the scientific evidence of the ABC link suggests a much stronger likelihood of causation. A plausible biological hypothesis is substantiated by numerous studies, which consistently show a statistically significant association that has not been vitiated by any evidence of bias or confounding. Most objective triers of fact would find this situation presents at the very least a strong likelihood of causation and that this probability could be significant in a reasonable patient's decision-making process. This potential significance is more than sufficient to establish a duty to warn. In short, the evidence shows that abortion providers have been, and are now, breaching a duty to inform women about the ABC link before they consent to the procedure.

V. CAUSATION OF INJURY

A plaintiff bringing an action under a negligence theory of liability must demonstrate that the defendant's breach of duty was a legal cause of her injury. In the informed consent context, this element requires the plaintiff to prove "two links in the causal chain:" first, that the defendant's failure to adequately inform was a legal cause of the patient's consent to the procedure ("decision causation"); and second, that the

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212. Id. at 523.
213. Id. at 525.
214. Id.
Unauthorized procedure was a legal cause of the patient's injury ("injury causation").

After a general discussion of causation in terms of what must be proved ("burden of proof") and to what degree of certainty ("standard of persuasion"), this Part separately considers decision causation as it relates to a plaintiff who was not informed about the ABC link before she consented to an abortion, and injury causation as it relates to such a plaintiff who has been diagnosed with breast cancer.

A. Burden of Proof and Standard of Persuasion

The plaintiff has the burden of proving legal causation. According to the Restatement (Second) of Torts, "negligent conduct is a legal cause of harm to another if . . . [the] conduct is a substantial factor in bringing about the harm." This substantial factor test incorporates the traditional requirements that the defendant's negligence be both a cause-in-fact and proximate cause of the plaintiff's injury in order to establish liability. The word "substantial" preserves the equitable purposes of the proximate causation doctrine, limiting liability to those acts which reasonable people would regard as a "cause . . . in the popular sense, in which there always lurks the idea of responsibility, rather than in the so-called 'philosophic sense,' which includes every one of the great number of events without which any happening would not have occurred." The latter sense defines "but for" causation, also known as "actual" causation or "cause-in-fact." Some courts have interpreted the substantiality test as displacing the plaintiff's traditional burden of proving cause-in-fact in the problematic context of toxic tort litigation. An alternative approach to effecting

215. Heinemann, supra note 42, at 1083.
216. See generally Steve Gold, Note, Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence, 96 YALE L.J. 376 (1986) (arguing that the challenges of proving causation in toxic torts have created confusion between the substantive "burden of proof" and the "standard of persuasion" which must be met to satisfy the burden).
220. RESTATEMENT, supra note 217, § 431 cmt. a (1965).
the policy judgments implicit in such decisions is to more explicitly acknowledge that standards of persuasion, such as "more probable than not," are not inflexible rules that demand of the fact-finder a mathematically precise degree of certainty that the fact alleged is true. Rather, the trier of fact should remain free to consider all related factors, including the defendant's responsibility, in determining whether she is persuaded that the defendant's conduct was a legal cause of the plaintiff's injury.223

This flexibility in the standard of persuasion is illustrated by the so-called "lost chance" line of medical malpractice cases, in which courts have found liability despite also finding that the plaintiff more likely than not would have suffered the harm alleged even with proper treatment.224 In Ehlinger v. Sipes,225 for example, the Ehlingers alleged that their obstetrician's negligent failure to diagnose Mrs. Ehlinger's pregnancy as a multiple pregnancy was a substantial factor in causing the injuries her twin children suffered as a result of premature birth.226 Expert testimony at trial established that the obstetrician's negligence deprived Mrs. Ehlinger of the opportunity to receive treatment which "could" have prolonged her pregnancy and thus lessened or avoided the twins' injuries, but did not establish that such treatment more probably than not "would" have done so.227 Nevertheless, the Wisconsin Supreme Court concluded that the expert's testimony was sufficient to present the causation question to the trier of fact.228 The court "refuse[d] to place upon an injured plaintiff the burden of proving what more probably than not would have happened had the defendant not been negligent," reasoning that such a

The substantial factor standard—which ascribes liability to a cause which has played an important part in the production of the harm, even though the harm may have occurred absent that cause—is particularly suited to injury from chronic exposure to toxic chemicals where the subsequent manifestation of biological disease may be the result of a confluence of causes. See also supra notes 266-67 and accompanying text.

223. See, e.g., Rebecca S. Dresser et al., Breast Implants Revisited: Beyond Science on Trial, 1997 Wis. L. Rev. 705, 742-43 ("Indeed, juries in the breast implant cases may simply be doing sub rosa what the legal system should do formally: shift the burden of proof to the manufacturers to disprove causation when the absence of safety research is due to the manufacturer's own neglect.").
224. Cf. Black & Hollander, supra note 219, at 19-20 (characterizing these cases as a "change" in the standard of persuasion).
225. 155 Wis. 2d 1, 454 N.W.2d 754 (1990).
226. See id. at 5, 454 N.W.2d at 755.
227. See id. at 5-8, 454 N.W.2d at 755-56.
228. See id. at 6, 454 N.W.2d at 755.
burden would require "obvious speculation and proof of the impossible." The court noted that:

While the plaintiff’s burden to present the causation question to the trier of fact is less than otherwise required, and to that extent does involve some measure of uncertainty, the courts have generally been liberal in allowing the trier of fact to determine whether the defendant’s negligence caused the plaintiff’s injury in such circumstances.

The concerns addressed by these “lost chance” cases parallel the difficult problems of proof presented by the decision causation and cancer causation questions. Decision causation, which generally asks whether a reasonable patient would have consented to the procedure had she been adequately informed, “encumbers the court in a host of post facto difficulties: . . . assuming that the offered treatment was itself reasonable (since otherwise the doctor presumably would be liable for negligent care), it is virtually impossible to determine what a hypothetical, ‘reasonable’ patient would have done” had the defendant not been negligent. Likewise, the epidemiological evidence suggests that while all women who have had abortions and been later diagnosed with breast cancer could prove that the abortion more likely than not significantly reduced their chance of avoiding breast cancer, and thus could have been a “but for” cause of their injury, fewer women would be able to prove that the abortion more likely than not was a cause-in-fact. Under such circumstances, the causation question should remain “a matter for determination by a jury upon due consideration of all related factors.”

B. Cancer Causation

Epidemiologists define a “cause” of a disease as “an event, condition, or characteristic that preceded the disease event and without which the disease event either would not have occurred at all or would

229. Id. at 18-19, 454 N.W.2d at 761 (citing Sumnicht v. Toyota Motor Sales, 121 Wis. 2d 338, 356, 360 N.W.2d 2 (1984) (quoting Mitchell v. Volkswagenwerk, 669 F.2d 1199, 1204-05 (8th Cir. 1982))).
230. Id. at 18, 454 N.W.2d at 761.
231. Heinemann, supra note 42, at 1083-84.
232. Roberson v. Counselman, 686 P.2d 149, 160 (Kan. 1984) (holding that issue of material fact existed as to whether defendant’s medical malpractice was a substantial factor causing patient’s death, even though patient would have had only a 40% chance of surviving even with proper treatment).
not have occurred until some later time."^{233} Thus defined, no single cause is sufficient by itself to produce disease.\textsuperscript{234}

This definition then does not define a complete causal mechanism but a component of it \ldots A "sufficient cause," which means a complete causal mechanism, can be defined as a set of minimal conditions and events that inevitably produce disease; "minimal" implies that all of the conditions or events are necessary.\textsuperscript{235}

A sufficient cause minus one of these necessary components is known as that component's "causal complement," defined as a "set of conditions necessary and sufficient for a factor to produce disease."\textsuperscript{236}

The legal issue in the cause of action contemplated, therefore, is whether the plaintiff's induced abortion was a component (i.e. "but for" cause) of the particular causal mechanism which produced the onset of her breast cancer. The likelihood of this historical fact is a question different in kind from that of "risk," which is the likelihood that an event will occur in the future. Unfortunately, the fraction of exposed cases for whom the exposure was a component of the disease mechanism (i.e. the probability of causation, or "etiologic fraction"), "[w]hile of great biologic and legal interest, \ldots cannot be epidemiologically estimated if nothing is known about the fraction F [of completions of other sufficient causes of the disease which were preceded by completion of the exposure's causal complement]."\textsuperscript{237} For example:

\begin{quote}
\textsuperscript{233} ROTHMAN & GREENLAND, supra note 2, at 8. Suppose that exposure to factor A leads to epilepsy after an interval of 10 years, on the average. It may be that exposure to a drug, B, would shorten this interval to 2 years. Is B acting as a catalyst or as a cause of epilepsy? The answer is both: a catalyst is a cause. Without B, the occurrence of epilepsy comes 8 years later than it comes with B, so we can say that B causes the onset of the early epilepsy. It is not sufficient to argue that the epilepsy would have occurred anyway. First, it would not have occurred at that time, and the time of occurrence is part of our definition of an event. Second, epilepsy will occur later only if the individual survives an additional 8 years, which is not certain. Not only does agent B determine when the epilepsy occurs, but it can also determine whether it occurs.
\end{quote}

\textit{Id.} at 15.
\textsuperscript{234} See \textit{id.} at 8.
\textsuperscript{235} \textit{id.}
\textsuperscript{236} \textit{id.} at 11.
\textsuperscript{237} \textit{id.} at 56.
Imagine a cohort in which, for every member, the causal complement of exposure, C, will be completed before the sufficient cause C1 [i.e. all sufficient causes of a disease that do not contain the exposure E] is completed. If the cohort is unexposed, every case of disease must be attributable to the cause C1. But if the cohort is exposed from start of follow-up, every case of disease occurs when C is completed (E being already present), so every case of disease must be attributable to the sufficient cause containing C and E. 238

Consequently, in an exposed cohort the incidence rate of cases which were caused by the exposure is not necessarily limited to the difference between the risk of disease when exposure is absent and the risk when exposure is present, since in the exposed cohort, a “fraction F of the cases that would have occurred without exposure will now be caused by exposure. In addition, there may be cases caused by exposure for whom disease would never have occurred.” 239

Nevertheless, the “attributable risk” or “rate fraction,” defined as the risk difference divided by the risk when exposure is present, has “often been incorrectly interpreted as the... etiologic fraction.” 240 Based on such an understanding, a number of courts in toxic torts cases have ruled the plaintiff’s evidence insufficient to meet the standard of persuasion and burden of production on the element of causation. The Federal Judicial Center’s Reference Manual on Scientific Evidence has unfortunately perpetuated this misconception in its chapter on epidemiology:

The threshold for concluding that an agent was more likely the cause of a disease than not is a relative risk greater than 2.0. . . . When the relative risk reaches 2.0, the agent is responsible for an equal number of cases of disease as all other background causes. Thus, a relative risk of 2.0 implies a 50% likelihood that an exposed individual’s disease was caused by the agent. . . . When the attributable risk exceeds 50% (equivalent to a relative risk greater than 2.0), this logically might be converted to a belief that the agent was more likely than not the cause of the plaintiff’s disease. 241

238. Id. at 54.
239. Id. at 54-55. See supra note 233.
240. Id. at 54.
241. REFERENCE MANUAL, supra note 12, at 168-69.
Epidemiologist Diana Petitti, in a review of this chapter in the Reference Manual on Scientific Evidence, reserves her harshest criticism for the above statements: "I could not find support for these statements in any textbooks of epidemiology, and there are no empirical studies to support them . . . Under no circumstances should [judges] accept the statements . . . at face value."242 She strongly recommends that the courts "re-examine the precedent they have set."243

Requiring the plaintiff to prove that the exposure more than doubled her risk of the disease in order to satisfy her burden of production is questionable not only on scientific but also on legal and equitable grounds. First, the 2.0 rule misapplies the standard of persuasion, both by confusing the standard with the substantive "burden of proof" and by enforcing a dubiously literal and impossibly precise interpretation of "more likely than not."244 Second, the rule necessarily leads to "unjust results."245 If an exposure increases the likelihood of a disease but does not more than double it, the tortious defendant escapes all liability for the harm actually caused to some plaintiffs.246 Ironically, the higher the background risk which is increased, the more likely the relative risk is to fall below the 2.0 threshold.247 This means that highly significant and material absolute risk increases may be less likely to support recovery than smaller risk increases. As an extreme example, increasing a background fifty percent risk of death to 100% would still not render the defendant liable under the 2.0 rule, since the risk was not more than doubled, even though the absolute risk increase was a very significant fifty percent.

243. Id.
244. See generally Gold, supra note 216, at 393-95.
245. See infra note 251.
246. See In re "Agent Orange" Prod. Liab. Litig., 597 F. Supp. 740, 836 (E.D.N.Y. 1984), aff'd, 818 F.2d 145 (2d Cir. 1987): In mass exposure cases . . . this all-or-nothing rule results in either a tortious defendant being relieved of all liability or overcompensation to many plaintiffs and a crushing liability on the defendant. These results are especially troublesome because . . . it may be possible to ascertain with a fair degree of assurance that the defendant did cause damage, and, albeit with somewhat less certainty, the total amount of that damage.
247. Judge Weinstein discussed the class action as one possible solution to the problems of the all-or-nothing rule: "The defendant would then be liable to each exposed plaintiff for a pro rata share of that plaintiff's injuries." Id. at 838.
Nevertheless, many courts have employed the 2.0 rule to find the plaintiff’s evidence of causation insufficient to sustain a jury verdict. On remand from the United States Supreme Court’s decision in Daubert, for example, the Ninth Circuit again affirmed the exclusion of plaintiffs’ proffered expert testimony on the link between Bendectin, a drug prescribed for morning sickness, and birth defects. Judge Kozinski, writing for the court, noted that the plaintiffs’ experts were only willing to testify that Bendectin is “capable of causing” birth defects, and that they did not attempt to quantify the probability that it did cause the plaintiffs’ injuries. The court ruled that “statistical proof... must establish not just that... Bendectin increased somewhat the likelihood of birth defects, but that it more than doubled it—only then can it be said that Bendectin is more likely than not the source of their injury.”

The court recognized that an epidemiological study showing a relative risk less than 2.0 could be combined with other evidence to establish causation by a preponderance of the evidence, but observed that in the instant case, “plaintiffs’ experts did not seek to differentiate these plaintiffs from the subjects of the statistical studies. The studies must therefore stand or fall on their own.”

Most courts have generally found epidemiological evidence sufficient to satisfy the burden of production on the issue of physical causation.


249. See Daubert v. Merrell Dow Pharms., Inc., 43 F.3d 1311, 1311 (9th Cir. 1995).

250. Id. at 1321.

251. Id. at 1320. The court was applying California tort law. Judge Kozinski recognized that “there will be unjust results under this substantive standard. If a drug increases the likelihood of birth defects, but doesn’t more than double it, some plaintiffs whose injuries are attributable to the drug will be unable to recover.” Id. at 1320 n.13. Some commentators advocate proportional recovery based on the attributable fractions of causation as an alternative to an all-or-nothing approach to damages. See, e.g., 2 AMERICAN LAW INST., ENTERPRISE RESPONSIBILITY FOR PERSONAL INJURY 369-75 (1991); Daniel A. Farber, Toxic Causation, 71 MINN. L. REV. 1219, 1237-51 (1987).

252. Daubert, 43 F.3d at 1321 n.16.

253. Judge Weinstein in the “Agent Orange” litigation noted two different rules applied by the courts regarding the types of evidence necessary to prove causation. Under the “strong” version, the plaintiff must produce both statistical evidence indicating that the probability of causation exceeds 50% in the exposed population and “particularistic” proof that the defendant’s conduct caused the individual plaintiff’s injuries. In re “Agent Orange” Prod. Liab. Litig., 597 F. Supp. 740, 835 (E.D.N.Y. 1984), aff’d, 818 F.2d 145 (2d Cir. 1987). The “weak” version, on the other hand, permits verdicts to be based
The Supreme Court of Texas, however, uses a somewhat stricter standard than did the Ninth Circuit in *Daubert*. In *Merrell Dow Pharmaceuticals, Inc. v. Havner*, the court reversed an appellate court's affirmation of a jury verdict for plaintiffs, finding the plaintiffs' expert testimony on Bendectin legally insufficient to establish causation. The court found that none of the more than thirty published, peer-reviewed epidemiological studies on the relationship between Bendectin and birth defects had shown a statistically significant association, while none of the contrary findings offered by the plaintiffs' experts had been published or otherwise subjected to peer-review. In finding the plaintiffs' scientific evidence unreliable, the court adopted the requirement that epidemiological studies show more than a "doubling of the risk" in order to support a finding of causation. In addition, the court ruled that "if there are other plausible causes of the injury or condition that could be negated, the plaintiff must offer evidence excluding those causes with reasonable certainty." The court declined to decide whether epidemiological studies showing a relative risk less than 2.0, in combination with other evidence of causation, could be legally sufficient evidence of causation.

The Utah federal district court's detailed and often-cited opinion in *Allen v. United States*, on the other hand, explicitly rejected the notion that the court is "constrained by simplistic models of causal probability impressed upon the judicial 'preponderance of the evidence' standard." In *Allen*, twenty-four plaintiffs had brought civil actions against the United States to recover for cancer or leukemia allegedly caused by exposure to fallout radiation from open-air atomic testing conducted near their homes. The court found the United States liable for negligently failing to adequately warn nearby residents of the known

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254. 952 S.W.2d 706 (Tex. 1997).
255. See id. at 708.
256. See id. at 726-28.
257. See id. at 718.
258. Id. at 720.
259. See id. at 719.
261. Id. at 418.
262. See id. at 247.
or potential long-range biological consequences from such exposure. The court would not presume a causal relationship from the government’s negligence, the court held that “so long as the evidence will support an inference that defendant’s conduct contributed to the victim’s injury,” even though contrary inferences can be drawn, “it is for the finder of fact . . . to draw the most appropriate inference using . . . judgment, experience and common sense in light of all the circumstances.” The court described the “double the risk” test as a “refabrication of the ‘but-for’ test of causation in mathematical form,” and observed that when the defendant has a duty to protect the plaintiff “from even the possibility of harm, or where, as here, defendant’s wrongful conduct arguably has denied to plaintiff a potential opportunity to avoid serious or lethal injury, analysis using ‘but-for’ tests in any form falls far short of the mark.” The court applied instead the Restatement’s substantial factor test to determine legal cause. The court ruled that where such a substantial factual connection exists between the plaintiff’s injury and the defendant’s negligence, “but selection of ‘actual’ cause-in-fact from among several ‘causes’ is problematical, those difficulties of proof are shifted to the tortfeasor, the wrongdoer, in order to do substantial justice between the parties.”

As trier of fact, the Allen court considered epidemiological and particularistic evidence for each of the twenty-four plaintiffs, and found

263. See id. The court criticized the government’s public information program in the following terms:

The public pronouncements as given do not really warn and do not sufficiently educate. They reassure. They don’t talk of potential long-term dangers. They talk of how effectively the program is being managed . . . . They demonstrate that responsible persons at the operational level of continental nuclear testing neglected an important, basic idea: there is just nothing wrong with telling the American people the truth.

Id. at 404.

For a discussion of the duty to disclose “potential” risks in the context of low level radiation exposure, see id. at 360–62. The court concluded:

From the preponderance of the historical and scientific materials now before this court . . . . a reasonable person, exercising great care in light of the best of available scientific knowledge, would err on the side of caution by assuming no “safe” threshold exposure to atomic radiation, i.e., that any degree of exposure equates with some corollary degree of biological risk.

Id. at 362.

264. Id. at 413.

265. Id. (quoting Leon Green, The Causal Relation Issue in Negligence Law, 60 Mich. L. Rev. 543, 560 (1962)).

266. Id. at 418–19.

267. See id. at 415–16.

268. Id. at 411.
that in ten of the cases a substantial factual connection with the defendant’s negligence more likely than not existed. In the case of a plaintiff diagnosed with breast cancer, a “fractional causation” estimate of 47.5%, in combination with evidence that she was exposed to radiation at a young age, when susceptibility to increased breast cancer risk from radiation is greater, and that her twenty-five year latency period following exposure was consistent with that observed among the victims of Hiroshima and Nagasaki, was found to “[preponderate] heavily in favor of an inference that exposure to fallout radiation was a substantial factor contributing to her injury.”

In sum, establishing the causal connection between a plaintiff’s induced abortion and her breast cancer will be a formidable but surmountable challenge to the viability of the plaintiff’s cause of action. The use of epidemiological evidence to prove individual causation is a complex and still-evolving area of the law, and the courts use varying standards in evaluating the sufficiency of such evidence. Several jurisdictions, through dubious use of the “double the risk” sufficiency threshold, have made no allowance for just claims by plaintiffs who were negligently exposed to weak—but quite significant—relative risks between 1.0 and 2.0. Some plaintiffs suffering from breast cancer may therefore have difficulty getting to a jury if they can “only” show that their risk of the second-leading cause of death among middle-aged women was “merely” increased by half. On the other hand, the epidemiological evidence suggests that the risk of breast cancer is more than doubled by an induced abortion in many cases, particularly among women who are African-American, women who were relatively young or old at the time of the induced abortion, and women who have a family history of breast cancer. Moreover, plaintiffs who can rule out the common risk factors for breast cancer increase the plausibility that they would not have developed breast cancer but for the induced abortion. This suggests that their individual relative risk from the induced abortion exceeded the average estimate and meets the 2.0 threshold.

C. Decision Causation

Most jurisdictions have followed Canterbury v. Spence in adopting the so-called “objective” standard for establishing decision causation. While the “subjective” standard focuses on whether the

269. See id. at 429-43.
270. Id. at 441.
271. See supra Part IV.B.
particular plaintiff would have withheld consent to the therapy had all material risks been disclosed, the "objective" standard requires the trier of fact to determine what a "reasonable person in the plaintiff's position" would have decided had she been adequately informed.\textsuperscript{274} In making this determination, "[t]he trier of fact must objectively weigh the necessity for the operation against the incidence of risk . . . and the severity of the potential injury."\textsuperscript{275} Generally, the more serious the condition being treated is, the more likely the jury is to find that the patient would have undergone the procedure even if she had been informed of all the risks.\textsuperscript{276} Conversely, when the procedure is not necessary to preserve the life or health of the patient, a material risk of the procedure weighs more heavily in the decision-making process.

A material risk is by definition one which a reasonable patient would consider significant in deciding whether to forego the procedure.\textsuperscript{277} A finding of materiality therefore implies that the risk has the potential to actually change the patient's decision, either alone or in combination with other factors.\textsuperscript{278} The decision whether or not to terminate a pregnancy is often beset by strong social and economic pressures to terminate. Yet other powerful considerations presumably move many women away from the abortion, so that a woman often contemplates the decision with an ambivalent and divided mind. In such circumstances, it is quite plausible that a significantly increased risk of breast cancer could be the deciding factor that tips the scales against abortion. The availability of adoption as an alternative to child-rearing underlines the plausibility of the reasonable person arriving at this choice.

Decision causation is ultimately a jury question. Although under the objective standard the plaintiff's testimony about what she would have decided is not determinative, it is still relevant.\textsuperscript{279} Unless the plaintiff's position at the time of the decision to abort was extraordinary in some way, the jury will have no sound basis for doubting that a reasonable person in her position would have foregone the abortion if she had known about the material risk posed by the ABC link. To deny recovery based

\textsuperscript{274} Waltz & Scheuneman, supra note 137, at 647.
\textsuperscript{276} See 2 LOUISELL & WILLIAMS, supra note 26, ¶ 22.13.
\textsuperscript{277} See supra note 51 and accompanying text.
\textsuperscript{278} See Waltz & Scheuneman, supra note 137, at 638.
\textsuperscript{279} Although a single risk of a given magnitude may not cause a patient to forego a therapy, two or more such risks in combination might have that result. A standard of materiality limited to risks which, in isolation, would cause a patient to refuse a therapy deprives the patient of the opportunity to contemplate possible combinations of risks.

\textit{Id. at 639} (footnote omitted).
\textsuperscript{279} See 2 LOUISELL & WILLIAMS, supra note 26, ¶ 22.14.
simply on a belief that fewer than fifty-one percent of women intending to abort would change their mind if properly informed cannot be what the objective standard calls for. Such an interpretation would bury the duty to warn by essentially requiring that the procedure itself be unreasonably dangerous to support recovery. "The doctrine of informed consent does not exist to tell health care providers whether or not to offer certain treatment." The plaintiff's attorney must therefore ensure that the jury properly understands the objective standard, as well as appreciates the flexibility inherent in the standard of persuasion.

VI. DISREGARD OF PLAINTIFF'S RIGHTS AS AN INDEPENDENT INJURY

Establishing causation is not essential to an informed consent cause of action if the failure to inform is recognized as a harm in and of itself. Commenting on the courts' presumption in cases such as *Reyes v. Wyeth Laboratories* that consumers would have heeded warnings of even remote risks had they been given, Professor John Kidwell hypothesized that ideals of individual sovereignty were at work in the causation issue:

Perhaps a person who received warning of the risks attendant to a choice incurs less loss when the harm actually results than the person who was not warned; the fact of having been warned, apart from any ability to respond to the warning, reduces the sense of injury that occurs when the risk eventuates. In this sense the lack of warning causes an independent injury.

In the informed consent context, recognition of this independent dignitary interest is especially clear in the minority of jurisdictions which continue to apply the battery theory of liability. In *Gouse v. Cassel*, for example, the Supreme Court of Pennsylvania affirmed the lower court's refusal to "eviscerate the doctrine of informed consent by

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280. In any event, such a belief may be highly questionable with regard to women with a family history of breast cancer. *See supra* notes 149-51 and accompanying text.
282. 498 F.2d 1264 (5th Cir. 1974).
283. John A. Kidwell, *The Duty to Warn: A Description of the Model of Decision*, 53 TEx. L. Rev. 1375, 1408 (1975); *see also* Planned Parenthood of Southeastern Pa. v. Casey, 505 U.S. 833, 882 (1992) ("In attempting to ensure that a woman apprehend the full consequences of her decision, the State furthers the legitimate purpose of reducing the risk that a woman may elect an abortion, only to discover later, with devastating psychological consequences, that her decision was not fully informed."). *quoted in* Thomas R. Eller, *Informed Consent Civil Actions for Post-Abortion Psychological Trauma*, 71 Notre Dame L. Rev. 639, 663 (1996).
predicating materiality and, thus, the mandate for disclosure of risks and alternatives, upon a factfinder’s determination that a plaintiff-patient would have declined treatment had the disclosures been made.  

Rather, once the patient demonstrates that she was not informed of material facts concerning a proposed operation, “the causation inquiry ends,” and the sole remaining issue is the determination of damages.

Accordingly, a plaintiff traditionally can state a claim under battery theory based on dignitary injury alone, even if the undisclosed risk has not materialized. Of course, such a lawsuit typically is not worth bringing unless the plaintiff can claim substantial compensatory or punitive damages. In the case of a woman whose risk of breast cancer has been increased by an induced abortion without her knowledge or consent, such damages might include expenses for “medical monitoring” and “fear of disease.”

In *Trogun v. Fruchtman,* the Supreme Court of Wisconsin first recognized, at the urging of the plaintiff-appellant, informed consent as a cause of action under negligence rather than battery. The court listed five reasons for the inadequacy of the battery theory of liability in the informed consent context, including: “the failure to inform a patient is probably not, in the usual case, an intentional act and hence not within the traditional concept of intentional torts,” and “these essentially negligence cases do not fit the traditional mold of situations wherein punitive damages can be awarded.” Perhaps, however, the courts should allow the battery theory—or a new cause of action—for the atypical informed consent case in which the failure to inform may in fact

285. Id. at 335.
286. Id.
287. See Faden & Beauchamp, supra note 28, at 27. “Treatment without consent is itself an actionable wrong in battery law. The defendant . . . who intentionally causes an unpermitted contact with the plaintiff . . . has committed a ‘technical battery’ and is liable for damages that usually are ‘token’ or ‘nominal’—a small sum not meant as compensation for measurable harm.” Id.
290. 58 Wis. 2d 569, 207 N.W.2d 297 (1973).
291. See id. at 598-600, 207 N.W.2d at 312-14.
292. Id. at 599-600, 207 N.W.2d at 313.
have been intentional, and wherein punitive damages may be appropriate, but where negligence theory would not support compensation because the risk has not materialized into an injury. Such an action would seem particularly justified for a woman who was intentionally not informed of the ABC link, and who must now live with the knowledge that her risk of breast cancer has been increased without her knowledge or consent. If a jury finds that the defendant acted in an "intentional disregard of the rights of the plaintiff," then it may award punitive damages in a sum to be determined in part by considering the "potential damage which might have been done by such acts as well as the actual damage." As recently noted by the Wisconsin Supreme Court in the course of reinstating a jury verdict for $1 in nominal damages and $100,000 in punitive damages for a trespass to land, "a right is hollow if the legal system provides insufficient means to protect it." Abortion providers' continuing failure to inform patients about the steadily accumulating evidence of the ABC link can also be considered a trespass, not against land, but against the patient's "right to determine what shall

293. Cf., e.g., Northern, *supra* note 65. "[I]n the few cases . . . in which a doctor intentionally has deprived a woman of a procreative choice by withholding information the doctor knew was material to the woman's decision, a battery action is appropriate. If such abuses occur, they should be cured." *Id.* at 507-08.


295. See generally Christopher J. McAuliffe, Comment, *Resurrecting an Old Cause of Action for a New Wrong: Battery as a Toxic Tort*, 20 B.C. Envtl. Aff. L. Rev. 265, 290-93 (1993). "Punitive damages not only punish defendants but also can compensate plaintiffs for otherwise noncompensable injuries." *Id.* at 290.


297. *Jacque v. Steenberg Homes, Inc.*, 209 Wis. 2d 605, 608, 563 N.W.2d 154, 160 (1997). In *Jacque* the plaintiffs had expressly and repeatedly forbade the defendants from delivering a mobile home via the plaintiffs' snow-covered field. *See id.* at 609, 563 N.W.2d at 156. Nevertheless, the defendants deviously plowed a path through the field and delivered the mobile home. *See id.* at 628, 563 N.W.2d at 164. The court agreed with the plaintiffs' contention that the "rationale for not allowing nominal damages to support a punitive damage award is inapposite when the wrongful act involved is an intentional trespass to land," because "both the individual and society have significant interests in deterring intentional trespass to land, regardless of the lack of measurable harm that results." *Id.* at 617, 563 N.W.2d at 159.
be done with [her] own body."298 This right is far from trivial, and deserves effective legal protection.

VII. CONCLUSION

Although the scientific evidence may not yet prove beyond the shadow of a doubt that induced abortion causes breast cancer, there is no doubt that abortion providers have a duty to inform women considering the procedure about this significant health risk before an abortion is performed. Moreover, public health officials must take active measures to inform the public at large about the ABC link, so that women may take extra care to avoid pregnancies they are not ready to carry to term. Women have been led to believe that abortion is a safety net, when in fact its safety is in serious doubt. Information about the ABC link is also vital for women who have already had abortions, so that they may take extra precautions for the early detection of breast cancer. Malpractice lawsuits of the type outlined in this Comment may serve an important role in raising public awareness of the ABC link, particularly if public health officials continue to neglect their responsibilities in this regard. Such litigation and public education should remain separate and distinct from the moral and political debate over abortion. What is at stake is nothing more—and nothing less—than the right to know the truth.
